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The Use of Radiofrequency in the Medicine

Revised by Dr. Klára Esztó

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1.4. Richard Gerber
1.5. Charlene Boehm
1.6. Hulda Regehr Clark

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7. HUMAN PATHOGENIC FUNGAL INFECTIONS
7.1. Human Pathogenic Thermally Dimorphic Fungi
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7.1.2. Blastomycosis
7.1.3. Cryptococcosis
7.1.4. Histoplasmosis (Histoplasma capsulatum)

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Human Pathogenic Parasitic Infections

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8.2. Giardiasis (Giardia lamblia)
8.3. Malaria
8.4. Toxoplasmosis
8.5. Leishmaniasis (Sandfly Disease, Dum-Dum Fever, Kala azar)
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8.5.2. Leishmania Tropica Complex
8.5.3. Leishmania Mexicana Complex
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8.6.2. Trypanosomiasis in America (Chagas disease)
8.7. Babesiosis
8.8. Sarcosporidiosis (Sarcocystis)
8.9. Trichomoniasis
8.10. Dientamoebiasis
8.11. Cyclosporiasis

9. HUMAN PATHOGENIC HELMINTHS (WORMS)
9.1. Human Intestinal Roundworm Infections
9.1.1. Ascariasis (Ascaris lumbricoides)
9.1.2. Enterobiasis

9.1.3. Hookworm Infections (Ancylostoma duodenale and Necator americanus)
9.1.4. Strongyloidiasis
9.1.5. Trichuriasis
9.1.6. Trichinosis
9.1.7. Filariasis
9.1.7.1. Onchocerciasis („River Blindness")
9.1.7.2. Dirofilariasis
9.1.7.3. Mansonellosis (former Dipetalonemiasis)
9.1.7.4. Loiasis
9.1.8. Haemonchus Contortus
9.2. Human Intestinal Tapeworm Infections
9.2.1. Taeniasis and Cysticercosis (Taenia Saginata and Taenia Solium)
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9.2.3. Echinococcosis (Hydatid disease)
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9.3. Human Pathogenic Flukes
9.3.1. Schistosomiasis (Bilharziasis)
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9.3.3. Fluke Infections
9.3.4. Paragonimiasis
9.3.5. Clonorchiasis
9.3.6. Fascioliasis
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INFECTIONS OF SPECIAL ORGANS

10. DISORDERS OF MENTAL HEALTH, BRAIN AND NERVES
10.1. Depression
10.2. Bipolar Disorder
10.3. Delirium and Dementia
10.3.1. Alzheimer's Disease
10.3.2. Alzheimer's-like Dementia in Neuroborreliosis
10.4. Insomnia
10.5. Connatal Rubella Syndrome
10.6. Reye's Syndrome
10.7. Connatal Syndromes with Vascular Defects
10.8. Rett Syndrome
10.9. The Tuberous Sclerosis or Bourneville's Disease
10.10. Attention Deficit Disorder (ADD) and Attention Deficit Hyperactivity Disorder (ADHD, AD/HD)
10.11. PANDAS or TIC Disorders
10.12. Stress Syndrome
10.13. Anorexia Nervosa and Bulimia Nervosa
10.14. Autism
10.15. Migraine Syndrome in General
10.16. Headache and Migraine Syndrome
10.17. The Development of Autoimmune Brain Processes
10.18. Chronic Brain Diseases and Mycoplasma
10.19. Schizophrenia
10.20. Curious Neurological and Psychiatric Symptoms in Borreliosis

10.21. Infectious Emotional Crisis
10.22. Multiple Sclerosis
10.22.1. Schilder's Disease
10.23. Progressive Multifocal Leukoencephalopathy
10.24. Parkinson's Disease (Paralysis Agitans, Extrapyramidal Syndrome of Abnormal Posture, Involuntary Movement)
10.25. Amyotrophic Lateral Sclerosis (ALS, Lou Gehrig's Disease)
10.26. Prion Diseases (Mad Cow Disease, Creutzfeldt-Jakob Disease)
10.27. Cerebral Palsy
10.28. Meningitis
10.28.1. The Most Frequent Bacteria Causing Bacterial Meningitis
10.28.2. The Viral Meningitis Syndrome
10.28.3. Haemophilus Meningitis
10.28.4. Sleeping Sickness
10.28.5. Naegleria Fowleri Meningoencephalitis
10.28.6. Tropical Eosinophilic Meningitis
10.28.7. Chronic Meningitis
10.28.8. Other Types of Meningitis
10.28.9. Central Nervous System Candidiasis
10.29. Encephalitis Syndrome
10.30. Acute and Chronic Transverse Myelitis
10.31. Epilepsy
10.32. Poliomyelitis
10.33. Herpes Simplex Virus Infections in the Nervous System
10.34. Tropical Spastic Paraparesis
10.35. Encephalomyocarditis Viruses Infection
10.36. Sciatic Neuralgia, Lumbago, Ischias
10.37. Intercostal Neuralgia
10.38. Facial Palsy Syndrome (Bell's Palsy)
10.39. Diabetic Neuropathies
10.40. Polyneuropathy
10.41. Tardive Dyskinesia Forms
10.42. Foix-Alajouanine Syndrome

11. DISORDERS ASSOCIATED WITH INFECTIONS OF THE RESPIRATORY TRACT
11.1. The Common Cold
11.2. Scarlet Fever
11.3. Moraxella Catarrhalis Infections
11.4. Croup
11.5. Bronchitis
11.5.1. Swyer-James Syndrome
11.6. Pneumonia
11.6.1. Klebsiella Pneumonia
11.6.2. Legionnaire's Disease Pneumonia
11.6.3. Haemophilus Influenzae Pneumonia
11.6.4. Pneumonia Caused by Pseudomonas Species
11.6.5. Chlamydial Pneumonia and Psittacosis
11.6.6. Anthrax Pneumonia
11.6.7. Staphylococcal Pneumonia
11.6.8. Pneumococcal Pneumonia
11.6.9. Mycoplasmal Pneumonia

11.6.10. Viral Pneumonia
11.6.11. Fungal Pneumonia in General
11.6.12. Pneumocystis Pneumonia
11.6.13. H1N1 Influenza
11.7. Tuberculosis
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11.9. Parasite Cystic Diseases of the Lung
11.10. Paragonimiasis of the Lungs
11.11. Eosinophilic Pneumonia, Hypersensitivity Pneumonitis
11.12. Moldosis
11.13. Bronchopulmonary Aspergillosis
11.14. Cystic Fibrosis
11.15. Pulmonary Fibrosis
11.16. Pulmonary Wegener's Granulomatosis
11.17. Bronchiectasis
11.18. Pleurisy (Pleuritis)
11.19. Pulmonary Hypertension

12. HEART INFECTIONS AND BLOOD VESSEL INFECTIONS
12.1. Rheumatic Cardiac Fever
12.2. Endocarditis, Myocarditis and Pericarditis
12.3. Cardiobacterial Endocarditis
12.4. Endocarditis Caused by Veillonella
12.5. Mitral Valve Stenosis and Aortic Valve Stenosis
12.6. Cardiomyopathy
12.7. Angina Pectoris
12.8. Sudden Death caused by Coxsackie Viral Cardiac Infection
12.8.1. Sudden Infant's Death (SIDS)
12.8.2. Sudden Cardiac Syndrome
12.9. Brugada Syndrome
12.10. Temporal Arteritis (Giant Cell Arteritis)
12.11. Kawasaki Syndrome
12.12. Churg-Strauss Syndrome
12.13. Polyarteritis Nodosa
12.14. Leukocytoclastic Vasculitis
12.15. Hypertension
12.16. Atherosclerosis
12.17. Diabetic Angiopathy
12.18. Antiphospholipid Syndrome and Mycoplasma
12.19. Intravascular Coagulation Syndrome
12.20. Venous Disorders. Varicose Veins
12.21. Malignant Atrophic Papulosis (Degos Disease)
12.22. Thromboangiitis Obliterans (Buerger's Disease)

13. GASTROINTESTINAL DISORDERS ASSOCIATED WITH INFECTIONS
13.1. Esophageal Diseases
13.1.1. Gastroesophageal Reflux Disease
13.1.2. Mallory-Weiss Syndrome
13.2. Peptic Ulcer
13.3. Gastritis
13.4. Gastroenteritis
13.4.1. Salmonella Infections

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PUBLICATED ARTICLES BY DR. CSABA VÉRTESI

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INTRODUCTION

A brief summary of the radio frequency resonance method

The radio frequency resonance-method (RFR) uses the resonance of the radio frequency signal for examination and treatment. It is a common knowledge that only the high-power radio frequency signal sources (eg. the vicinity of broadcast transmitters) are responsible for the damaging of health, whereas the frequency generators used in medical practice do not pose any danger.

The RFR method examining the lower microorganisms uses the 200-1200 kHz band of the frequency generator (with low current intensity), the connecting peripheries – making use of the principle of interference – indicate and measure the resonant frequency bands of the microorganisms. It is made possible by the fact that the resonance band of viruses, bacteria and lower fungi is much lower than that of the living organisms having much more complicated DNA structures and being more highly organised. The electron running along the DNA chain functions like a resonant circuit. It is common knowledge that, in case of resonance, the resistance of the resonant circuit changes, and the voltage drops in the electric circuit in a measurable way.

It are mainly the traditional, registered medications that are used to destroy pathogens, but if they are not effective, the RFR technology can come into play in medical practice, always accompanied by traditional clinical and laboratory controls.

The giants of the Hungarian medical science (among them Imre Hajnal, Gyula Petrányi, Imre Magyar) – just like the representatives of major classical scientific circles of the world – have always emphasised that therapy should always be based on the principle of cause and effect, and the symptomatic treatment of patients is only allowed if the cause-and-effect principle cannot, for some reason, be used – either because the pathogen has not been identified, or because the effective medications capable of destroying the already identified pathogens are not available (eg. in case of certain viruses, antibiotic-resistant bacteria and fungi).

The radio frequency method can, by pin-pointing the resonating pathogens, identify the infection of the organism. These findings can be brought into connection with the patient's symptoms, and the accurate clinical diagnosis, based on traditional microbiological tests, on the results of clinical laboratory measurements as well as on traditional and modern medical examination methods.

The basis of the treatment with radio waves is their potency to penetrate into all tissues of the body, and that they are very likely capable to destroy microorganisms even at places where other, traditional methods fail to succeed, for example in the brain, where the otherwise effective antibiotics have difficulty to overcome the blood-brain barrier. Even if they do overcome this barrier, they cannot achieve appropriate bacteriostatic or bactericide concentrations.

As it is well-known, the primary structure of the DNA consists of two complementary twin strands, and constitutes a so-called double helix, which in turn creates a secondary structure. Then this double helix coils up forming a tube, the length of which– within one species – depends on the length of the DNA chain (in other words on the number of the basis pairs), whereas its width is value specific to the species. The "biological coil", developed depending on the secondary structure of the DNA, can resonate in the radio frequency space. Between the windings of the coil – owing to their similar charge – a

"distancing" force appears trying to push them apart. As the coil extends, its resonance frequency will drop, and thus will not resonate on the same frequency. As a result of its extension, a pulling force will come about in the "flexible biological coil" and if the extension is reversible, the specific frequency of the DNA will, triggered by the flexible pulling force, slowly return and will become able to resonate again to the same sinus signal. If the change in the DNA of the pathogen is irreversible – eg. the DNA chain get severed – the resonance can not be repeated, the affected virus, bacterium or fungus will get destroyed, and the relevant systems of the body will decompose the remains of the pathogen and remove them from the body.

Our book is trying to bridge the huge gap existing between the results of researches into the RFR method, published with many an error by other authors in the past six decades, and the up-to-date, advanced scientific medical thinking of today. Recently, the RFR-technology has become known as a method used by nature healers, rather than as a method of treatment in a traditional sense. At this point we have to call the attention of the readers to the fact that this book does not deal with some hazy, mystic bioresonance-method used by nature healers, but a modern application of up-to-date physics, where the RFR-technology makes it possible to study pathological processes in a different way.

Given the capabilities of the RFR-technology, we put the main emphasis on the prevention of diseases, thus first of all concerning tumor diseases, immune-autoimmune, vascular diseases when certain pathogens required but not sufficient for the development of the given illness can be identified and destroyed even years before the onset and full development of the disease. The opportunity to gain this is given by the fact that these chronic diseases arise as a result of infections caused by several pathogens, and will be manifested only if all kinds of the causative pathogens are already present. Until not all kinds of the pathogens required for the development of the disease are present, the disease will not appear, but the existing microorganisms can however already be destroyed. This will be discussed in details in the relevant chapters of the book.

Our book is meant for medical doctors who are open to modern methods, and we hope that it will influence the direction to be taken by medical science in the coming years.

The author of the book wishes to offer his thanks to all those who in the United States as well as in Hungary have in the past 15 years assisted him in his research, in creating the electrotechnical equipment for research, in compiling the research material, helped him to review, assess and prepare it for publication, and to those who have assisted his research with their constructive advices. First of all the author is indebted to his wife, who has always supported his work, and without whose unselfish activity this book would have never seen the light of day. The author would like to extend his thanks in particular to the following persons: Károly V. Lévay, Professor Emeritus (Venezuela, USA), the late Richard Rigler, hospital director (USA), János Mészáros, electric engineer (USA). Out of the Hungarian professionals to Péter Gergely professor, Klára Esztó, head doctor, Lajos Ábrahám, assistant professor, István Gazda and Mrs. Bodor Ágnes Sipos who have edited the book, and last but not least to those, who has made it possible to publish the book in such a beautiful format.

Dr. Csaba Vértesi

1. HISTORY OF THE ELECTRIC WAVE HEALING AND VIBRATION MEDICINE

The first pioneer of up-to-date electric wave healing was Royal R. Rife, who used this method for curing cancer in the years of 1920s. Ever since, this method developed as an alternative medicine method, and many researchers used it for healing different diseases. The prevailing attitude of dogmatic scepticism among medical authorities has kept most of these types of research underground. One can only hope that further adequate researches in the field of electromagnetic therapy of cancer and other diseases will one day recreate the incredible success of Royal Rife in ending the suffering of patients with cancer or other diseases throughout the world.

1.1. Royal Rife

Royal Rife, a brilliant researcher living in San Diego California, began his research for the cure of cancer and other illnesses in the 1920s. His first success was achieved with the Rife Microscope, an optical instrument unlike any other of its day, using an unusual combination of quartz optics. This microscope was capable of magnifying living cells, bacteria and even viruses. One of Rife's important discoveries was that every organism has its own resonant frequency, referred to by Rife as its Mortal Oscillatory Rate. Putting a live bacterial culture under the microscope, Rife could turn on a frequency device, known as the Rife Beam Ray producing an electromagnetic field tuned to this mortal frequency. All bacteria instantly stopped moving and died. Rife could destroy common bacteria, cure chronic infections, and even cure cancer by destroying the purported microbial causes of illnesses. By using the simple principle of vibratory resonance, he could destroy bacteria or viruses as easily as shattering a wine glass by producing its resonant note. Rife believed that cancer was caused by a virus or micro-organism, named BX and BY.

As all of the cancerous tumors disintegrated, leaving the animals healthy, Rife moved on to treat cancerous individuals. Modern microbiology does not accept Rife's theory of the BX and BY viruses.

Fortunately, there remained remnants of the original Rife's Beam, Ray and Rife's Universal Microscope being researched today, albeit behind closed doors. Researchers, studying Rife's method nowadays are careful to do their work in secret less they should befall the same fate as Rife. Before Rife died, he worked with fellow researcher Ed Crane in the 1960s to develop another system for healing cancer, which system was differing from the original Rife Beam Ray. The data of the original Rife Mortal Frequency and those of his followers' are well known. There were some preliminary anecdotal reports of success in treating cancer with these devices, but representatives of FDA have confiscated a number of the generators.

1.2. George Lakhovsky

In 1926, a Russian born engineer, George Lakhovsky developed in Paris an instrument named Multi-wave Oscillator that was able to cure cancer in plants and humans. Today, the multi-wave oscillator is banned by the FDA as an other example of medical quackery.

1.3. Wilhelm Reich

Wilhelm Reich built a specialized box known as an „orgone accumulator", a type of subtle energy capacitor. The orgone device stored a naturally occurring subtle life energy, named, by Reich, orgone. Using his device, a number of successfully treated cases of cancer were reported by Reich and other medical researchers. Similar to Royal Rife before him, Reich also hypothesized that cancer was due to a micro-organism. Reich named this micro-organism „T"-bacillus. The microbial or viral theory of cancer is one that has gained the

support of some modern cancer researchers. However, it is likely that an infection by a virus is not the only factor involved in cancer causation. Like Rife, Reich isolated T-bacilli from the cancer tissue. But, he found this same organism in healthy people, too. Reich hypothesized that psychological and emotional blocks in individuals set up metabolic and energetic changes in their bodily tissues and immune systems that eventually create the necessary environments for the cancer to begin to grow. He thought that cancer was due to this bio-energetically devitalized condition that somehow allows the formation of tumor cells in the body.
Researches of the Institute of Heart Math in Boulder Creek, California, (including Glen Rein and Roland McCarty), have discovered a fascinating phenomenon that tends to confirm the concept that love is a real healing energy with measurable physiological effects, even at DNA levels. Rein found that individuals who sat and meditated in a state of love, compassion, and caring actually generated greater coherence in their electrocardiogram pattern than those who were simply at rest or had discordant emotions. This coherence was only noted when conventional ECG recordings from the heart of a loving individual were fed into a computer that did Fourier transform analysis to show the underlying frequency patterns of the individual's naturally variable heart rate. Ever when love was present, spectral analysis of the electrocardiogram revealed a surprisingly ordered, cascading pattern of harmonic frequencies. Heart rhythms associated with love were characterized by a smooth, sinus wave like pattern. In this wave the peaks of frequency activity were separated in regular intervals. Most interestingly, the frequency peaks of this almost musical waveform were evenly separated by a proportionality factor identical to pi, the so called golden mean ratio of mathematics and architecture.

1.4. Richard Gerber

Richard Gerber, the author of' Vibrational Medicine, summarized the diagnostic and healing approaches to illness using energy in various forms and frequencies. This type of medicine will be the medicine of the future. Although this book was written by a physician and discusses various methods of healing, it is not meant to give specific recommendations of advice for the treatment of particular illnesses. This, being an examination of the mechanisms of a variety of alternative therapies, may hold promise to be an adjunctive treatment for conventional medical approaches.
He meant that „the Einsteinian model of medicine sees human beings as molecular, cellular systems which are in dynamic interplay with a variety of organizing, information-bearing energy fields".

1.5. Charlene Boehm

Charlene Boehm tried to find correlation between resonance and DNA, the first step in this way. The connection of the resonance frequency is only the secondary structure of DNA with no direct bearing on the number of the basis pairs. After a lengthy examination process, a U.S. patent was granted on October 9, 2007 (U.S. 7,280,874).

1.6. Hulda Regehr Clark

Hulda Regehr Clark discovered an electronic technique for scanning the human body. According to Clark, electricity can be used to eliminate bacteria, viruses and parasites within minutes and not days or weeks, like antibiotics do.
Clark dreams of a new world, i.e. that given sufficient voltage 5 to 10 volts, (lasting for seven minutes) using frequencies from 100 kHz to 500 kHz. any positively offset frequency will kill all bacteria, viruses and parasites simultaneously. Generating positive offset frequencies is the best way to kill all pathogens quickly. She wrote that every illness originates from two causes, i.e. parasites and pollutants.

The killing of parasites, the removal of pollutants and the clearing of gallstones and kidney stones from the body require a powerful combination of treatments.
This was a powerful recipe from Hulda Clark.
Hulda Clark has a useful table concerning the pathogen microorganisms: molds, viruses, bacteria, fungi and worms, in her book named „The Cure for all Diseases".
She was the first able to measure the pathological frequencies in the human body. Although there are many mistakes in her work, as f.i. her cancer hypothesis is very bizarre and nonsensical, she is, nevertheless, a most positive person.

2. ELECTRONIC CONCEPT-FORMATION

Hospitals nowadays utilize electric energy medicine in a variety of forms. In contemporary clinical settings, electromagnetic healing is the primary component of electric-medicine. Physical therapy departments utilize ultrasound devices to soothe aching muscles. They also use percutaneous electric nerve stimulation units in order to suppress pain electrically. In radiology departments they pulverize the patients' kidney stones painlessly with sound wave machines known as shock wave „litho-tripters". Orthopedic surgeons work with bone stimulators that send out pulsed electromagnetic fields to accelerate the healing of poorly knitting bone fractures. In operating rooms, surgeons use laser scalpels to remove diseased gall bladders. In the radiation oncology departments, cancer patients regularly undergo therapies with x-rays of high intensity. The x-ray and MRI are routine methods in the clinical medicine. I hope, RFR technology will be the next to develop into a conventional medical therapy. RFR technology can now be used to eliminate all known, and unknown bacteria, viruses, fungi and larvae of parasites, some even within a few minutes or a few hours, and not in days or weeks as in case of antimicrobial drugs. Furthermore there is no resistance, and all microorganisms are always sensitive to this method. It does not interfere with conventional medication, or drug treatment, is quite safe and has usually no intensive side effects. RFR method is very effective against infectious diseases. So that, the suddenly released bacterial toxins may cause side effects and transitional functional tissue damages. RFR is a step into a new medical world.

2.1. Sinusoidal radio frequency (RF) waves

A wave is generally an energy bearing self-propagating disturbance. Waves can be observed in different forms; for example mechanical, acoustic, electromagnetic, etc., We are here interested in electro-magnetic waves. The electromagnetic waves come usually repeatedly and periodically as many single waves after each other. The repetition rate is called frequency. The unit of frequency is Hertz (Hz), i.e. cycles per second. The number of Hz tells us how many times a wave repeats itself in one second. Electromagnetic waves are categorized according to their frequency or their wavelength. The wavelength can be calculated as the speed of light divided by frequency. The range is very wide and spans from 0 Hz to 10^{22} Hz or higher. The common, official names of the categories are:

Name of band	Abbr.	Frequency		range		Wavelength		
			(approx.)					
Very low frequency	VLF	0 kHz	...	30 kHz		∞	...	10 km
Low frequency	LF	30 kHz	...	300 kHz		10 km	...	1 km
Medium frequency	MF	300 kHz	...	3 MHz		1 km	...	100 m
High frequency	HF	3 MHz	...	30 MHz		100 m	...	10 m
Very high frequency	VHF	30 MHz	...	300 MHz		10 m	...	1 m
Ultra high frequency	UHF	300 MHz	...	3 GHz		1 m	...	10 cm
Super high frequency	SHF	3 GHz	...	30 GHz		10 cm	...	1 cm
Extremely high frequency	EHF	30 GHz	...	300 GHz		1 cm	...	1 mm

The name of the electromagnetic waves of higher frequencies (those above 300 GHz) are, as follows: infrared, visible light, UV, x-ray, Gamma ray, cosmic ray.

In this book we focus on the low frequency (LF) and the medium frequency (MF) range of the sinusoidal electromagnetic waves. (Note that this frequency range corresponds to the LW and MW bands of the AM radio broadcast.)

To describe the shape of the waves we usually use the sine function. The sine function is a well-known trigonometric function, that is periodic, repeating its value in multiples of 360° (or wavelength). Consequently, the sinusoidal (i.e. sine) wave is a form of periodic energy propagation.

2.2. Propagation of Waves, Energy Propagation, Electromagnetic Field

The RF energy can propagate two different ways: by radiation or by conduction. For example if a MF (medium-frequency) AM (amplitude modulated) broadcast signal has to be transmitted, the signal first propagates via a conductor to the antenna, but from the antenna to the receivers the energy propagates by radiation. From the antenna of the receiver again, the signal propagates by conduction. Consequently, the antenna works as a converter, as it converts the conducted signal to an electromagnetic field and vice-versa. The strength of the field can be calculated or measured, depending on the power radiated from the antenna. The strength of the field can be expressed in V/m, i.e. potential difference divided by the distance of the potential difference. If the energy is high enough, most remarkable works can be done with it (for example heating food in the microwave oven).

The radio frequency radiation can travel through many obstacles like air and other dielectrics (for example wood, plastics and other non-conductive materials). The higher the field strength the further the energy can travel overcoming various obstacles. A good conductor metal sheet (for example copper or aluminum) behaves as a virtual non-transparent obstacle for the RF waves. These materials are used for shielding. There are, however, materials with mixed properties, i.e. partially conductive media. For example the human body can be considered as a special, mixed material, since it is not a very good conductor, and the RF radiation can partially go through the body, while a part of the RF energy is absorbed by the body. This phenomenon is used for treatment of different body parts. For example, short wave (considered as high frequency (HF) irradiation is used locally to cure some rheumatic diseases, while gamma rays are used locally for the treatment of certain tumors.

2.3. Distortion and Modulation

2.3.1. Distortion

If the shape of a wave is not a clean sinusoidal shape, it still can be described as the sum of multiple sinusoidal waves. For example, if a flutist is playing a single note, we can hear a fairly clear sound, as the flute generates beside the fundamental waves still other acoustical waves, which have less of other frequencies than other instruments have. The wave of the flute is close to the clean sinusoidal signal. Other instruments (for example the violin) generate acoustical waves with several other frequencies, usually (but not exclusively) harmonics which are integer multiples of the base (or fundamental) wave. That is why we can differentiate between various instruments. Moreover, an experienced listener can distinguish between the flutes as well.

If we would like to generate a clean wave, we have to prevent the appearance of any other frequencies than the fundamental, or base ones, i.e., we have to generate a distortion free wave (or signal). In general, the distortion is a change in the waveform. If the wave does not have a clean, same sinusoidal shape for numerous cycles it gets distorted.

2.3.2. Modulation

One form of distortion is the modulation. If the modulation is not wanted we name it distortion, noise, jitter, or other similar words. If the modulation is generated on purpose, we do not use the term distortion, we simply call it modulation. Usually we generate two kinds of modulation, i.e. amplitude modulation and frequency modulation or their combination.

Amplitude modulation

If the frequency of the wave is constant and only the magnitude of the signal is modified, we call it an amplitude modulated (AM) signal. The base signal (otherwise called carrier) has usually a higher frequency than the modulating one. The magnitude of the amplitude modulation can be expressed in percentage, which shows the ratio between the amplitude of the carrier and the modulating signal. (For instance the MF (MW) or the HF (SW) AM broadcast has about 30% modulation.)

Frequency modulation

If the magnitude of the wave is constant and only the frequency of the signal is changing, we call it a frequency modulated (FM) signal. The carrier usually has a higher frequency than the modulating frequency. In this case, the momentary frequency is changing relative to the base (carrier) frequency. The magnitude of this change is called deviation. (For instance the FM broadcast has about 0.05% deviation.)

There is a special case of frequency modulation when the deviation is higher and the modulating frequency lower than if they are in regular communication. This is called sweeping. Sweeping is usually used for measuring various electronic circuits.

2.4. Resonance of Physical Systems

In general, every physical system has one or more natural vibration frequencies. These specific frequency values are characteristic concerning the system, and can, in most cases, be calculated or measured. For instance a string of a piano, or of a violin has several natural resonant frequencies, but the lowest frequency value is usually dominating when we strike the string. The other resonant frequencies can be heard when the string is struck in another (usually well defined) place. This resonance phenomena can be observed in case of many systems, for example mechanical, acoustical, electronic, molecular, atomic, etc.

The specific resonant frequencies of a system can be measured using the appropriate method. Naturally, the procedure depends on the type of the system. For instance an electronic circuit consisting of a capacitor and an inductor in series or in a parallel connection exhibits a resonance at a specific frequency. This frequency can be measured using suitable electronic measuring instruments and techniques.

2.5. Technical Background of Experiments

The RFR technique can detect viruses, bacteria, fungi and parasites. This method rests on radio electronic principles, using a sinusoidal radio frequency wave. It seems obvious, that a living human body broadcasts electric signals, just like a radio station, but over a wide band of frequencies and at a very-very low energy level, which has not been detected and measured until nowadays. Everything emits a characteristic range of frequencies. This electric signal has a very low amplitude and is of a sinusoidal character. In general, the more primitive the organism, the lower its bandwidth. Advanced animals have higher frequencies and a wider range than primitive organisms.

2.6. Experimental Technique

A new application of the RF field did emerge, when it was discovered that the microorganisms have specific electromagnetic resonant frequencies. It is well known that if an object with a specific resonant frequency receives an excessive energy with the same

frequency, it can be destroyed. For example a bridge can be destroyed when an army marches through with synchronized steps of the specific frequency of the bridge (unless a well designed damping mechanism prevents the development of higher amplitude vibrations). A similar phenomenon can be observed, if a microorganism (a pathogenic one) receives at its specific frequency an excessive electromagnetic energy in form of sinusoidal waves (which energy in this case is fairly low, but for the microorganism high enough). Thus it can be destroyed without harming other microorganisms with different specific frequencies. It is important to use a clean, sinusoidal wave with very low distortion (in other words the signal should not have other frequency components) in order to avoid the damage of other microorganisms or cells. (For instance, the square wave has beside the base or fundamental frequency many other frequency components.)

If we want to destroy a pathogen with electromagnetic waves, the pathogen should be placed in the electromagnetic field with the same frequency as the specific resonant frequency of the pathogen. In this case the pathogen interacts with the field. If the field is part of a special electronic circuitry, this interaction can be used for measuring the specific resonant frequency of the pathogen. This measurement can be done by watching the change of the amplitude of the field in the media where the pathogen is embedded, or by detecting the frequency interference caused by the pathogens. The measurements should be conducted very carefully, because the changes being small and easily disturbed by any surrounding EMI (electromagnetic interference). Since the pathogen is a living creature, and has at least one resonant frequency, it behaves as a living resonant electronic circuit. This means that the pathogen can slightly change its resonant frequency. It can absorb energy from the field and may emit some energy as well, which may cause interference. These facts are supported by experiments. Naturally,still more experiments and studies should be conducted to clarify many unknown details.

Since the resonant frequency of a pathogen can be measured, or, in some cases, calculated, a field with suitable parameters can be applied to destroy a pathogen. If the pathogen is embedded in a medium, for example in an animal or human body, the medium or the body should be placed in the electromagnetic field. The strength of the field should be as low as possible in order to avoid unknown side effects, but should high enough to be able to destroy the pathogens. Some pathogens can be destroyed more easily than others, depending on their characteristics, for instance their size. If the resonant part of the microorganism is relatively small, (like retroviruses or plasmids), it seems, that we have to apply a somewhat higher field strength than in case of a larger microorganism, in order to reach the destroying level of the potential difference across it. Likewise, if the pathogen is embedded in a somewhat better conductive environment behaving as a partial shield, we have to increase the field strength to compensate for the shield effect. The useful strength of the field in the media where the pathogens are is about 1-10 V/m_{pp}. To avoid the use of an extremely high field strength, the treatment should, in most cases, be repeated several times (two or three times a day for a few days) to ensure, that the previously hidden pathogens (surrounded by a better conductive medium) get drifted out from there by body fluids. The other important reason for repeating the treatment is that some not completely destroyed pathogens may recover in a few hours. If the instrumentation registers a certain resonance, i.e. indicates the presence of a pathogen, the treatment should be continued until the interaction can still be detected. But, because of the above mentioned facts, this does not mean that the treatment is completed. The treatment should be repeated until the day the interaction becomes negative. The course of one treatment can widely vary: from less than one minute to some hours, depending on the characteristics and the number of the pathogens.

If the treatment is ended although the interaction can still be detected, or if the field strength is too low, the pathogens are not yet eliminated. Moreover, even their multiplication may be stimulated. Perhaps the low level field strength helps their interaction.

The interaction that the capable instrumentation can detect is intermittent and not continuous. The time between the indications generally depends on the number of the microorganisms, and is lasting usually for seconds, in some cases tens of seconds.
Since the pathogens have radio frequency (RF) resonance it is not recommended to use DC offset. The DC offset may help to destroy the pathogens, but it is capable of destroying other components of the body by dissociation of all kinds of body fluids. The dissociation process generates toxic ions and free radicals. The best way to avoid any DC component is to have none or only one galvanic contact.

2.7. Instruments

The main part of the instrumentation is a good radio frequency (RF) generator. The generator should have a suitable frequency range (at least from 50 kHz to 1300 kHz). The magnitude of the output signal should be controlled so as to be from (close to) zero to at least about 20 V_{pp} (peak to peak) without load and with relatively low distortion. These types of generators usually have 50 ohm output impedance, though 600 ohm is acceptable as well. Since the pathogens are living creatures, they can slightly change their resonant frequencies. The quality factor of their resonating system is fairly good (in the range of 100), so that the accurate tuning is thus very important. But since they are changing their resonant frequencies, frequency modulation (FM) should be applied with a few kHz deviation from their nominal frequencies to make sure, that we can „catch them". The modulating frequency should be in the Hz range. For detection a special circuitry is necessary that can detect even very small changes of the field.
Preliminary experiments showed that using frequency and amplitude modulation (AM) together will give better results, nevertheless, many more studies should be conducted to give more detailed specifications. The data and methods in this book do not assume any AM modulations. After more extensive studies, a future publication will discuss this technique.

2.8. The Sensitivity of RFR Method

The RFR method is one of the most sensitive technical examination methods. The concentration limit of the RFR technique is 1-0, 1 femtogram per milliliter. (One femtogram= 10^{-15} grams.) The sensitivity of the method regarding bigger volumes can be increased, if the concentration remains the same.

2.9. Periodicity of Resonances

When, to treat different illnesses, Rife used the electromagnetic field, he used low frequency (100 Hz ... 2500 Hz) square waves as the amplitude modulating signal of a high frequency (about 27 MHz) carrier. Recent experiments show that resonance can be detected at these low frequencies also without the high frequency carrier, getting but very weak response. The experiment also showed that not only the low frequency, but the upper harmonics of these frequencies did induce responses as well, in an even more detectable manner. This phenomenon underlines the theory, mentioned in Chapter 2.3. that a system can have more than one resonant frequency, and that resonance can be induced not only by the base frequency in the system. Preliminary measurements did also show, that the resonance appeared when the frequency was doubled (2×, 4×, 8× ... 128×, 256× and so on). This might explain that low frequency square wave (which has lots of upper harmonics) worked well in Rife's experiments. When we go higher and higher in harmonics, we can reach the frequency range which Clark published as a well detectable one. Of course many more experiments should be conducted to get more detailed

informations of the behavior of the microorganisms when they are exposed to an electromagnetic field.
The application of square waves for curing proves thus to be efficient according to the good results published by several scientists. But notwithstanding the remarkable results, the frequency range of the applied square waves may become critical when we increase the frequency. As already mentioned, the square wave has many upper harmonics, and when these harmonics reach the MHz range with a significant amplitude, they may damage some organs of the body. To avoid this bad „side effect", the frequency of the applied square waves should be kept under about 50 kHz. Another disadvantage of the square wave treatment (especially in case of research projects) is that the harmonics can destroy some other microorganisms not aimed at originally. The use of sine wave signals secures the most selective detection and treatment.
Data of the amplitude and frequencies: use sine wave in capacitive coupling (non-galvanic) and square wave in galvanic contact.
Sine wave: amplitude from 5-40 V (peak to peak), 10 kHz-1300 kHz
Square wave: amplitude from 6-12 V, 300 Hz-40 kHz
The square wave frequency number is = sine wave frequency number divided by 128 or 265. (I prefer sine wave.)

2.10. The Way Leading to the Biological Balance

When should RFR method be applied? RFR method is necessary in case of a latent or manifest illness.
The microorganisms living in a host organism maintain a complicated balance with each other and with the immune system of the host's organism. In case of normal conditions the different bacteria, viruses and fungi normally prevent the multiplying of each other, resulting thus in a biological balance. It rarely occurs that some groups help each other in multiplying. The immune system of the host controls the homeostasis and reacts differently to each of the microorganisms. Some of the bacteria are useful and we live with them in symbiosis (see Chapter about the normal intestinal friendly flora). Moreover, the presence of these friendly bacteria inhibits the settling and growth of other, pathogen virulent strains, against which the immune system is not tolerant, showing a continuous active defense against them. It would be damaging to eliminate all the friendly bacteria with RFR method. Thus one should never extirpate all resonant microorganisms. If the balance – which is the basic state of health – is disturbed, illness will occur. If the most virulent microorganism multiplies excessively, a clinical illness will be developing, in case of which we have to determine with the help of the clinical symptoms and laboratory methods, which microorganism is multiplying excessively causing sickness, then we have to find the resonant frequencies. The fact should not be neglected that there can possibly be a friendly microorganism with the same resonant frequency that will be destroyed; so that it is necessary to reintroduce them as is the custom after an antibiotic treatment. In the case of certain immunodeficiency, the causative bacteria or even the friendly ones can produce a disease or clinical symptoms; the cases of which should be considered separately.
We always have to extirpate the causing microorganism by RFR method and at the same time try to maintain the sane biological balance. One of the big advantages of the RFR method is that as it is greatly selective it is able to extirpate even one single strain of virus or bacterium. But it may happen that another causative microorganism will get increased which previously was suppressed by an earlier one. It frequently occurs that a broadband curing increases the presence of one or several fungi. Do never forget that the normal flora gives a defense against a lot of illness-causing microorganisms and that during the RFR method we should try to reach the biological balance.

2.11. Safety Requirements

One lives in an environment of high-voltage transmission lines, microwave ovens, cathode ray tubes, and other powerful electrical devices, which probably have negative biological effects which are not determined as yet. Recent studies have hinted at an increased incidence of childhood cancers in children of families living close to high-voltage transmission lines. The unseen subtle toxicities of harmful environmental substances, the established safety levels of various chemical and mineral pollutants, background radioactivity, and electromagnetic radiation are solely based on the measurement of gross negative biological effects, i.e. the development of cancer and of certain fetal abnormalities.

All living systems exist within a planetary energy field. Similarly, there are specialized energy rhythms within all organisms entrained by living in the field of natural energy oscillations of he Earth. Stressful effects upon human health caused by abnormal fields associated with a particular geographical region, are referred to as geopathic stresses. The geopathic field probably acts in concert with a variety of other predisposing factors including diet, genetics, environmental carcinogens, viruses, bacteria, fungi, abnormal electromagnetic radiation exposure, as well as the subtle-energy factors which affect the general vitality and the immune competence. The interface which regulates the flow of these higher energies into the physical framework is made up of the chakra-nadi system and the acupuncture meridian system, working in conjunction with the biocrystalline and bioelectronic networks of the body. In this energy system an integrated RFR experiment is needed. Rife's plasma tube technology should be rejected, as it can, similarly to soft x-ray radiation, be toxic. Its first symptome is fatigue, its prolonged application can lead to the damage of the patient's immune system, can be the cause of the dysfuntion of the parenchymal organs, premature aging, greying, greyish color of the skin, loosening of the teeth, temporary or definite impotence, anemia and certain malignant illnesses.

RFR method is a new technology of the experimental healing technique, still in an experimental state. For ten years we collected data of toxicology, but these are not enough by far. The American Food and Drug Administration does not permit the RFR method. Several alternative systems were using this method and by which experience the present knowledge was developed. Be wise in choosing candidates for experimental healing effect. The range of frequencies in primitive microorganisms is lower than the human resonant frequency range. The resonance range of molds, viruses, bacteria and worms are from 50 kHz to 1500 kHz. The human range is from 1500 kHz to 11-12 MHz. The resonance range of several microorganisms such as plasmids of the bacteria, can seldom be found in the human range. In the human resonance frequency area no treatment should be done. Do not use higher than 1300 kHz. To treat in the range of the human frequency is very dangerous!

3. THE STRUCTURE OF THE WORK

The first part of our book approaches the question how the radio frequency resonance method can be used in medical practice from the aspect of the that microorganisms. In this part we will provide a list of the characteristics of the pathogens and their possible frequency range. The second part will discuss the relationship we have identified between the diseases and the pathogens in the background from the aspect of the diseases based on the numerous measurements we have made in the past decades.

3.1. The Role of the RFR-method in Identifying the Etiology and Pathogenesis of Diseases

With the RFR-technology we were trying to create a method which helps to identify the root of the diseases, that is which makes it possible to identify the pathogen(s) that is (are) specific to a given disease, and which can be destroyed with the RFR-method even if they are viruses, fungi, or antibiotic resistant bacteria. **For the first time in the history of medical science, the RFR-method has provided an opportunity to cure chronic diseases (such as cardio-vascular diseases, allergies, autoimmune diseases, diabetes, and different tumours) with causal therapy.**

Pathological processes have a well-definable dynamism, which I would like to illustrate with but one example. The carcinogenic microorganisms create from healthy cells tumor cells with slow transition, which – with further modification – create tumors consisting of less and less differentiated tissues, getting closer and closer to the stem cell type, more and more fast growing, ending often in sarcoma-like tumour cell formations. Such is, f.i., the phenomenon when the lymphoid tissues transform, step by step, into reticulosarcoma. Parallelly with the step by step transformation it may occur that also itself the virus responsible for the process undergoes mutation, and as a result of this, its resonance frequency will change, too.

It is well-known for a long time past that if resting differentiated cells get incorporated with active viruses, they start to divide and differentiate. For the first time biologists identified this phenomenon in tissue cultures in connection with the so-called Sendai viruses, which are similar to flu viruses. The infectious viruses somehow modify the surface of the cell, thus making them prone to merge. Soon after the fusion with the virus, the DNA synthesis starts in the previously stationary nucleus.

For instance, a tumor will start to develop when a certain oncogenic virus f.i. a Human Papilloma Virus (HPV) gets into an epidermic cell, in the vicinity of which, there are one or more so-called Colonia Stimulating Factor (CSF)-producing microorganisms at the same time. Colonia stimulating factors accelerate the changed, primitive metabolism and the DNA synthesis of the HPV-infected cells as well as the multiplication of the tumor cells. The immune system can still eliminate the resulted tumor cells, as the immune reactions target the tumor specific antigens on the surface of the cancer cells. On becoming a tumor cell, the cell displays such a new surface configuration, which is considered alien by the immune system of the host's organism, acts against it, destroys and breaks it down. If, however, the immune system fails to respond to the tumor cells, or any of the steps of the response is faulty, the tumor cells will survive.

It is known that in the body capable of immune response the mutated cells will be recognized by the so-called patrol-cells, which will mark the transformed cell surfaces with

iodine and peroxide, while, subsequently, the different eliminating mechanisms will destroy these cells, thus preventing them to become tumor cells. If, however, just one step of this mechanism fails to work, the tumor cells will multiply so that the tumor becomes perceptible.

There may be several factors responsible for the failure of an effective immune response among which factors the infection with immune suppressive viruses (eg. HTLV, HBLV, EBV) and/or mycoplasma,as well as any hereditary or acquired damage to the immune system is of paramount importance.

In the majority of cases the administration of immune stimulants to the patient fighting against cancer does not work, as the stimulation of certain parts of the immune system triggers (via "feedback") the inhibition of other sub processes, so that the immune balance will, most certainly, remain at a level insufficient to eliminate the immune suppressive microorganisms. **It seems today that the best way to overcome a tumor danger is to destroy the microorganisms (mainly viruses) that inhibit the working of the immune system,** which .can be effectively done also with the **RFR-method.**

3.2. The Opportunities Offered by the RFR-method to Clarify the Role of Virus Components and Other Microorganisms in the Process of Tumor Formation

We will seek an answer to the question which virus components and how they can trigger the specific DNA replicating genes of higher organisms, and how viruses and other microorganisms contribute to the process of tumor formation.

Biologists have always known that one of the basic characteristics of the existence of multicellular organisms is the regulation of the cell division. A differentiated cell has during its life span the opportunity to divide or to perform certain functions and to produce proteins or other materials, which – in their own way – will contribute to the sustenance of the person's homeostasis. **The different tumor diseases arise from different differentiated cells via a Human Papilloma Viral infection (HPV).** (Today we know some 176 types the complete DNA sequences of which have already been identified.) Though the developed cancer cells infected with viruses show many of the morphological and functional features of the normal precursor cells, they, nevertheless, lack the normal regulating mechanism that prevents excessive cell division. This will be nullified by the command of the virus to devide. The carcinogenic viruses can be identified and destroyed with the RFR method. After the destruction of the virus, the proteolytic enzymes will break down the mutated cells, and, after destroying the other microorganisms inhibiting the effective immune response, the organism itself will also be able to destroy a certain amount of tumor cells.

The viral origin of tumor cell formation is even nowadays (2009) not yet accepted. It, particularly, had not been accepted when I wrote my book "Infectious Disease Treatment with Radio Frequency Resonance" published in 2004. Since then researchers in New York have proved the "primitive retrovirus" origin of breast tumors, and today an antiviral vaccine is available to prevent cervical cancer. Despite of this, the viral origin of tumors is not widely accepted by the medical community, as it has not been completely proven. An additional twist to the issue is given by the fact that an organism can coexist with the tumor-viral infection even for decades without developing a noticeable tumor. This **means**

that several factors have to coexist for the mechanism of tumor-formation! In addition to HPV, immune suppressive agents (such as Human T-cell and B-cell Lymphotropic Viruses, Mycoplasma fermentans, etc.) inhibiting the effective antitumor activity of the immune system always play a role. I wish to prove the above said statement with my book as well.

In certain cases the RFR-technology can provide but limited therapeutic opportunities. The hindrance of its application is f.i. given by the fact that the destruction of a tumor spreading all over a complete bowel wall section can cause bowel perforation, or that the liquefaction of a strongly vascularized tumor can result in intense bleeding. In case of large brain tumours an other limit can be the brain oedema caused by the destruction of the tumor, which oedema increasing the pressure in the brain, can cause a so-called brain herniation. An inoperable, final stage patient should not at all be treated with RFR-technology.

3.3. Conclusions to be Drawn from the Research Conducted Hitherto into the RFR-method for Chronic Diseases and for the Development and Treatment of Pathological Autoimmune Processes

The following important problem is the development and treatment of the so-called "chronic diseases", such as, f.i. Amyotrophic Lateral Sclerosis (ALS), SLE, RA, the chronic diseases of the cardio-vascular system, vasoconstriction, metabolic diseases, such as diabetes, etc. The RFR-technology provides the opportunity to shed new light on these important issues to explain them in a novel way, and to cure them within certain limits.

This technology will enable us to identify and destroy the pathogens present in the above mentioned diseases, something we have not been able to accomplish till now. The most important feature of these diseases is that they are caused by the simultaneous presence of several pathogens. A significant portion of them are viruses, which cannot be destroyed in the traditional way (with medication). One or two kinds of pathogens among them (f.i *Mycoplasma species, Human T-cell and B-cell Lymphotropic Viruses, Epstein-Barr Viruses*, etc.) block the immune system. It being difficult or even impossible to destroy these pathogens, the disease will become protracting and will remain during the whole life span of the patient.

It is often the case that beside *Mycoplasma pneumoniae* and *Mycoplasma genitalium*, the but recently identified *Mycoplasma fermentans* also contributes to the development of chronic diseases and pathological autoimmune processes. The *Mycoplasma fermentans* has no characteristics of its own (is only a 'co-factor'), meaning that the manifestation of the disease to which it contributes always depends on the associated other pathogens. The **Mycoplasma fermentans** can be identified almost in case of all chronic diseases, and the other pathogens present in the body cannot be destroyed till the Mycoplasma fermentans inhibits the immune protection of the host's organism. Our numerous measurements verify that this pathogen **directly participates in the development of autoimmune diseases.** We will give a detailed treatment pattern as to this in the relevant chapter. The **Human T-cell and B-cell Lymphotropic Viruses** play a similar role in the development of chronic and autoimmune diseases. These **viruses** can today be only eliminated from the body with RFR-technology, and this elimination can lead to recovery in cases where the above mentioned agents play an inhibiting role. In case of autoimmune diseases the killing of Mycoplasma fermentans can cause severe symptoms and a temporary worsening of the general condition or of the autoimmune symptoms (similarly to the Jarisch-Herxheimer

reaction when treating syphilis). This phenomenon can often be observed in case of sclerosis multiplex, too.

Further examples concerning this theme are the Coxsackie and ECHO viruses – both well-known since a long time from the clinical picture of heart diseases - which can cause chronic heart diseases, which so far could only be treated symptomatically. The RFR-method makes it possible to show the underlying reason for the **chronic heart and vascular diseases and syndromes** (f.i. chronic myocarditis, pericarditis, chronic myocardiopathy, subendocardial fibroelastosis, etc.) caused by Coxsackie and ECHO viruses, as well as to identify and eliminate them completely.

The RFR-method can save lives being in the early stages of aseptic (viral) meningitis, or suffering from similar viral diseases. More over, the RFR method can play a significant role in the treatment of tropical diseases as well.

The demonstation of the viral origin of diabetes mellitus constitutes a separate chapter in the history of medical science. This kind of origin can, in certain cases, even promise recovery. Lerner already in 1977 mentioned in his book that diabetes mellitus can be caused by Coxsackie virus B4. Nevertheless, its etiology is much more complex. Though the frequencies characteristic of the Coxsackie virus B4 can always be generated in diabetic patients, the development of this disease (just like that of all chronic diseases) is extremely complex, and is the result of the simultaneous presence of several pathogens. The Coxsackie virus is never the only factor in diabetes, meaning that this virus cannot cause diabetes on its own. The Coxsackie virus B4 can be found as one of a set of pathogens, generally together with Mycoplasma fermentans and HTLV specieses. Diabetes mellitus displays, however, even such characteristic frequencies, which cannot be linked to any identified pathogen as yet. In case of diabetes mellitus the Coxsackie viruses B4 live in the beta cells of the islets of Langerhans, and destroy them. As a result of the natural "feedback mechanism" the cells differentiating from reserve cells, after undergoing a phase of maturing and thus almost becoming beta cells, will get infected with these viruses and be destroyed, partly by the viruses and partly by the triggered autoimmune processes (see autoimmune diabetes mellitus type 1). This regeneration process regulated by the "feedback" lasts until the regeneration capability of the islets are exhausted. A slow regenerative process, which result in the arising of new functioning beta cells can only occur if the Coxsackie viruses, Mycoplasma fermentans and other pathogen species signalizing the diabetes frequencies are eliminated. The presence of the mycoplasma prevents the destroying of the Coxsackie virus, so that at first the mycoplasma shall be eliminated. After the elimination of the pathogens it takes a long time for the autoimmune process to peter out.

In the past decade the increase of the number of **antibiotic-resistant bacterium strains** could be experienced, as well as their spreading all over the world. Today we already know Mycobacterium tuberculosis strains that do not respond to antibiotics at all, causing thus grave danger. Certain "docile strains" mutated with gene replacement learn how to break down antibiotics, thus rendering them ineffective. Our increasing knowledge shows that certain bacteria can, unfortunately, transmit this aforesaid acquired capability. The RFR-method, however, is effective even concerning these docile strains. Borrelia bacteria can become resistant to Doxycyclin, changing their resonant frequency values, too, resonating thus at 6–8 kHz higher than before. This phenomenon shows that the resonance of the non-resistant strains differs from that of the resistant strains. Even these strains can be eliminated at this higher resonance. Experience shows that the formation of the antibiotic resistance goes hand in hand with a shift towards a somewhat higher resonant frequency.

In the past decades some diseases have completely disappeared or have been completely eradicated as a result of vaccination and preventive measures; for example hemorrhagic smallpox, plague, and certain other illnesses causing fear all over the world. However, there have appeared new diseases, mainly of viral origin, such as the HIV infection and the diseases caused by the members of the SV virus group. For the time being traditional medicine does not provide protection against them, but we trust that the RFR-method will offer good results even in this field.

Some rarely occuring diseases that have been known since a long time, have become so widespread that they are almost endemic diseases. Its typical example is **Borreliosis, the Lyme disease**. Its treatment is greatly problematic when the pathogen penetrates the central nervous system, causing there grave personality disorders and other symptoms. These patients can be but hardly healed with traditional medicines because, as I have already mentioned above, it is difficult for the antibiotics to get into the brain and reach the pathogens in the nerve cells. It means that in many cases the required medicine is not available, and/or its effective concentration cannot be achieved.

The Borrelia B. s. l. family has several well-known plasmids, which enable the development of species with new and new antigenity and possible antibiotic resistance. Its vegetative, so-called gemma (or cystic) form does not show any metabolism, and can in no way be killed with antibiotics. We hope that the human-clinical tests of the RFR-technology will offer a positive answer to these problems as well. It is expected that in order to achieve complete and final recovery it would be practical to combine antibiotic treatment with the RFR-method.

RFR-measurements done **in case of grave allergy** indicate that the immune system is, in these cases, usually infected with Mycoplasma fermentans and/or Human T and/or B limfocitotropic viruses, altering thus the immune response in a pathologic way. After destructing these pathogens the proneness to allergy will be reduced, or may, later on, when the pathological processes have run their course, even come to an end.

3.4. The Limits of the Use of the RFR-method

The RFR-technology can only be used for diagnostic purposes (to identify bacteria or viruses) in a limited extent, as several microorganisms can resonate at the same frequency, so that one can decide the fact which one of the microorganisms causes the given resonance, solely after checking it against the clinical picture. After aquireing an appropriate practice, one can well exclude and/or verify the presence of certain pathogens with the RFR-technology.

We have learned how to coexist with the microorganisms living in our environment, and have developed such protective proteins whose codes are stored in the DNA. Mutations, however, can do damage to the genetic programmes, so this specific protection can weaken or come to an end, and we can become predisposed to a pathogen or a disease. We have been able to observe long since that certain persons are more predisposed to certain diseases than others, (are predestined), meaning that they fall ill with the given disease much more easily than others. A typical example of this is the Herpes viral infection, which some people do not catch or do not come down with, or only catch in case of a large number of germ cells. Even if they do carry the virus, it does not cause them symptoms, while others – after their infection has been eliminated with the RFR-technology – can fall

ill again and again even if they are only exposed to but a small number of viruses. As it seems to be the case, the "repair" of the above-mentioned systems is done by so-called "repair" systems, whose operation cannot be boosted. However, there is no need to boost them, solely the factors inhibiting their effective operation have to be eliminated. Such inhibiting factors can be, f.i. viruses, which can be eliminated with the RFR-method.
The above examples have only been chosen at random showing the possible scope of the medical application of the new RFR-technology. At random, as the number of its possible applications is infinitely huge. I do hope that **the RFR-technology, just like the MRI method, will be able to help the development of medicine in the coming years.**

INFECTIOUS DISEASES IN GENERAL

4. BIOLOGICAL RESPONSE

The vast majority of human and animal diseases of known etiology are produced by living agents: viruses, rickettsias, mycoplasma, bacteria, fungi, protozoa, or metazoa (nematodes and ectoparasites). Although there do remain important exceptions, infectious disease as a class is more easily prevented and more effectively cured than any other major group of disorders. Despite the virtual elimination of certain infectious diseases and the significant reduction in the morbidity and mortality of many a man, this does not mean, that men became free of infections. In fact, the total percentage of human illnesses produced by microbial parasites has decreased only modestly, primarily through smallpox and malaria control and improved health care in developing countries. As certain specific microbial infections have been controlled, others have emerged as troublesome therapeutical and epidemiological problems.
The interaction between microorganism and man resulting in infection and disease is complex.

4.1. Microbiology of Infectious Diseases

4.1.1. General aspects of Microbiology of Infectious Diseases

What kind of relationship does exist between the host and the microorganisms? Relationships can occur between a microorganism and its human or animal host as follows:

1. Symbiotic, in which both the microorganism and the host benefit.
2. Commensalic, in which the microorganism gains while the host does not suffer any harm.
3. Semiparasitic, in which the microorganism gains as some of its effects are beneficial, while others harm in the host's organism.
4. Parasitic, in which the microorganism gains but the host is harmed.

Bacteria and fungi account for most microorganisms that have symbiotic and commensalic relationships. The only places of symbiotic relationship with microbas are on the skin and the mucosa.
There can be three sorts of microorganism on the body surfaces:

1. pathogen parasites, the presence of which means illness (obligate pathogen) or in case of relative resistance or immunity a carrier state (facultative pathogen)
2. apathogen parasites, which could build the resident or commersalic flora of the surfaces permanently or transiently.
3. saprophytes

A healthy person lives in harmony with the normal microbial flora, which establishes itself on certain particular body surfaces. The normal microbiological flora usually occupying a part of the skin or the mucosa is called the resident or friendly flora.
Colonization is a microbiological phenomenon in which the microorganism establishes and multiplies itself permanently on the body surface, while the host shows no marked reaction. Rather than causing disease, the resident flora with its colonization usually protects the body against disease-causing organisms, this phenomenon is called colonizational resistance. These microorganisms living in colonization can produce Colony Stimulating Factor (CSF) citokines. (Its importance see in Chapter 26.1.6.) If disturbed, the

friendly flora promptly regenerates itself. Microorganisms that colonize the host for hours or weeks, but don't establish themselves permanently, are called transient flora.
Infection is also a microbiological phenomenon, in this case the host gives a marked answer to a pathogen. The characteristics of pathogeny are, over and above adhesion and colonization, invasiveness, infectivity and virulency. Infections can be (even those lasting for a long time) asymptomatic or symptomatic, namely an illness.
The virulence of a microorganism, or its degree of pathogenicity, should be distinguished from its invasiveness, or the ability to spread and disseminate in the body. For example, Clostridium tetani is pathogenic and, by virtue of its exotoxin, highly virulent, but almost completely noninvasive.
Numerous types of commensalic microorganisms have the potency to cause disease. Many of these live on the skin, in the mouth, in the airways, in the intestines, and in the genitalia without causing any pathological process. Every colonized state has the potency to cause infection. There are three factors possessing the ability of infectiveness of the colonized microorganism:

1. the size of inoculum
2. the virulency of the microorganism
3. the effectiveness of the host's defence system

In most cases the biological linkage begins when microorganisms adhere to a host's cells. Adherence is a very specific process, involving lock and key connections between the human cell and the microorganism.

4.1.2. Special Aspects of the Microbiology of Infectious Diseases

4.1.2.1. Competitive Inhibition

Sometimes microorganisms from within the resident flora can become invasive, causing disease in people whose defence mechanisms are disrupted.
One bacterium can prevent the multiplication of another, and the most appropriate one can inhibit the breeding of others. If the inhibitor bacterium is destroyed, the formerly suppressed bacterium can multiply and establish itself. These emergent microorganisms can then attack and cause new infections. The bile duct infection is a typical example of this, when six to eight other types of bacteria and fungi surface after the primarily dominant microorganism has been eliminated.

4.1.2.2. Asymptomatic Chronic Infections

The human organism can live for years with pathogen viruses or other microorganisms without developing a disease, but if and when, perhaps years later, another particular pathogen surfaces, a disease is more likely to develop.
The onset of these infections can be marked by high grade fever, lymphadenomegaly or other typical symptoms of infection (e.g. EBV infection); or it can even happen without producing any outward signs of their presence (e.g. HTLVs, Retroviruses). After several years of latency, such infections can turn into a disease.
This book offers an explanation for diseases, which are probably caused by viral infections passed through generations in the families, which may surface unexpectedly when the immune system is weakening. In the following chapters I will present a new approach to the therapy for such illnesses. The aim of my research has been to discover an effective treatment of ailments caused by infectious organisms, both recently-acquired and of long duration, contracted by direct exposure or by transgenerational passage. This book

suggests that the study of this method may lead to a new solution for the treatment of infectious diseases.

4.1.2.3. Immunomodulatory Activity of Infective Agents

A large number of microorganisms or their product can influence on an aspecific way the responsiveness of the immune system to heterolog antigens. This immunomodulative effect could be both stimulative or suppressive.

Immunosuppressive viruses are e.g. the HIV, HTLV viruses, Morbilli, Adenoviruses; immunosuppressive bacteria are e.g. the Mycoplasmas, Staphylococcus pyogenes, Treponema pallidum pallidum, Borrelia Burgdorferi sensu lato, Mycobacterium leprae; immunosuppressive protozoa: the Plasmodium malariae, Toxoplasma gondii; immunosuppressive metazoa: the Trichinella spiralis etc.

An Immunostimulative activity have for example the Propionibacterium acnes, Mycobacteria, Brucella and NDV virus.

An infection with a microorganism of immunosuppressive activity can lead to coinfections, to infections caused by an opportunistic pathogen or to a tumorous transformation of the host's cells.

4.1.3. New presuppositions

In the infected area of a chronic illness there occures very frequently a colonization of different pathogens, e.g. viruses, bacteria or fungi, which together can provoke one specific illness. The host's organism cannot overcome the illness caused by a combined infection like this. Such a concomittant infection can inhibit the host's immune system similarly as a pathogen with immunosuppressive potency would. The datas of my studies offer a well-founded suspicion, that e.g. the Alzheimer's disease is caused by a combination of viruses and fungi, the Parkinson's disease and Multiple Sclerosis being the result of a combined attack by bacteria and viruses. Moreover, in most human tumors a multilateral illness caused by several viruses and/or bacteria and/or mycoplasma is always present. Similarly, in the rheumatic process there is yet another combined infection present.

These specific illnesses mentioned above can only develop in instances when all necessary invading components are being present.

4.2. Biological Response of the Host in Case of an Infection

An individual who is susceptible to infections is called a compromised host. This susceptibility may occur due to an impairment in the host-defence mechanisms (e.g. damage of mucosal or epithelial barriers; in tissue vascular supply, in genetic or other factors of innate and acquired immunity), or because the normal surface microbial flora had been changed by antibiotics or any other therapy, or because a foreign body had been implanted, etc.

The biological response can be either:

- effective and appropriate: the host's organism overcomes the bacterial attack;
- partly ineffective but appropriate: when a small number of microorganisms survives the response of the host,
- partly effective and unappropriate: which may lead to an autoimmune process or to allergy
- ineffective but appropriate: when microorganisms survives within the host.

4.2.1. Innate and Acquired Immunity

The body's defence mechanisms against infections include natural barriers, such as the skin and mucosa; nonspecific mechanisms and cells of innate immunity (e.g. complement system, macrophages, NK cells, etc.; their receptors, their mediators, e.g. citokines and their effects such as fever, fagocytosis, etc.) and specific mechanisms of acquired immunity (cellular and humoral, with antibodies). The latter is characterized by its antigen specificity, diversity, selectivity, sensitivity and immune memory.
An acute inflammation at the onset of an infection is an example of the nonspecific defence mechanism.
Certain infections cause changes in the blood, heart, lungs, brain, kidneys, liver, intestines or other organs. Due to the body's defence system against infection the white blood cell count usually becomes elevated. The number of neutrophils increases first. If an infection persists, the number of monocytes, lymphocytes, and macrophage cells will increase. In case of a viral infection as well as in a chronic bacterial infection the number of lympho-monocytes multiplies. The number of yet another type of white blood cells, the eosinophils, will increase with allergic reaction and parasitic infections.
If an infection develops, the immune system will come into action. The immune system is characterized by its immune cells and soluble substances. The most important cells of the immune system are the different types of white blood cells (granulocytes, T- and B-cells, monocytes, eosinophils), NK-cells and macrophages; the most important soluble substances are the antibodies, complements, and cytokines. Some soluble substances act as messengers called to attract and activate other cells. The Major Histocompatibility Complex molecule plays the most significant role in the identification of a person's own versus foreign tissue, as well as in the antigen presentation. When T-lymphocytes are triggered through their T-cell receptors, they will produce several cytokines which help to recruit other lymphocytes, amplifying thus the immune response. Some cytokines can also activate a nonspecific immune response. Cytokines therefore bridge the innate and acquired immune processes.
Usually, if an organism gets through the body's natural barriers, the nonspecific (and specific) defence mechanisms will destroy it before it multiplies.

4.2.2. Insufficient, Inadequate or Pathological Immune Response

4.2.2.1. Bacteremia, Sepsis and Septic Shock

If an infection persists and the response of the host's organism is insufficient, from the local infection microbas spread into the bloodstream. Invasion into the bloodstream causes **bacteremia or viremia.** In consequence of the organotropism of some bacteria there could occur after a bacteremia a chronic local inflammation in the tissues (e.g. osteomyelitis, abscessus, metastatic bacteremia, pyemia)
If the immune system can not kill the bacteria in the bloodstream and could not stop their multiplication, septicemy will develop.
Sepsis, also known as Systemic Inflammatory Response Syndrome (SIRS), is a syndrome in which from a local microbal focus bacteria come temporarily or permanently into the bloodstream. This is a serious medical condition caused by the body's response to an infection.
Septic shock is a condition caused by an infection in the bloodstream (sepsis) in which blood pressure falls dangerously low and in several organs malfunction will be present due to inadequate blood flow. It is usually characterized by bacteremia, and most often caused by Gram-negative enteric bacilli: Enterobacteriaceae, Pseudomonas, etc. Gram-negative bacteremia is caused by infections with a primary focus, usually of the genitourinary, biliary, or gastrointestinal tract. Septic shock may be associated also with Gram-positive

infections, notably those due to Pneumococci or Streptococci. Gram-negative anaerobic bacteremia produced by the bacteroides species can be also a precursor of septic shock, in which situation the syndrome is less fulminating than with aerobic gramnegative bacilli.
The **shock syndrome** is not due to the invasion of the bloodstream by bacteria, far rather due to cytokines and to the releasing of endotoxin (the lipopolysaccharide moiety of the microba's cell wall) into the circulatory system. Endotoxin exerts its major effects on small blood vessels. It causes the blood vessels to dilate, which results in the drop of the blood pressure, causing reduced blood flow to vital organs – particularly the kidneys and brain. This reduction occurs despite the attempts of the body to compensate by increasing both the heart rate and the volume of the blood pumped. The toxins and the increased work of pumping weaken the heart, resulting in a poorer blood flow to vital organs causing hypoxia or anoxia in their tissues. The walls of the blood vessels will leak, allowing fluid to escape from the bloodstream into tissues causing their swelling. Leakage and swelling in the lungs cause difficulty in breathing (respiratory distress). Myocardial failure and coma are late and often terminal manifestations of the shock syndrome.
Predisposing factors can include: people who have a chronic disease (for example diabetes mellitus, cirrhosis), different types of tumors (such as lymphoma, leukaemia or disseminated viral carcinoma), as well as other different types of immunosuppressive states triggered by surgical procedures, burns, or antecedent infections in the urinary, biliary, or gastrointestinal tracts, or severe viral infection e.g. AIDS or HTLVs. Most patients with Gram-negative sepsis are elderly males, but neonates and pregnant women are also prone to develop this syndrome.

4.2.2.2. Allergic Reaction as a Pathological Outcome in Infectious Diseases

Allergy is a pathological protective immune reaction in which an antigen (allergen) provokes the immune system to a hypersensitive answer which produces an inflammatory tissue damaging process in the body. In infectious diseases the invading microba can trigger such a hypersensitivity. The immune response kills (or partly kills) the microorganism, but the hypersensitive inflammatory reaction will harm in tissues. The type of this pathological immune reaction provoked by microbas can be either humoral or cellular.
Bacteria or fungi triggered IgE antibodies, triggered by bacteria and fungi, play a role in *anaphylaxia (Type I) reactions.*
Cytotoxic (Type II) reaction can develop e.g. in Trepanosoma infection.
HBV, CMV, Streptococcus pyogenes, Mycobacterium leprae, Treponema pallidum pallidum, Plasmodium malariae, Toxoplasma gondii etc. can provoke an *immune complex mediated (Type III) damage.* The scene of such a reaction is usually the skin or mucosa or the wall of blood vessels in different organs (skin, kidney, lung, connective tissue, joints etc).
Herpes viruses, the Lymphocytic choriomeningitis virus, Mycobacterium tuberculosis and leprae, Candida sp., Trypanosoma cruzi, etc. can provoke a *cellular, (Type IV) lymphocytes mediated immune damage.*

4.2.2.3. Autoimmune Reaction as a Pathological Outcome in Infectious Diseases

Autoimmune diseases do not generally have a simple, single cause. There are two major factors that are involved in causing autoimmune diseases: genetical and environmental. In an autoimmune disease there are multiple genes involved; they collectively increase the vulnerability or susceptibility to become an autoimmune disease. Susceptibility of autoimmunity is associated with the HLA type. Since certain particular combinations of

genes cause susceptibility, only a relatively small number of the population appears to be genetically susceptible to get a given autoimmune disease. Among them only in those can develop an autoimmune disease of infectious etiology, who encounter a certain infectious agent. The activation and the clonal expansion of autoreactive T cells is required for the development of autoimmune diseases. It has long since been considered that also infectious agents can activate autoreactive T cells. It can happen in several ways, e.g. via *intracellular signalling pathways* manipulated by lymphotropic viruses; via *molecular mimicry* (microbial peptides with a sufficient sequence similarity to self-peptides e.g. MHC/ peptide/TCR complexes) or via *microbial superantigens* (activating large numbers of T cells that express particular Vß gene segments, also a subpopulation of these activated cells can be specific for a self-antigen.)

The inflammatory situation that results in a viral or bacterial infection leads to the local activation of antigen-presenting cells and can result in an *enhanced processing and presentation of self-antigens* present on that locus. A strong association of certain microbas with autoimmune diseases is published, e.g. between Klebsiella pneumoniae and Ankylosing Spondylitis; or between Coxsackie virus B and Diabetes Mellitus Type 1, etc.

In the near past a study about Chagas Heart Disease (CHD), caused by the parasite Trypanosoma cruzi was published. With antiparasitic therapy was found a direct link between the parasite level and the presence of autoimmunity. The results of the treatment suggested that the elimination of the parasite may result in the reduction or elimination of autoimmunity even in the chronic phase of infection.

In my work with RFR method I found just the same occurrence. The definite eliminating of the microbas, causing a chronic infection of very long duration and provoking autoimmunity can finish the autoimmune process, while the discontinuity of the infection will lead to the healing of the autoimmune disease.

5. HUMAN PATHOGENIC VIRAL INFECTIONS

All viruses are obligate intracellular parasites which use the functions of the host's cell to multiplicate themselves. A virus attaches itself to a cell, often to a specific type of cell. The virus releases its DNA or RNA inside the attached cell, while its DNA or RNA is taking control over the various biochemical processes of the metabolism of the cell, so that generated proteosynthesis and viral DNA multiplications will eventually proceed.
Some viruses kill the cells they infect inducing cytolysis. Others alter the cell function in such a way that the cell loses control over the normal cell division and becomes malignantly transformed as the result of the following command: divide! given by the virus. Some viruses incorporate a part or all of their genetic information into the DNA of the host cell. Viruses attach themselves only to host cells, which possess specific receptors for them (this phenomenon is called *species specificity*). Most viruses have a preferred host, specifically called host viruses. Other viruses, such as the influenza virus, can infect human beings and a variety of certain animals. The viral infection will take place only in a tissue, the cell of which has specific receptors for the virus in question. This phenomenon is called *organotropism* (e.g. enteroviruses, cold viruses, neurotrop viruses). In some cases only one or a few determined cell types have specific receptors for the given virus (e.g. EBVs can solely attach to the CD21 molecule of B-lymphocytes). In other cases the receptors for the given virus are found on a lot of human cell types. (e.g. CMVs attach to the MHC I molecules).
The immune defence against viruses can be characterized by defence against the virions as well as with the defence against the infected own cells. The first mentioned happens with specific neutralizing antibodies and IFN-alfa citokines, the second one with cytotoxic T-cells, citokines as INF-gamma, TNF, NK-cells etc.
The pathogenicity of the viruses is influenced also by their *survivor strategies*. These mechanisms try to evade the host's immune defence. Some examples of these mechanisms are the following: the changing of the viral antigenic structure (e.g. influenza), the overproduction of solubile antigens (e.g. HbsAg), the incorporation of surface-antigens of infected cells (retrovirus), the intracellular latency (e.g. HSV), the integration into the host DNA (e.g. papova viruses), the invasion of the body with suppressed immune reactive potency (e.g. VZV), the blocking or damaging of the antigen presentation(CMV, adenoviruses), the infection of immune cells (e.g. HIV), etc.
Chemotherapy of viral diseases with antibiotics and antiviral agents does not solve the problem of most of the viral infections.
A *vaccine prophylaxis* with live or inactivated vaccines is available but for a few viral diseases, e.g.: poliomyelitis, measles, smallpox, mumps, rubella, varicella, influenza, rabies, yellow fever, FSME.
The RFR method could be a new solution in healing viral infections, considering the lifecycle of the virus, the eliminating can happen with repeated treatments.

5.1. The Most Frequent Human Viral Diseases Caused by RNA Viruses

5.1.1. Myxoviruses – helical nucleocapsid with envelop

Most of the human viral illnesses are caused by myxoviruses. They have a dominant affinity to mucopeptids on the surface of the host's cells.

5.1.1.1. Myxovirus Influenzae (-)ss RNA

Influenza is an acute respiratory infection caused by Myxovirus influenzae hominis.
Relying upon serological differences it has three types named A-, B- and C- Influenza virus. Influenza tends to spread rapidly in seasonal epidemics. Influenza A virus is the pathogen agent of major epidemics that tend to recur at intervals of 2 to 4 years in the winter months. This influenza virus has some subspecies mutants. Infection with one subtype confers no immunity to infection with an other one. Influenza B usually occurs sporadically or in localized outbreaks. Influenza C is rarely detected from flu-like illnesses because of its mildness and sporadicity first of all among quite young children.
Symptoms of influenza are characterized by a sudden onset of high fever with headache, myalgia, and malaise, non-productive cough, sore throat and running cold. Most infected people recover within one or two weeks requiring no medical treatment. However, in case of very young, or elderly people and those in serious medical conditions, the infection can lead to severe complications. The chief complications of influenza are pneumonia: either primary or secondary bacterial pneumonia superimposed on the lesions produced by the influenza virus. In addition, bacterial infections of the paranasal sinuses as well as the middle ear may occur. If the immune system of the patient is immature or suppressed, influenza can lead to death.
Prevention done by vaccination.
The diagnosis is based on the symptoms and the epidemiological datas.
The treatment depends on the severity of the symptoms, usually symptomatic or focussed on complications.
The RFR method can eliminate the virus. It is recommended in every case of immune deficiency, in case of children with immature immune system, in other complicated cases and for those, who take immunosuppressive drugs.
Frequency values:
The general frequencies of the Influenza A and B viruses are: 309-324, the most frequent ones of them are: **310-313 kHz**
The influenza mutants and subspecies are:
1957 „A" type: 293, 393 kHz
1978 type: 313, 322, 416, 432 kHz
1979 type: 289, 403, 524-534 kHz
1983 type: 310, 373-375, 434-438 kHz
1989 type: 311, 320, 375, 382 kHz
1993 type: 311-314, 398, 435, 490, 534 kHz
1994 type: 352-357, 408 kHz
1997 type: 312, 372, 402, 450, 476 kHz
1998 type: 311-313, 476 kHz
1999 type: 310-313 kHz
2000 type: 310-315, 317, 319, 321 kHz
2001 type: 308-313, 315, 317 kHz
This list is not complete; there are other subspecies of influenza virus that have different wave resonances. **You have to measure!**
Some other influenza A virus frequencies are: 317-318, 328-338, 350, 384, 397, 411, 441, 452, 487-488, 521, 572 kHz
Some other influenza B virus frequencies are: 290, 303, 369, 378, 476, 452-552, 568 kHz
Influenza from V-1 to V-75 grippe type: 322-340, 350-360, 372, 426-427, 440, 456, 471-472, 482-483, 506, 522, 530, 562-566 kHz
Grippe V-75: 316, 350, 522 kHz
Grippe VA-2: 340, 372, 426-428, 471, 488, 506 kHz
Grippe VA-2L: 456 kHz

Grippe VAPCH: 312, 350 kHz
Grippe in general: 305, 350, 441, 448-454, 524, 552 kHz
Grippe 1986: 300, 337, 346, 472, 508, 544 kHz
Influenza virus of swine: 339, 422, 429, 442, 472, 509 kHz
General influenza frequencies: 308-324 kHz
Original avian type influenza virus: 308-324 kHz
Original swine type influenza virus: 318-324, 338-341, 422-429, 442-445, 507-510 kHz
H1N1 (2009) influenza virus: 276-286, 309-311, 560 kHz
H1N1 (2010) influenza new mutans virus: 250-268 kHz
Proposition by the RFR method: The antigen structures and the resonance frequencies of the influenza viruses often change. As the influenza viruses have a fast growing cycle they must be eliminated repeatedly in every six hours. A person with weakened immunity can be reinfected and may require prophylactic treatments every week.

5.1.2. Paramyxoviruses – helical nucleocapsid with envelop

5.1.2.1. Paramyxovirus Influenzae (-)ss RNA

On the basis of antigenic differences, parainfluenza viruses are divided into four types, of which type 4. is divided into two subtypes. These viruses only cause sporadic, local endemic acute respiratory infections mostly among children between six months and three years of age, usually in late autumn, winter and early spring. The viruses have only a few serological variations, so that infection in adults is very rare.
Symptoms often include running cold, brassy cough and a slight fever develop within about 2-6 days after exposure. The virus is usually limited to the upper respiratory tract and pharyngitis, laryngotracheitis and *croup* (laryngotracheobronchitis) can develop in case of younger infants having smaller airways. Some of the most distinctive characteristics of croup is the sudden onset, the pattern of symptoms (it is beginning in the middle of the night with a cough that sounds like the bark of a seal. Another common sign of croup is the inspiratory stridor, which is a loud, high-pitched, harsh noise, that children with croup often have when breathing in, caused by inflammation in the larger airways. The symptoms of croup are due to inflammation, swelling and the developing of mucus in the larynx, windpipe and the bronchial tubes. Infections of the lower respiratory tract (e.g. in case of very young children) lead to more serious symptoms.
In contrast, older children and adults have only cold symptoms when infected by the same virus.
A bacterial coinfection can also be a possible complication.
Treatment: according to the symptoms
RFR method: eliminates the virus. It is adviced in long-continued or severe cases.
Its frequencies are: 338, 368-372, 381-384, 390-397, 410, 534, 564, 568 kHz

5.1.2.2. Respiratory Syncytial Virus (RSV)

RSV infections often occur in epidemics that last from late in the autumn to early spring. RSV is highly contagious and can be spread through droplets containing the virus when a person coughs or sneezes or when a person touches an object contaminated by the virus.
The symptoms in case of adults are *similar to those of influenza*, but cause a more serious respiratory illness of the lungs and the breathing passages (bronchiolitis or pneumonia) in young children. The limitation of epidemic disease to the younger age group is also evidence for unchanging antigenicity of the agent, in contrast to situation with influenza virus, in which antigenic shifts are associated with recurrent epidemics in persons of all

ages. The incubation period of naturally occurring disease in children is about 4 days. The illness usually lasts for about a week, but in some cases it may even last for several weeks. A bacterial coinfection can be a possible complication.
Diagnosis: symptomatically, and antiviral therapy
Differential diagnosis: infection of Rhinovirus, Parainfluenza, Mycoplasma pneumoniae, Chlamydia pneumonia.
Treatment: according to the symptoms
RFR method: detects and may eliminate the virus. It is adviced in long-continued or severe cases.
Its resonant frequencies are: 340-342, 362-365, 378-383, 566-569 kHz

5.1.2.3. Paramyxovirus Parotitidis (Mumps)

Mumps is a contagious human pathogen viral infection causing a painful enlargement of the salivary glands. It is an illness of childhood. The infection usually provides a lifelong immunity. It is contracted by inhaling the virus via droplets of moisture, sneezed or coughed into the air, or by having direct contact with objects contaminated by infected saliva.
Symptoms include chills, fever, headache, poor appetite, a feeling of illness, nausea or vomiting. One or more salivary glands may begin to swell. Pain in the salivary gland is experienced when chewing or swallowing. Especially in case of adults, the infection may affect also other organs causing orchitis and pancreatitis, myocarditis, glomerulonephritis etc. Due to its neurotropism severe complications may occur in the CNS, including meningoencephalitis, myelitis, cerebellar ataxia, Guillain-Barré syndrome, demyelinating processes etc.
Prevention can be done by vaccination (trivalent MMR-vaccina)
Treatment: according to the symptoms
RFR method: usually not needed, it eliminates the virus and is adviced only in severe cases.
Its resonant frequencies are: 299, 308, 318, 324, 328, 336, 344, 348, 372-389, 392, 402, 476-492, 513, 528, 544 kHz
Resonant frequencies of the Mumps vaccines are: 363, 373, 556, 564 kHz

5.1.2.4. Paramyxovirus Morbilli (Measles)

Measles is an acute, highly communicable human illness causing rash due to a virus transmitted by direct contact with infectious droplets or by airborne spread. From the nasopharynx the virus infects the whole body generalizing via viraemia.
Symptoms: The onset of illness is characterized by fever, cough, running cold, conjunctivitis and erythematous maculopapular rash. Koplik (tiny white) spots, an enanthem present on buccal mucous membranes, are pathognomonic for measles. The most frequent complications include diarrhea, middle ear infection and pneumonia. Encephalitis or other severe complications can occur among children younger than 5 and adults older than 20 years of age.
Prevention: measles vaccine is one of the routine immunizations. The vaccine is usually given in combination (MMR):
The frequencies of the vaccines are: 369-373, 382, 390, 436 kHz
Treatment: depends on the symptoms and the coinfections.
RFR method: is not needed since vaccination. Nevertheless detects and eliminates the virus. It is adviced solely in severe cases.
The frequencies of the measles are: 364-373, 381-387, 390, 402-407, 450-456, 478, 492, 522-536, 564 kHz

5.1.3. Rhabdoviruses – helical nucleocapsid with envelop (-)ssRNA

There are >200 Rhabdoviruses known (probably still an under-estimate of the total), which infect human beings. Transmission varies depending on virus/host, but most of them are transmitted by direct contact – e.g. rabies – animal bites or insect vectors. Receptor molecules for Rhabdoviruses are not known, but are believed to be phospholipids.

5.1.3.1. Rabies virus (Lyssa virus)

Lyssa virus is the most important member of the rhabdoviruses. This virus is present in the saliva of infected animals. The general hosts of rabies are foxes, dogs, cats, bats, skunks, raccoons, coyotes, mice, rats, rabbits, deer, squirrels, chipmunks, and others. Its pattern is usually endemic. The entry of Lyssa virus occurs by wounds or abrasions of the skin directly into the bloodstream (animal bite). Symptoms usually begin 30 days after the infection. Primary replication occurs locally in the muscle and the connective tissue (causing no symptoms), but the virus eventually infects peripheral nerves, then travels along the neuronal axons to CNS, where it produces severe and fatal encephalitis. It subsequently travels via nerves to the salivary glands and into the saliva. Virus is a dead-end infection for people.

Symptoms: Rabies starts with depression, restlessness, a sick feeling, fever, and paralysis in the lower legs moving up through the body. Restlessness increases to uncontrollable excitement, the person produces a lot of saliva. Persons with rabies are unable to drink due to hydrophobia.

Prevention: Although in the veterinary medicine rabies vaccines are used as a preventive measure, vaccination of human beings is done mainly after being bitten by a rabid animal. Different kinds of vaccination are recommended, varying in number and method (muscle or skin) of injections. In the case of severe exposure, vaccination is often accompanied by injection of rabies immunoglobulin. Neither the administration of vaccine nor that of rabies immunoglobulin appears to be helpful once the symptoms having developed in a person.

Therapy: There is no effective drug treatment against rabies, but a passive immunization can be of value. This is one of the cases where therapeutic (post-exposure) vaccination is important - the aim is not to prevent infection but to moderate the severity of the disease.

RFR method: can only help until the patient is free of symptoms.

The resonant frequencies of the lyssa virus are: 406, 409, 488, 558 kHz

This list is not complete.

5.1.4. Coronaviruses – helical nucleocapsid with envelop (+)ss RNA

Five different currently known strains of coronaviruses (such as HCoV 229E, HCoV OC43, SARS-CoV, NL63 and HKUl) can infect human beings.

The strains **HCoV 229E** and **HCoV OC43** are generally known as viruses responsible for the *common cold syndrome.* Coronaviruses cause cold syndroms among people primarily in winter and in the early spring. The significance of the coronaviruses as causative agents of a common cold are hard to assess as they are difficult to grow in the laboratory. The best known human coronavirus is the **SARS-CoV**, which cav infect both the upper and the lower respiratory tract and can cause gastroenteritis too. The illness, called *Severe Acute Respiratory Syndrome (SARS)* caused an epidemic outbreak in Asia 2003) with over 8000 infected people and 10% mortality.

The within 2-3 days appearing initial **symptoms** of the SARS infection, are like those of flu (i.e. myalgia, lethargy, gastrointestinal symptoms, cough, sore throat etc.) The sole

symptom common to all of the patients is high fever. About 10-20% of the cases require mechanical ventilation.
Therapy: There is no effective drug treatment against coronaviral infections
RFR method: detects and may eliminate the virus.
The most frequent resonances are: 310-320, 350, 357, 381-387, 389, 395-398, 445, 464-475, 478-481 kHz
The resonant frequencies of certain mutant Coronaviruses are: 458-462 kHz

5.1.5. Picorna viruses – cubical nucleocapsid without envelop (+)ssRNA

Picorna viruses can cause infection both in human beings and animals. Two groups of these viruses, i.e. Rhinoviruses and Enteroviruses include human pathogens. Some Picorna viruses live in the upper respiratory tract – this group is called Rhinoviruses 1-100. Others prefer the gastrointestinal tract – this group of picorna viruses is named Enteroviruses, such as Polioviruses 1, 2, 3, Coxsackie viruses A1-22, A24, B1-B6, ECHO viruses 1-34, Newer Enteroviruses 68-71 -and more. The Hepatovirus, which causes liver inflammation is the Hepatitis A virus, its other name is Enterovirus 72. This *traditional taxonomy* of Enteroviruses was based on the associated disease in humans and animal model systems, sometimes resulting in overlaps between groups and difficulties with classification.
Current taxonomy takes into account molecular and biologic characteristics and divides human enteroviruses into four species i.e. human enterovirus (HEV) A, B, C, and D, but keeps traditional names for individual serotypes. More and more new enteroviruses are identified and described. ECHO viruses 22 and 23 have been reclassified as a new genus i.e. Parechovirus in Picornaviridae and are termed human parechoviruses 1 and 2, respectively. Although they belong to genetically and biologically distinct genera, human parechoviruses and human enteroviruses share many epidemiologic and clinical characteristics.

5.1.5.1. Rhinoviruses

More than 100 types of rhinoviruses have been identified. Rhinovirus infection (generally called common cold) represents fifty percent of the respiratory illnesses in children and adults. The first signs of disease are a scratchy throat, nasal congestion and discharge, malaise, and mild headache. There is usually no fever. Recovery is rapid and complete.
RFR method: Is seldom necessary, but it can detect and eliminate the virus.
Its resonant frequencies are: 296, 318, 340, 367-372, 381-384, 391-402, 409-411, 418, 450-454, 475, 488-504, 508, 511, 544-564, 568 kHz
This list is not yet complete, as there are other rhinovirus groups that have different resonance frequencies.

5.1.5.2. Enteroviruses

5.1.5.2.1. Polioviruses (1-3)

Poliomyelitis is a common acute viral infection which, naturally only occurs in human beings producing wide variety of clinical symptoms. In its most severe form it attacks parts of the central nervous system. The poliovirus, as an enterovirus, is spread through ingestion, such as water contaminated by infected feces. The virus is recoverable from pharyngeal secretions for only a few days but is demonstrable in the feces for several weeks. The infection spreads from the intestine throughout the body, but the brain and the spinal cord are the most severely affected. Strains of the poliomyelitis virus vary greatly in their ability to invade the nervous tissue and to destroy neurons.

Symptoms: Nonparalitic poliomyelitis is characterized by prodromal manifestations, signs of meningeal irritation, and abnormalities of the spinal fluid. The syndrome of paralytic poliomyelitis consists of prodromal manifestations, signs of meningeal irritation, abnormal spinal fluid, and the involvement of motor nerve cells in the spinal cord, brain, or cranial nerve nuclei, resulting in paresis or paralysis of various muscles. Before vaccines became available, outbreaks occurred during the summer and autumn months in temperate climates. Symptoms begin three to five days after infection, and include an overall feeling of illness, a slight fever, headache, a sore throat, and vomiting.
Diagnosis is confirmed by identifying the poliovirus in the stool or pharyngeal secretions, and by detecting high levels of neutralizing antibodies against the virus in the blood.
Treatment: Poliomyelitis cannot be cured, and antiviral drugs do not affect the course of the disease.
Prevention: polio vaccine is included among the routine childhood immunizations.
RFR method: detects and eliminates the virus.
Its resonant frequencies are: 289, 336-338, 372-379, 382-385, 397, 403, 419, 438, 450, 473, 488, 493, 552, 576-579 kHz
This list is not yet complete, because there are other subspecies with different frequencies.

5.1.5.2.2. Coxsackie Viruses

5.1.5.2.2.1. Group A Coxsackie Viral Infections

Symptoms: Group A Coxsackie viruses can cause a lot of symptoms and diseases as herpangina, lymphonodular pharyngitis, upper and lower respiratory disease, cutaneous eruptions, hepatitis, aseptic meningitis, paralytic disease, myopericarditis, and sudden unexpected deaths in infancy. Also diarrhea can result from group A Coxsackie viral infections.
The most characteristic disease caused by some types of Coxsackie viruses group A is **herpangina**, a common febrile illness characterized by small, papular, vesicular, or ulcerative lesions on the anterior pillars, soft palate, tonsils, pharyngeal mucous membrane, and the posterior part of the buccal mucosa. Herpangina has been diagnosed by types 1 to 10, 16, and 22 of the group A Coxsackie viruses.
The Coxsackie virus A10 may induce **acute lymphonodular pharyngitis**. Lesions here are raised, discrete, white to yellow 3-6-mm. papules surrounded by a zone of erythema. All of the papules appear at the same time and they do not ulcerate.
The Coxsackie virus A21(Coe virus) and A24 are predominantly an **upper respiratory tract** pathogens. The first is regularly isolated from the throat (and only occasionally from feces). It produces ilnesses that resemble the common cold, except for a higher incidence of fever.
Illnesses of the **lower repiratory tract** such as tracheitis, bronchitis, croup, bronchiolitis and pneumonia can affect infants and children, (rarely adults) by serotypes Coxsackie viruses A7, A9 and A16.
The first enterovirus etiologically implicated in pneumonia was the Coxsackie virus A9. Interstitial diffuse **polylobed bronchopneumonia** with alternate areas of atelectasis and emphysema were found.
A mucocutan syndrome, i.e. the **Hand-Foot-and-Mouth Disease** is usually caused by Coxsackie virus A16. The illness usually occurs in summer and in the early autumn.
A variant of Coxsackie virus A24 was responsible for epidemic cases of **acute hemorrhagic conjunctivitis**, which is highly contagious, characterized by a sudden onset of pain, photophobia, conjunctivitis, swelling of the eyelids, and subconjunctival hemorrhages.

Instances of respiratory or **gastrointestinal symptoms**, *acute renal disease*, thrombocytopenia, and hemolytic anemia were reported to have affected infants and children with Coxsackie virus A4.
Aseptic viral meningitis occasionally with some paralytic disease occur, mostly in developing countries (caused by one of the concerning Coxsackie viruses A2, A4, A5, A7, A9, A10, A16). Attack rates are generally highest in children. Its first symptoms are typical of an undifferentiated febrile illness.
Acute myo- and pericarditis was associated with infections by Coxsackie viruses A (types 1, 2, 5, 8, and 9); more substantial etiologic data exist for types 4 and 16. An estimate suggests that min. 23 percent of acute viral cardiomyopathies and a lot of cases with chronic cardiac muscle hypertrophy may be caused by Coxsackie viruses A (many of these diseases caused by other viruses hadbeen controlled with vaccines). A significantly greater incidence of infection with the Coxsackie virus A9 was reported conserning mothers of infants with connatal heart disease. Neonatal infections frequently result in severe myocarditis with high mortality, whereas conserning older children and adults, pericarditis often predominates and the disease is generally benign and abates by itself.
Its frequencies are: 287-290, 292-304, 346, 388, 393, 407-408, 432-434, 444, 471-472, 552 kHz

5.1.5.2.2.2. Group B Coxsackie Viral Infections
Symptoms: Infections with Coxsackie viruses group B can cause pleurodynia, cardiac diseases, aseptic viral meningitis, gastroenteritis, *chronic myalgias*, upper respiratory syndromes, pneumonia, a hemolytic uremic syndrome and exanthems. Coxsackie virus B4 may via pancreatitis cause diabetes mellitus. Vesicular lesions of herpangina are less commonly caused by Coxsackie virus B1-5.
Epidemic pleurodynia (synonims include epidemic myalgia, Bornholm disease, devil's grip etc.) is an acute febrile illness, the principal causes of which are especially Coxsackie viruses B3 and B5. Its prodromal symptoms are malaise, sore throat accompanied by sudden onset of lower thoracic, intercostal or abdominal pain. The pain is accentuated by moving, breathing, coughing, sneezing, and hiccupping, and may spread to the shoulders, neck, or scapulae. Pain and spasms of the anterior abdominal muscles occur in about half of the cases, often in combination with chest pain. Muscle tenderness is usually not prominent, but some patients do complain of intense cutaneous hyperesthesia and paresthesia over the affected area. Meningitis, myocarditis, or hepatitis may ensue early in the course of theillness. Orchitis can be a late complication of the infection.
Cases of **acute myocarditis** of infective etiology were most frequently caused by strains of Coxsackie viruses group B. If connatal or neonatal infections occur, the course of the illness is often rapidly fatal, with concomittant myocarditis, encephalitis, hepatitis, and sometimes adrenal necrosis. Cardiac inflammation varies in intensity and in the degree of muscle necrosis. Pericarditis may dominate the clinical presentation, with myalgia, fever, precordial pain, friction rub, and even cardiac tamponade. During infancy, the mortality rate of patients with acute infectious myocarditis is about 50 percent.
Infections caused by Coxsackie viruses B often lead to a **chronic heart disease**. Similarly, there is an epidemiological evidence that associates Coxsackie viruses B3 and B4 with **connatal heart disease**. Also Coxsackie viruses group B can cause **aseptic meningitis**, usually beginning with fever, headache and a stiff neck. Localized sensory or motor deficiencies are unusual.
Coxsackie virus B3 and B4 can be an etiological cause of **gastroenteritis**, occuring in summer or winter in sporadic endemic cases. Typically, repeated vomiting, retching, and chills ensue. Abdominal cramps and myalgia may occur too. The disease is highly contagious, multiple cases occurring simultaneously within a family or following closely one after the other.

Acute and chronic pancreatitis manifested itself in the pancreatic Langerhans' island conserning the infection of Coxsackie virus B4. Pancreatitis is characterized by constant pain in the abdominal area and the back. This infection could initiate **diabetes type I** directly by infecting and destroying pancreatic beta cells. Several serological studies gave evidence of a higher frequency of Coxsackie viral infection group B effecting children suffering a fresh onset of diabetes type I (IDDM), moreover, maternal enteroviral infections during pregnancy became associated with the subsequent development of diabetes type I in their offsprings's early childhood.
The **Summer Grippe** is a febrile respiratory illness also can be caused by Coxsackie viruses B2, B3 and B5. It occurs in summer or early in the autumn and is marked by headache, sore throat and anorexia.
Diagnosis: by isolation of the virus, or serological diagnosis.
Treatment: symptomatically
RFR method: detects and eliminates the virus.
Frequencies:
Coxsackie viruses B1: 287-290, 300, 360-370, 392, 426 kHz
Coxsackie viruses B2: 287-293, 297-301, 360-362, 443, 546 kHz
Coxsackie viruses B3: 287-293, 297-301, 333-335, 444, 498 kHz
Coxsackie viruses B4: 307-308, 360-366, 419-426, 430, 534-544, 552-554 kHz
Coxsackie viruses B5: 287-291, 331, 360-362, 396, 472, 533, 553-555 kHz
Coxsackie viruses B6: 336, 340-343, 350, 366-376, 407-416, 498, 564 kHz
Other Coxsackie viruses: 294-295, 313, 345, 389, 445, 475, 557 kHz

5.1.5.2.3. Enteric Cytopathic Human Orphan (ECHO) Viruses

In cases of infections caused by ECHO viruses.
Symptoms are as follows: ECHO viruses cause a wide variety of conditions.
ECHO viruses types 1, 3, 6-9, 11-14, 18, 19, and 22-24 were proved to be etiologically implicated as a cause of gastroenteritis occuring in summer or winter. ECHO virus infections are common, taking usually the form of *gastrointestinal infection* and *skin rashes*. This virus can cause *endometriosis*.
ECHO viruses (types 1-4, 6, 9, 11, 19-20 and 25) too can cause a febrile illness called *summer grippe*, with inflammation in the upper respiratory tract. Seldom also *Herpangina* can be caused by ECHO viruses, types 9 and 17.
ECHO viruses 4, 8, 9, 11, 12, 14, 19 20, 21, 25, and 30 were also associated with *lower respiratory tract illnesses* of infants and children, although rarely of adults. More serious infections occur less frequently but are nethertheless of significant importance.
One out of five cases of *aseptic meningitis* is thought to be caused by an ECHO virus. These infections are sometimes causing epidemics. Viral brain infections can produce three different kinds of symptoms. Some infections are mild, causing fever and a general feeling of illness, often without specific symptoms. The viral meningitis usually produces fever, headache, vomiting, weakness, and a stiff neck. Encephalitis disrupts the normal brain functions, causing changes in the personality, seizures, weakness of one or more parts of the body, confusion, sleepiness that can lead to coma, and to the symptoms of meningitis.
Diagnosis: e.g. CT, MRI, and immunological tests are used to measure antiviral antibodies.
Treatment: symptomatically
RFR method: detects and eliminates the virus.
Its resonant frequencies are: 308-321, 369, 379, 391, 395-405, 470-476, 526 kHz

5.1.5.2.4. Enteroviruses of Newer Serotypes and Human Hepatitis A Virus (serotype 72)

Quite recently identified enteroviruses are not included in the original classification; such as serotype 68 which was associated with illnesses *of the lower respiratory tract* in regard to infants and children. The Enterovirus serotype 70 was held responsible for a lot of cases of *Acute hemorrhagic conjunctivitis* (AHC). *Hand-foot-and-mouth disease* is usually caused by Coxsackie virus A16, though it can be caused by Enterovirus serotype 71 too.
The **Human Hepatitis A virus** (HAV) is identical with Enterovirus serotype 72, causing the infection called Hepatitis A. Similar to other enteroviruses, its transmission happens fecal-oral way. Due to the way it is spread, the Hepatitis A virus tends to occur in water-borne and food-borne epidemics and outbreaks. Isolated cases, usually arising from person-to-person contact, are also common. The illness is characterised by liver inflammation. Once a person has had Hepatitis A, a lifelong immunity develops, one cannot get the disease again. People who do not have symptoms can none the less spread the virus. Infection with HAV is known to occur throughout the world. The risk of infection is greatest in developing countries or places with poor sanitation or poor personal hygienic standards.
Symptoms: Infections with the Hepatitis A virus seldom cause symptoms, remaining thus unrecognized. The prodromal symptoms, usually developing between 2 to 6 weeks after infection are variable and systemic. Constitutional symptoms of nausea, vomiting, diarrhea (especially as regards children), loss of appetite, rash, low grade fever, tiredness, malaise, headache, mild weight loss (photophobia, arthralgias, myalgias, pharyngitis, cough, coryza, alteration in olfaction and taste) may precede the onset of jaundice by one to two weeks. With the onset of clinical jaundice the constitutional prodromal symptoms usually diminish. Hepatitis A does not cause chronic (long-term) diseases..Although the liver does become inflamed and swollen, it mostly heals completely without causing any damage in a few months.
Prevention: strict personal hygiene and hand washing help to prevent transmission of HAV to others. Contaminated surfaces should be cleaned, water and food should be heated to 85 C. There are vaccines that help to prevent infection with HAV and also the passive immunization with immune globulin.
Treatment: There are no specific medicines to cure the infection. Symptomatically.
RFR method: Detects and eliminates the virus. Be careful! The liver is very sensitive.
Its resonant frequencies are: 285-295, 320-330, 340-356, 361, 366, 403, 420-436, 449, 487-488, 498, 570-590 kHz

5.1.6. Reoviruses (Respiratory Enteric Orphan) – cubical nucleocapsid without envelop dsRNA

The Reoviridae family has been divided into 9 genera, 4 of which – Orthoreovirus, Coltivirus, Rotavirus, and Orbiviruses – can infect people as well as animals. Reovirus infections usually have a benign course.

5.1.6.1. Orthoreoviruses

Viruses of this group are isolated from the *respiratory and enteric tracts*, which are assumed to represent the natural portals of entry for orthoreoviruses into the host.
Symptoms: these viruses are associated with upper respiratory infections, enteritis, fever, and febrile exanthema in childhood. Orthoreoviruses usually cause mild physical illnesses. In rare cases complications (e.g. pneumonia, encephalitis, meningitis may occur.

5.1.6.2. Coltivirus (Colorado Tick fever Virus)

Colorado Tick Fever (CTF) is an infection caused by Coltivirus and transmitted to human beings by tick vector; the symptoms are mild, there is no rash, fever is not excessive, and the disease is rarely fatal. Its synonima is Tick fever 5. The virus is transmitted to

mammals, including human beings, principally by the adult Rocky Mountain wood tick (i.e. Dermacentor andersoni.) Clinical cases reach their peak between May and July. Human erythrocytes are known to carry the virus, CTF has a benign course, and an excellent prognosis.

Symptoms: The triad of high fever, severe myalgia, and headache is typical but not specific. In some cases, involvement of the central nervous system, clouding of the sensorium, neck stiffness and vomiting may be present. Rarely, also encephalitis, aseptic meningitis and hemorrhage were reported too.

Its resonant frequencies are: 295-296, 311-323, 354, 381, 384, 388, 403, 407-408, 427, 432-433, 441, 452, 465, 479, 482, 489, 511, 524 kHz

5.1.6.3. Rotavirus

Infection with human rotavirus appears to cause a great number of cases of gastroenteritis affecting children aged from 6 months to 2 years. The disease was found on every continent and among all races, though its worldwide prevalence is not known. Six serological groups are identified, three of which (groups A, B, and C) infect human beings. This virus is known to be an important cause of diarrheal diseases of infants and young children. The presence of rotavirus in the feces is not always associated with a symptomatic disease. Morbidity is highest among babies aged 6-11 months, while the mortality is highest concerning infants and children aged 1 year. The transmission of rotavirus infections is believed to be fecal-oral, with little evidence of airborne transmission. Rotavirus Group B, also called adult diarrhea rotavirus that is ADRV, has caused major epidemics of severe diarrhea in China.

Symptoms: Infants and young children most commonly have fever, vomiting, diarrhea, and (occasionally) dehydration. The disease is characterized by *vomiting and watery diarrhea* lasting for 3-8 days, frequently with *fever and abdominal pain.* Symptoms of an apparent *upper respiratory tract infection* may be present. The patient's stools can be watery, rarely contain mucus and number as many as 10 per day. In most cases, diarrhea is generally persisting no longer than 3-4 days. Temporary lactose intolerance may occur. Because the virus remains alive, transmission can happen through ingestion of contaminated water or food or due to contact with contaminated surfaces. Association with other enteric pathogens may play a role in the severity of the disease. In developing countries, rotavirus accounts for 10-20% of gastroenteritis-associated deaths (i.e. 5-10 million deaths every year).

Treatment: in case of persons with a healthy immune system, rotavirus gastroenteritis is a self-limited illness, lasting but for a few days. Treatment is nonspecific and consists of oral rehydration therapy to prevent dehydration, or infusion. About one among forty children with rotavirus gastroenteritis will require hospitalization needing intravenous fluids.

Prevention: a live virus vaccine (Rotashield) in case of children's use.

RFR method: detects and eliminates the virus!

The resonance frequencies in regards to all groups of pathogen rotaviruses are unknown. Should only be used in cases of serious illness.

Its resonant frequencies are: 311-320, 334, 346, 369, 373-375, 392, 397, 403, 420, 432, 444, 472 kHz

5.1.6.4. Orbiviruses

Symptoms: Orbiviruses usually cause *illnesses with neurological symptoms*. Orungo virus, isolated in Africa, was implicated in an acute illness with myalgias and headache. Lebombo virus is another orbivirus isolated from people in Africa. Kemerovo virus was implicated in neurologic infections in central Europe and Russia. Serologic evidence of infection with Lipovnik or Tribec virus was demonstrated in patients with polyradiculitis in former Czechoslovakia. Changuinola virus was isolated from people in Panama.

Treatment of reoviruses can solely be symptomatic.
RFR method: not known.

5.1.7. Arboviruses (Arthropod-borne viruses) – cubical nucleocapsid with envelop

There exists a large group (numbering more than 400) of enveloped RNA viruses, transmitted primarily (but not exclusively) by Arthropod vectors (mosquitoes, sand-flies, fleas, ticks, lice, etc.). This disordered assemblage was filed into 4 virus families: Togaviridae, Flaviviridae, Bunyaviridae and Arenaviridae. The term 'arbovirus' has no taxonomic significance.
Its resonant frequencies are: 428-435 kHz

5.1.7.1. Encephalitides Transmitted by Arthropod Vectors

The Arboviruses that cause **human encephalitis** are members of three virus families: such as the Togaviridae (genus Alphavirus), Flaviviridae and Bunyaviridae. Arboviral encephalitides have a global distribution. Certain illnesses occurring in the United States are the following: The *Eastern Equine Encephalitis*, caused by a serotype of Alphavirus, the *Western Equine Encephalitis,* caused by a serotype of Alphavirus, the *La Crosse Encephalitis,* caused by a serotype of Bunyavirus and the *St. Louis Encephalitis,* caused by a serotype of Flavivirus, all of which are **transmitted by mosquitoes**. The last mentioned virus is the most common mosquito-transmitted human pathogen in the U.S. Most cases of arboviral encephalitis occur in summer (from June to September), when the arthropods are active. The *West Nile Encephalitis* is caused by a flavivirus, its vectors are also mosquitos. Infections in human beings are incidental and do not facilitate the spread of the virus. The virus passes from the salivary glands of the mosquito to the bloodstream of the vertebrate host. The virus reaches the skin and the reticuloendothelial system (i.e. the spleen and the lymph nodes), where the primary infection occurs, followed by viraemia causing a systemic infection, which can involve the CNS (encephalitis), skin/bone marrow/blood vessels (haemorrhagic fevers).
Symptoms: The majority of human infections are asymptomatic or may result in a nonspecific flu-like syndrome. The onset may be insidious or sudden, causing fever, headache, myalgias and malaise. Muscle trembling, mental confusion, convulsions, and coma may rapidly develop. The infection may lead to encephalitis, with a fatal outcome or permanent neurologic sequelae.
Prevention: vaccination is partly available. Personal protective measures and public health measures are taken to reduce the number of infected mosquitoes.
Treatment: can solely be symptomatic
RFR method: detects and eliminates the virus.
Its resonant frequencies are: 295-300, 302-310, 317-320, 339, 354-356, 373, 420-423, 430, 444, 495, 570 kHz
Their number is not yet ascertained, since there are still other subspecieses with different resonances.
Powassan virus is a flavivirus which is a **tick-borne** arbovirus, occurring in the United States and Canada. The **Venezuelan Equine Encephalitis** virus is an alphavirus, causing encephalitis in horses and people in Central and South America. The **Japanese Encephalitis** Virus is a flavivirus, widespread throughout Asia. The **Tick-borne Encephalitis (TBE)** is caused by two closely related flaviviruses. The eastern subtype causes the Russian Spring-Summer encephalitis, transmitted by tick Ixodes persulcatus, whereas the western subtype is transmitted by tick Ixodes ricinus, causing the Central European Encephalitis. Though the great majority of TBE infections happen by an exposure to ticks, or else through the ingestion of milk of infected cows or goats.

Symptoms: The incubation period is 7 to 14 days. The infection usually presents itself as a mild, influenza-type illness or as a benign aseptic viral meningitis, but may nevertheless result in a fatal meningoencephalitis. Fever is often biphasic, severe headache, neck rigidity, transient or residual paralysis of the limbs and shoulders or, less commonly, the respiratory musculature might also occur.
Prevention: can partly (f.i. in Europe and Russia) happen with vaccination
Treatment: solely symptomatically
RFR method: hypothetically detects and eliminates the virus.
Its resonant frequencies are not known yet.

5.1.7.1.1. West Nile Fever

The West Nile virus is a group B arbovirus. The transmission cycle is believed to be bird-to-mosquito-to-bird or -to-human, with a Culex type insect as principal vector. Although human beings and a variety of other vertebrates, often horses, are infected by the virus, their involvement is believed to be tangential.
Symptoms: In one-third of patients the temperature quickly rises to 38-40 °C degrees with chills. Symptoms include drowsiness, severe frontal headache, ocular pain and pain in the abdomen and of the back. A small number of patients have anorexia, nausea and dryness of the throat. The signs observed include flushing of the face, conjunctival infection and coating of the tongue. Prominent finding is the general enlargement of the lymph nodes, which are of moderate size, not hard and only slightly tender. The occipital, the axillary, and the inguinal nodes are usually involved. The spleen and the liver are slightly enlarged in a small proportion of patients. The rash occurs predominantly over the trunk and consists of pale roseolar macropapular lesions. The illness is self limited and lasts 3 to 5 days. In a few patients, transitory meningeal involvement may be encountered. The spinal fluid examinations may reveal pleocytosis and increase in protein concentration. The patients have leukopenia and a moderate lymphocytosis. Complications very rarely occur, by elderly or very young patients meningoencephalitis may develop and foul fatalities ensued.
Diagnosis: rests on virus isolation, which can be accomplished, because the viremia persists for 8 days, or on the demonstration of a rising specific antibody titer.
Treatment: symptomatic.
RFR method: detects and eliminates the virus!
Its resonant frequencies are: 295-300, 302-310, 317-320, 339, 354-356, 373, 409, 420-423, 430, 444, 495, 570 kHz

5.1.7.2. Rubella Virus (+)ssRNA

Rubella virus has certain properties which are unique as regards the Togaviruses. It has a special own genus: the Rubiviridae. It is the only Togavirus known to be transmitted via the *respiratory route*. Rubella or „German Measles" is an ubiquitous illness of the human population. As human beings are the only reservoir, WHO targeted to eradicate the virus. The Rubella virus can act as a teratogen. German measles spreads mainly through inhaling droplets of moisture containing the virus, coughed into the air by an infected person. It is less contagious than measles, many children never become infected.
Symptoms: In case of children, the illness begins with a 1-5 days period of feeling mildly ill, with enlarged suboccipital and retroauricular lymph nodes. One can see a mild reddening of the skin, fine pink maculae appearing on the face and rose-coloured spots on the roof of the mouth, later on merging with each other into a red blush. The rush typically spreads over the trunk and the limbs within 2 days, though in some cases there is no rush apparent. Mild arthropathy may also be present. Rubella panencephalitis is a very rare progressive brain disorder affecting children. As concerns pregnant women the virus crosses the placenta and multiplies itself in the fetus. Up to 85% of infants, infected in the

first trimester of pregnancy, get connatal rubella syndromes such as low birth weight, deafness, CNS involvement, abortion. The earlier in pregnancy the infection occurs, the worse it will be.
Prevention: may happen with MMR vaccination
Treatment: symptomatically
RFR method: detects and may eliminate the virus.
Its resonant frequencies are: 372, 402, 440, 450-451, 468, 520-530 kHz

5.1.7.3. Flaviviruses Not Yet Mentioned (+)ssRNA

5.1.7.3.1. Yellow Fever

Yellow fever, a viral disease **transmitted by mosquitoes,** results in fever, bleeding, and jaundice. It can be fatal. Yellow fever is an acute infection of short duration and extremely variable severity. Recovery from infection is followed by lifelong immunity. **The classic triad of symptoms** – *jaundice, hemorrhages, and intense albuminuria* – is present only in case of severe infections, which now comprises only a small percentage of the total number of cases. Yellow fever is nowadays the most dramatically serious arboviral disease of the tropics. Infections result from two basically different cycles of viral transmission: urbanic and sylvatic. The lesions are predominantly visceral.
The attacks of Yellow fever greatly vary in severity. In case of mild yellow fever the only symptoms may be the abrupt onset of fever and headache. Additional symptoms may include nausea, epistaxis, relative bradycardia and a slight albuminuria. Headache is soon followed by pain in the neck, back, legs then nausea with vomiting and retching.
Moderately severe and malignant attacks of yellow fever are characterized by three distinct clinical periods: i.e. infection, remission and intoxication. On the third day of illness, the fever may drop suddenly and the patient is given remission; elseways, in the malignant form, copious hemorrhaging, anuria, or delirium may occur. Jaundice becomes detectable approximately on the third day; moreover, the disease often remains difficult to detect, even in potentially fatal cases. Increased epistaxis, melena, and uterine hemorrhages are common. Among the classic signs, black vomit is more characteristic than jaundice. In case of malignant infections, coma frequently emerges two to five days before death.
Treatment: symptomatically and by given electrolytes.
Prevention: by immunization
RFR method: detects and eliminates the flavivirus
Its resonant frequencies are: 303, 374-379, 398-400, 420-422, 471-473, 510-516 kHz

5.1.7.3.2. Dengue Fever

This illness is worldwide a major health problem (Asia, Africa and America). Caused by one of four closely related virus serotypes of the genus Flavivirus, and **transmitted by mosquitoes,** which feed in the day-time Primary infection produces a (relatively) mild, self-limited, febrile illness. Re-infection with a different antigenic type of the virus may result in dengue haemorrhagic fever: *high fever, haemorrhagic shock, myocarditis, encephalitis.* Its mortality is 15% the symptoms are probably by an autoimmune way mediated.
Treatment: symptomatically
Prevention: There is no vaccine up to now.
RFR method: detects and eliminates the flavivirus.
Its resonant frequencies are: 315, 320, 327, 336-337, 339, 372, 376, 378, 396, 402, 409, 422, 450, 512, 564 kHz

5.1.7.3.3. Hepatitis C Virus

The HCV is a member of the Flaviviridae family, it mainly multiplicates within hepatocytes in the liver by the mechanism of host trophism. The Hepatitis C virus causes the highest percent of hepatitis cases caused by blood transfusion, many other scattered cases of acute hepatitis.
Symptoms: 80% of the infected people have no symptoms. Others feel tired, have jaundice with dark urine, abdominal pain, nausea. Some again will have a clinically insignificant or minimal liver disease without any complications. Other again will suffer a clinically apparent, chronic hepatitis. Among these, some will develop *cirrhosis* as well as the end stage liver disease. Patients suffering cirrhosis are also in danger of developing a hepatocellular carcinoma.
Treatment: ribavirin, interferon, liver transplantation
RFR method: detects and may eliminate the virus.
The most frequent resonances of the Hepatitis C virus are: 324-339, 350-352, 370-374, 396, 400-402, 450-456, 475-482, 540-541, 559-563 kHz

5.1.7.4. Bunyaviruses (-)ssRNA

The Bunyavirus family has more than 200 species, it is the largest family of viruses most of them being important emergent viruses.
Its pathogenesis is generally as follows: The bite of an insect results in transient viraemia; after which a viral replication will occur in the target organs. The severity of illnesses varies from mild to severe just like one virus varies from another one.
Rift Valley Fever: In people, it produces an acute, 'flu-like illness. Transmitted by mosquitoes from animal reservoirs (e.g. sheep) to man it leads to epizootics. In case of the massive outbreak in Sub-Saharan Africa millions of people were infected.
Sand Fly Fever: Transmitted by sand-flies. This acute, febrile illness is common in the Mediterranean.
The Hantavirus pulmonary syndrome is a fatal illness, caused by a genus of Bunyaviruses. Hantavirus spreads from rodents (reservoir) to man by aerosolized faeces, saliva and urine (it has no insect vector). People become infected by breathing in these infectious aerosols. Early symptoms include fatigue, fever, nausea, abdominal pain, later coughing and shortness of breath follow. It causes a self-limiting infection with no viral persistence, manifesting itself as hemorrhagic fever causing renal syndrome.

5.1.7.5. Arenaviruses (-)ssRNA

This virus family is relatively a new one, having 17 types. These viruses do not require arthropoda to spread, they are called rodent viruses. In case of the illness, called
Lassa fever, the natural (i.e. rodent) host transfers the infection to man via droppings. Human infections though rare, but highly infectious, producing **severe, systemic febrile diseases with high mortality.** (This is in contrast with the rodent infection, where there is no illness.
Lymphocytic choriomeningitis is an illness caused by an other type of species called lymphocytic choriomeningitis virus, which is also a zoonotic pathogen.
The symptoms of this rodent-transmitted, viral infected, human disease are aseptic meningitis, encephalitis or meningoencephalitis.
Other illnesses of the arena virus family are e.g. the *Argentine hemorrhagic fever* (Junin virus) the *Bolivian hemorrhagic fever* (Machupo virus) the *Venezuelan hemorrhagic fever* (Guanarito virus) the *Brazilian hemorrhagic fever* etc.
Treatment: symptomatically.
Prevention: there is no vaccine available yet
RFR method: the frequency resonance valueses are not public yet.

5.1.8. Deltavirus (Hepatitis D Virus) circular(-)ssRNA

The Deltavirus (HDV) can cause an infection only in cases, if simultanously a Hepatitis B infection is present. HDV can be acquired either as a co-infection with HBV, or as a superinfection in persons with existing chronic HBV infection. This HBV/HDV coinfection may cause a more severe acute disease and a higher risk of developing acute liver failure compared with those cases infected with HBV alone. A progression to cirrhosis is believed to be more common with HBV/HDV chronic infections.

Treatment: by treating HBV infection

RFR method: detects and may eliminate the virus.

Its resonant frequencies are: 348, 375, 386, 410, 432, 450, 468, 471, 490, 532, 535-548, 550-563, 580 kHz

5.1.9. Hepevirus (Hepatitis E Virus) nonenveloped (+)ssRNA

Hepatitis E virus causes a liver infection via the faecal-oral route. Hepatitis E is a waterborne disease, and contaminated water or food supplies have been implicated in major outbreaks. Consumption of faecally contaminated drinking water has given rise to epidemics, and the ingestion of raw or uncooked shellfish has been the source of sporadic cases in endemic areas. There is a possibility of zoonotic spread of the virus, since, pigs, cows, sheep, goats and rodents are susceptible to infection. The risk factors for HEV infection are related poor sanitation in large areas of the world, and HEV shedding in faeces. In general, Hepatitis E is a self-limiting viral infection followed by recovery. Prolonged viraemia or faecal shedding are unusual and chronic infection does not occur. Fulminate hepatitis can occur more frequently in pregnancy and regularly induces a mortality rate of 20% among pregnant women in the 3rd trimester.

Treatment: symptomatically

RFR method: in severe cases needed. Detects and eliminates the virus.

Its resonant frequencies are: 348, 375, 386, 410, 432, 450, 468, 471, 490, 532, 535-548, 550-563, 580 kHz

5.1.10. Retroviruses ssRNA-RT

Retroviruses rely on the enzyme reverse transcriptase to perform the reverse transcription of its genome from RNA into DNA, which can then be integrated into the host's genome with an integrase enzyme. The virus then replicates as part of the cell's DNA. The family Retroviridae comprises a variety of RNA viruses, such as endogenous retroviruses, leukemia viruses, and HIV-1, the replicative strategy of which includes as essential steps reverse transcription of the virion RNA into linear double-stranded DNA and the subsequent integration of this DNA into the genome of the cell.

Retroviruses cause asymptomatic chronic viral infection in human beings and induce a variety of diseases including: *tumours, wasting and auto-immune diseases, immunodeficiency syndromes and aplastic and haemolytic anaemias* (see also Chapter 5.1.10.1.1.).

5.1.10.1. Human T-cell Lymphotropic Viruses (HTLV)

HTLV is a retrovirus having up to now 6 species, that usually establishes an asymptomatic, but chronic infection. All HTLVs infect CD4 bearing T cells. Polymerase chain reaction (PCR) analysis has been shown to be highly sensitive and accurate for HTLV detection. Serological criteria can be used to identify infected individuals. PCR analysis is useful for the detection of primary infection prior to seroconversion, for distinguishing between types and for determining viral tissue distribution using appropriate specimen types. A number

of seropositive individuals are detected by chance, e.g. during examination prior to the donation of blood.

5.1.10.1.1. Human T-cell Lymphotropic Virus-1 (HTLV-1)

The transmission of HTLV-1 is believed to occur from mother to child via breastfeeding; by sexual contact; and through exposure to contaminated blood, either through blood transfusion or sharing of contaminated needles. Seroprevalence is high in South West Japan, the Caribbean and parts of West Africa. In high incidence areas up to 10% of adults may be infected. The seroprevalence increases with age and family clustering of infection is common. Among a small percentage of patients infected with HTLV, the infection can progress into an **Adult T-cell leukemia/lymphoma (ATL)**. The tumours are only produced after a prolonged latent period. The virus does not contain an oncogene and it is believed that the malignant change is the result of interruption and disregulation of host DNA, by viral genome insertion. Amoung less than 1% of HTLV 1 infected individuals develop this malignancy.

Symptoms: are caused by aggressive tumorous process of CD4 T cells, which infiltrates the skin and brain If the course of the ATL is acute and aggressive (cc 50-75%), it is characterized by lymphadenopathy, both in the periphery and within body cavities. Further frequent features are hepatosplenomegaly, hypercalcemia, and lytic bone lesions. The cutaneous lesions are classified as indolent, nodular, indurated, and, occasionally, diffuse with features of exfoliation and erythroderma. Death is associated with pulmonary complications, opportunistic infections, and sepsis. In a few cases involvement of the cerebral meninges, muscle weakness, disturbed behavior, and/or headache are present. The protein content in the cerebrospinal fluid may still appear normal, while containing ATL cells. The lymphomatous type occurs in approximately 15% of the symptomatic patients. It is diagnosed on the basis of absence of blood and bone marrow involvement, by definition it is HTLV-1 positive, and provirus can be detected in the malignant cells on biopsy. These patients have large; firm peripheral lymphadenomegaly, and cutaneous findings might be present. The average survival time for patients suffering from this affliction is cc. 10 months.

In the chronic form the disease lingers for two years on the average, without bone lesions, hypercalcaemia, or neurological involvement. There may be hepatosplenomegaly, lymphadenopathy, or skin and lung alterations. This form later develops into acute ATL

The other disease caused by HTLV-1 is the **Tropical spastic paraparesis** in which an aggressive non-demyelinating spastic paraparesis. and myelopathy (HAM/TSP) develop. This illness is a slowly progressive degenerative disease that primarily affects the corticospinal tracts of the thoracic spinal cord, resulting in weakness and spasticity, predominantly in the lower limbs, along with sphincter and sensory dysfunction The disorder is somewhat more common in women than in men. The physiopathology of this condition may consist of the autoimmune destruction of nerve cells.

In cases of **HTLV-associated uveitis** HTLV specific viral sequences and infected lymphocytes in the vitreous fluid are present.

HTLV-associated infectious dermatitis is a severe and chronic form dermatitis described among children in Jamaica. It is commonly exsudative and spreads throughout the face, neck and scalp. Crusting and a diffuse papular rash is also present.

On an aspecific way chronic HTLV-1 infection can cause **wasting and autoimmune diseases and anaemias.** Opportunistic infections due to immunosuppression are common, including Pneumocystis carinii, systemic fungal infections, tuberculosis, etc. There is a risk of hyperinfection with Strongyloides stercoralis essentially among patients, who are being treated with corticoids. In regions where HTLV-1 is endemic, various inflammatory and autoimmune disorders, including uveitis, the sicca syndrome, pneumonitis, arthropathy,

and thyroiditis are attributed to this virus. However, more research is needed to confirm these findings.
Diagnosis: specific antibody diagnostical tests, PCR analysis, MRI.
Treatment: symptomatically
RFR method: detects and may eliminate the virus only over a long period of time.
Its resonant frequencies are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz

5.1.10.1.2. Human T-cell Lymphotropic Virus-2 (HTLV-2)

The virus characterized by an extensive nucleic acid homology with HTLV 1, but is distinct from HTLV-1. This virus can transform normal T-lymphocytes and can replicate in both T- and B-cell lines. It was first isolated from a patient with hairy cell leukaemia, but no specific pathology has been attributed to it. Unlike HTLV-1, which is a true human leukemia virus, HTLV-2 has no proven causative role in human lymphoproliferative diseases. However, the discovery of HTLV-2 as a lymphotropic virus suggested its association with various human leukemias. The list of HTLV-2 disease associations is growing. HTLV-2 produces no clear immunocompromised state, which leads to speculate that the virus promotes an enhanced inflammatory response and thereby creates an autoimmune problem. This may also explain links between HTLV-2 and pulmonary inflammations, arthritis, asthma, and dermatitis.
Its resonant frequencies are: 314, 320-324, 370-376, 493-501 kHz

5.1.10.1.3. Human T-cell Lymphotropic Virus type 3 (HTLV-3)

Initially, HTLV-3 was isolated in association with AIDS, but proved to have pathogenic and genetic characteristics distinctly different from those of HTLV viruses, therefore its name changed to HIV. Recently, a true third HTLV, which bears resemblance to simian T-cell leukemia virus 3 (STLV-3), was isolated from a pygmy in southern Cameroon.
Its resonant frequencies are: 307, 312, 320-324, 338-340, 365-367, 397-400, 416, 428, 435, 453-455, 484, 526-530 kHz

5.1.10.1.4. Human T-cell Lymphotropic Virus type 4 (HTLV-4)

A fourth HTLV has also been described in Africa bushmeat hunters, but it has no similar simian counterpart. (Initially the former HTLV-4 was supposed to be related with HIV2.)
Its resonant frequencies are: 297, 454, 540-545 kHz

5.1.10.1.5. Human T-cell Lymphotropic Virus type 5 (HTLV-5)

HTLV-5 DNA sequencies has been isolated from tumor cells of patients with Tac-antigen-negative cutaneous T cell lymphoma-leukemia (mycosis fungoides).
Its resonant frequencies are: 297-298, 315, 320-340, 354, 439, 480-482, 523, 544-545 kHz

5.1.10.1.6. Human T-cell Lymphotropic Virus type 6 (HTLV-6)

HTLV-6 is associated with immunesuppressive states and therefore with tumor genesis.
Its resonant frequencies are: 359, 374-376, 382-383, 474-476, 570-578 kHz

5.1.10.2. Lentiviruses

Lentiviruses are retroviruses differing from the HTLVs, infecting also human beings, causing a variety of diseases, including immunodeficiencies, neurological degenerations, and arthritis. There are known two species in this genus called Human Immunedeficiency Virus-1 and -2, these can cause emergent illnesses.
The frequencies of HIV-1 are: 317-319, 365, 371-372, 383, 396, 402, 450, 474-478 kHz
The frequencies of HIV-2 are: 318, 365, 372, 383, 396, 402, 426-430, 450, 508-516 kHz

The frequencies of HIV-3-4 are: 349, 365, 424, 460, 544-556, 569 kHz

5.1.10.2.1. Human Immunedeficiency Viruses (HIVs, or Lymphadenopathy Associated Viruses (LAVs), or AIDS Associated Retroviruses (ARVs)

The infections of the Human Immunodeficiency Viruses are caused by viruses progressively destroing lymphocytes, resulting in the **acquired immunodeficiency syndrome (AIDS)** and other severe diseases – such as Kaposi's sarcoma – as a consequence of an impaired immunity. The failure of the immune system that allows the **growth of rare cancers** and the **development of rare severe infections** has come to be known as AIDS. The disease is sexually transmitted. Sexual transmission can occur when infected sexual secretions of one partner come into contact with the genital, oral, or rectal mucous membranes of the other person. Infection can also happen via contaminated blood, by blood transfusions or by using infected needles. Transfer of the virus can occur from an infected mother to her child in utero or during the birth and in case of breast feeding.

Three groups of HIV-1 have been identified on the basis of differences in envelop: M, N, and O Group. (HIV-3 is an uncommonly used term designating HIV-1 subtype O). Group M is the most prevalent. It is subdivided into eight clades, based on the whole genome, which are geographically distinct. The most prevalent of them are subtypes B (found mainly in North America and Europe), A and D (found mainly in Africa), and C (found mainly in Africa and Asia); these subtypes form branches in the phylogenetic tree representing the lineage of the M group of HIV-1. Coinfection with distinct subtypes gives rise to circulating recombinant forms (CRFs).

The genetic sequence of HIV-2 is only partially homologous to HIV-1 and more closely resembles that of SIV (Simian Immunodeficiency Virus infects primates) than HIV-1. HIV-2 is less transmittable and is largely confined to West Africa.

All HIV viruses can produce emergent diseases. In order to establish infection in a person, the viruses enter lymphocytes. Being retroviruses, they store their genetic information as RNA rather than as DNA. After entering a target host cell the virus releases its RNA and reverse transcriptase enzyme, and then produces DNA, using the viral RNA as a pattern. The viral DNA is thus incorporated into the DNA of the host cell. Each time a host cell divides, it also makes a new copy of the integrated viral DNA along with its own genes. The viral DNA can take over the functions of the cell, causing the cell to produce new viral particles. These new viruses are released from the infected cell to invade other cells. HIV can infect a variety of immune cells such as CD4+ T cells, macrophages and microglial cells. The virus attaches first to lymphocytes that have a receptor protein, called CD4, in their outer membrane. Cells with CD4 receptors are usually called CD4 positive cells or helper T-lymphocytes. Helper T-lymphocytes serve to activate and coordinate other immune cells such as B-lymphocytes producing antibodies, and cytotoxic CD8 positive T-lymphocytes. All of them are helping to invading organisms and destroy cancerous cells. The acute viremia is associated in patients with the activation of CD8+ T cells, which kill HIV-infected cells, and subsequently with antibody production, or seroconversion. A good $CD8^+$ T cell response is linked to slower disease progression and a better prognosis, though it can not eliminate the virus. During the acute period (usually 2-4 weeks post-exposure) among most individuals develops an influenza or mononucleosis-like illness called acute HIV infection, which most common symptoms include fever, lymphadenopathy, pharyngitis, rash, myalgia, malaise, mouth and esophagal sores, headache, nausea with vomiting, enlarged liver/spleen, weight loss and neurological symptoms. Infected individuals may experience all, some, or none of these symptoms. A strong immune defense reduces the number of the viral particles in the blood stream, the infection enters into a clinical latency stage. The clinical latency time can vary between two weeks and 20 years. During this phase, the HIV is active within the lymphoid organs, where large

amounts of virus become trapped in the follicular dendritic cells (FDC) network. Macrophages and microglial cells are the cells infected by HIV in the central nervous system. The surrounding tissues that are rich in $CD4^+$ T cells may also become infected, and viral particles accumulate both in infected cells and as a free virus. The patients being in this phase are still infectious. The destruction of CD4 lymphocytes reduces the ability of the immune system to recognize new invaders and target them for attack. When $CD4^+$ T cell numbers decline below a critical level, cell-mediated immunity is lost, and infections of a variety of opportunistic microbes appear.

Several opportunistic infections and cancers are typical of the onset of AIDS, e.g. severe yeast infections (as Candida albicans) of the mouth, vagina and esophagus. Pneumonia caused by the fungus Pneumocystis carinii is a typical and recurring process. Chronic toxoplasmal and mycoplasmal infection persisting since childhood is common. Cryptosporidium, a parasite that may be acquired from contaminated food or water, often causes diarrhea by AIDS patients. Viruses that commonly infect people with AIDS include Cytomegaloviruses, Epstein-Barr Virus, and Herpes simplex virus-1 and -2.

Diagnosis: by HIV-1 testing (ELISA, IFA, Westernblot. PCR)

Treatment: antiretroviral drogs can only slow down the pathological process and have side effects; symptomatically

RFR method: detects and eliminates the virus

The resonant frequencies of HIV viruses: The normal pathological range of resonance frequencies of the primitive retroviruses and other informations about retroviruses can be found in Chapter 23. In particular, the retrovirus building up by the human DNA has a higher resonance frequency.

This range of frequency is the same as that of the human low range: 365-368, 383-384, 389-390, 393, 396 kHz

Much routine and practice is required to eliminate retroviruses effectively with the RFR method. Extensive observation and supervised practice are a prerequisite for undertaking treatment of a patient using these higher frequencies. After the first successful treatment process, recheck monthly for one year for retroviruses. Then check twice a year.

5.1.10.3. Human Endogenous Retroviruses (HERVs)

The silent regions in human DNA genom contain perhaps 50000 or more endogenous retroviruses and retroviral sequences which have entered the human genome. All appear to be defective, containing nonsense mutations or major deletions, and cannot produce infectious virus particles. They are called human endogenous retroviruses, or HERVs.

HERVs are suspected of involvement in some autoimmune diseases, in particular with Multiple Sclerosis. Investigations also suggest possible HERV involvement in the HELLP syndrome and pre-éclampsia.

Human Mammary Tumor Virus, or HMTV DNA sequences appear in about 90% of the human breast cancer patients tested and that the pro-viral DNA appears in the normal blood cells of substantially all of these patients.

The resonant frequencies of the Human Mammary Tumor Virus are: 314-318, 350, 368, 383, 385, 387, 389-390, 393, 396, 406, 408, 427-430, 444, 450, 469, 475-477, 554, 568, 578 kHz

5.2. The Most Frequent Human Viral Diseases Caused by DNA Viruses

5.2.1. Poxviruses – helical nucleocapsid with envelop dsDNA

Poxviruses are the largest in size and most complex viruses. Infections due to poxviruses occur in human persons and animals.

The **Orthopoxviruses** include smallpox (variola), monkeypox, vaccinia, cowpox, buffalopox, cantagalo, and aracatuba viruses.

The **Parapoxviruses** include the following: orf virus, bovine papular stomatitis virus, pseudocowpox virus, deerpox virus, and sealpox virus.

The **Yatapoxviruses** include the tanapox virus and the yabapoxviruses, which latter are primarily found in Africa.

The **Molluscipoxviruses** include the human molluscum contagiosum virus.

The **Vaccinia virus**, used for vaccination, can also infect human beings.

Smallpox and molluscum are specifically human viruses. Other poxviruses cause rare zoonotic infections in man.

Poxviral infections (like smallpox, vaccinia, monkeypox and cowpox) cause localized or generalized vesicular exanthem in human beings. The skin lesions form pustules, followed by scabbing and healing. The remaining poxviruses cause localized nodules in the area of the inoculation.

5.2.1.1. Poxvirus Hominis – Smallpox (Variola vera)

Smallpox was once an acute, severe, febrile, highly contagious infectious disease, caused by the poxvirus hominis. By vaccination, succeeded to eradicate smallpox worldwide.

This disease generally presented itself in 2 clinical forms, i.e. in the *Variola major*, the fatality rate of which amounted to cc. 30% and in an other, similar, but milder one, known as *Variola minor*, or Alastrim, which had a fatality rate of cc. 1%. Though the difference in the severity between these two diseases was apparent, the agents of smallpox and alastrim were biologically and immunologically indistinguishable from each other in the laboratory.

Symptoms were as follows: The virus gained access to the body through the respiratory tract, (or through a direct contact with the smallpox lesions of the skin and the mucous membranes, or through a contact with materials (e.g. bedding, clothing) contaminated by such lesions or by scabs) and multiplied itself in unidentified places, most likely in the lymph nodes or the liver. After several days, during which there was no evidence of infection, viremia ensued, with the swelling of the endothelium of the blood vessels in the corium resulting in a perivascular inflammation. Loculated vesicles were the result of the cellular destruction and exsudation of the serum. The initial symptoms of smallpox included the acute onset of fever, chills, headache, nausea, vomiting, and severe muscle aches. This state usually lasted for two to four days and was accompanied at times by a flush of the skin. By the fourth day of illness, the fever subsided and the characteristic smallpox rash appeared, forming vesiculopustules. During the pustulous phase, fever generally returned and the pustules began to form scabs. Of the Variola major disease people usually died between the 5th and 7th day of the illness. Two more serious forms of smallpox were also identified. In its most severe form (in cc 3% of persons afflicted with variola major) known as *purpura variolosa or hemorrhagic-type smallpox*, the initial stage of the illness (before the rash appears) was accompanied by a dark, purplish, blotchy flush of the skin. People with purpura variolosa usually experienced a severe loss of blood into the skin and the internal organs (i.e. hemorrhage), causing death already before the typical smallpox rash appeared. The other rare and deadly form of smallpox, referred to as *flat-type smallpox,* affected about 5% of patients suffering variola major. In this form of the disease the lesions developed more slowly and never rose the surface of the skin, remaining soft to the touch.

Prevention: by vaccination

Treatment: no antiviral substances have proved effective for the treatment of smallpox. Patients afflicted with smallpox should be offered supportive therapy as well as antibiotics, indicated to treat occasional secondary bacterial infections.
RFR method: detects and may eliminate the virus.
Its resonant frequencies are: 291, 302, 326-340, 363-372, 396, 403-410, 420, 426, 448-451, 482-486, 518-522, 545, 554, 576 kHz
Look for the virus during the vesicular and pustular phases of the disease, then detect and eliminate them.

5.2.1.2. Poxvirus Officinale (Vaccinia virus)

Vaccinia infections result from iatrogenic or accidental inoculation of the virus. The infection initially appears on the skin as a localized maculopapular lesion, wich evolves into vesicles and pustules, which then resulting in scabs, healing with scarring. Patients may have fever and regional lymphadenopathy. In case of patients with some skin damage (e.g. atopic dermatitis, ekzema, or some other dermatitis, mechanic wounds etc.), the vaccinia virus can cause ekzema vaccinatum. In some cases or as regards patients with some immune deficiency, the infection might disseminate. Constitutional symptoms may be severe, with high fever and generalized lymphadenopathy, moreover, death can be common concerning these patients.

5.2.1.3. Monkeypox Virus

Monkeypox viral infections (which generally occur in villages in tropical regions) can produce a disease similar to variola minor clinically indistinguishable from it.

5.2.1.4. Infections Caused by Other Zoonotic Poxviruses

Other human poxviral infections are f. i. the **cowpox**, which causes a contagious pustular dermatitis; the **bovine papular stomatitis**, the pseudocowpox **(milker's nodule)**, the **sealpox**, the **tanapox**, and the **yabapox**. All of them are rare zoonotic infections, resulting from cutaneous inoculations and from the close proximity of human beings with animals. The cowpox causes a localized pustular skin lesion the course of which is similar to that of uncomplicated vaccinia infections. The remainder of the infections produce a localized nodular lesion that resolves after some time.
Prevention: vaccination might be effective
Treatment: no antiviral substances have proved to be effective concerning these infections as yet.
RFR method: detects and may eliminate the virus.

5.2.1.5. Molluscipoxvirus - Molluscum Contagiosum

This poxvirus is pathogen solely touching human beings, transmission happens mostly among children or by sexual contact among young adults, causing a benign disease of the skin.
Symptoms: By infected patients develop single or multiple discrete, painless, small pearly nodules (cc. 1-2 mm in size) in the epidermis. These nodules have usually a characteristic central umbilication. The infection generally ceases after some time.
Treatment: no therapy is universally effective as yet, treatment with systemic or topical antiviral drogs, the destruction of the lesions with a variety of chemical or physical agents may be beneficial.
RFR method: is only needed in immunodeficient cases, when the numerous nodules last fore a too long time. Detects and eliminates the virus.
The most frequent resonances are: 290-291, 307, 319-320, 325, 332-338, 348, 363-372, 376, 396, 401-410, 420, 425-426, 454, 482-486, 448-451, 518-522, 544-545, 551-554 kHz

5.2.2. Papova Viruses – cubical nucleocapsid without envelop

The group of papova viruses comprises a number of small icosahedral virus species, all isolated from mammals. The members of this group, such as the papilloma, polyoma and the so-called vacuolating Simian vacuolating virus 40 (SV-40) are all oncogenic, capable of transforming cells in vitro.

5.2.2.1. Human Papilloma Viruses (HPVs)

More than 100 different human papillomavirus types have been identified till now. These most significant human papova viruses cause warts and various tumours (see also Chapters 26.). Papillomaviruses multiply exclusively on human body surfaces, such as the skin, or the mucous membranes of the genitals, anus, mouth, or airways. Most people are infected with some strain of HPV during their lives. All HPVs are transmitted by skin-to skin contact.

Most papillomavirus types are liable to infect particular body surfaces. For example:

HPV types 1, 2, 4 tend to infect the skin of the soles, **types 1, 2, 7** the palms, feet, elbows etc. or **types 3, 10** infect the skin of the face, where they may cause **plantar, common or flat cutanous warts** (verruca vulgaris, verruca plana). In general, these wart-causing HPV types are not associated with an increased risk of cancer.

HPV types 6 and 11 are adapted to infect the genitals, where they can cause genital warts known as **condyloma accuminatum**, or can cause a rare condition known as **recurrent respiratory papillomatosis** (so also types 7, 16, 32), forming warts on the larynx or on other areas of the respiratory tract. These warts can recur frequently, may require repetitive surgery, may interfere with one's breathing, and, in extremely rare cases, can even alter to cancer.

Its resonant frequencies are: 438-448, 452-453, 459-464, 476-479, 517-521, 525-527, 538, 576 kHz

HPV types 13, 32 can cause **oral focal epithelial hyperplasia.**

Epidermodysplasia verruciformis is a hereditary disorder of the skin characterized by chronic infections with human papillomaviruses *(more than 15 sort of HPV types).* Skin eruptions of flat to papillomatous, wartlike lesions and reddish-brown pigmented plaques disseminated on the body and the face are characteristic.

About 30-40 HPV types have evolved to be transmittable through sexual contact infecting the anogenital region though some of them cany infect the genitals without causing any noticeable signs of an infection.

A persistent infection with a subset of about 13 so-called „high-risk" sexually transmitted **HPV types 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 68** may lead to the development of **cervical, vulvar, penile, or anal intraepithelial neoplasia**. These are precancerous lesions and can progress and become invasive cancer. Cancers caused by such „high-risk" HPV types kill several hundreds of thousands people per year worldwide.

Several types of **HPV, most particularly type 16,** have been found to be associated with **oropharyngeal squamous-cell carcinoma** (a form of head and neck cancer). HPV-induced cancers often have viral sequences incorporated into the cellular DNA. Some of the HPV „early" genes, such as E6 and E7, are known to act as oncogens which promote tumor growth and malignant transformation.

Prevention: by vaccination preventing cervical cancer (living in sexual monogamy, the use of condom and microbicids)

Treatment: no therapy is universally effective yet, systemic or topical treatment with antiviral drogs, immunomodulating agents, destruction of the lesions with a variety of chemical or physical agents, operation, etc.

RFR method: can detects and eliminate the virus
The most frequent resonances are: 314-319, 343-347, 401-410, 418-426, 427-438, 442-448, 452-453, 456-466, 467-479, 488-496, 501-507, 513-521, 525-527, 533-545, 556-564 kHz

5.2.2.2. Human Polyomaviruses

All known human polyomaviruses are linked up with cancer.

5.2.2.2.1. JC Virus

This virus can solely be found in human beings. It spreads throughout the human population. Its infection rarely results in disease, excepting the immunosuppressed people. Infecting JC virus leads to **progressive multifocal leukoencephalopathy,** one of the leading causes of death of those with HIV. It can also cause persistent **urinary tract infections**. Most people are subclinically infected with the virus already in childhood.
Its resonant frequencies are: 334-338, 361, 379-383, 395-398, 467, 475-480, 488-489, 550-553, 578-581 kHz

5.2.2.2.2. BK Virus

BK virus was isolated in a renal-transplanted patient. It can also often be found in the **urinary tract** of immunosuppressed individuals, but is not associated with the progressive multifocal leukoencephalopathy. This infection tends to occur early in life, and resulting in mild respiratory illness concerning children.

5.2.2.2.3. Simian Vacuolating Virus (SV-40)

It is not yet clear how and in which way the SV-40 infection is originated from monkeys to man. SV-40 DNA was discovered **in human tumours**, specifically in mesotheliomas, brain tumours, osteosarcomata and ependyomas.
Its resonant frequencies are: 331-332, 338-339, 343-345, 360-362, 379-380, 385-387, 405-407, 425-426, 440-443, 447, 450-451, 453-457, 480-481, 467-489, 494-498, 552-554 kHz

5.2.2.2.4. Merkel Cell Polyomavirus

This polyomavirus is strongly associated with a neuroendocrine cancer of the skin – also known as Merkel cell carcinoma.
Treatment: no antiviral substances have been proved effective against these infections
RFR method: detects and may eliminate the virus.
Its resonant frequency is: 443 kHz
The list is not complete.
As to the resonant frequencies of Papilloma viruses: see also Chapter 26.1.6.

5.2.3. Adenoviruses – cubical nucleocapsid without envelop dsDNA

There are 49 immunologically distinct Adenoviral types that can cause human infections. Although the epidemiologic characteristics of the adenoviruses vary in type, all are transmitted by direct contact, fecal-oral transmission, and occasionally waterborne transmission. Most Adenovirus infections involve either the respiratory tract or the gastrointestinal tract or the eye.
Symptoms: Adenoviral infections are very common and mostly asymptomatic. Adenoviruses types 1, 2, 3, and 5 are mostly prevalent in infants and children; to be found in the throat and rectum of both sick and healthy children. Adenovirus-associated illnesses

occur throughout the year and account for about 10 to 20 percent of all **respiratory illnesses** concerning infants and children, but its highest frequency throughout the time between fall and spring. The febrile pharingitis due to adenoviruses usually occurs sporadically or in small outbreaks among children.
Pharyngoconjunctival fever is febrile pharingitis associated with acute follicular conjunctivitis. This disease occurs in summer epidemics, frequently among children exposed to viruses in swimming pools. The adenoviral acute respiratory disease is a respiratory illness of military recruits, caused principally by adenoviruses types 4 or 7, following which, pneumonia can develop. This usually occur in winter and spring outbreaks.
Other types cause sporadic infections and occasional outbreaks; for example, **epidemic keratoconjunctivitis** is associated with adenovirus serotypes 8, 17, 19, and 37. Epidemics of febrile diseases with conjunctivitis are associated with the waterborne transmission of some adenovirus types, caused by inadequately chlorinated swimming-pools and small lakes. Enteric adenoviruses 40 and 41 cause **gastroenteritis**, usually as regards children.
Prevention: thorough handwash, desinfection, in some cases vaccination
Treatment: symptomatically
RFR method: detects and may eliminate the virus.
The most frequent resonances of adenoviruses are: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz
General resonances of adenoviruses are: 370-387, 393 kHz

5.2.4. Herpesviruses – cubical nucleocapsid with envelop, dsDNA

The members of the Herpes virus family are the most frequent causes of human viral diseases, second only to influenza and the cold viruses. They are capable of causing overt diseases or to remain latent for many a year, only in order to be reactivated again. The family is divided into 3 sub-families: the Alpha-, Beta, and Gammaherpesvirinae. Eight or more herpes virus types are known to infect man frequently.
The definitive eliminating of the herpesviruses by RFR method can only occur, if any and all other immunosuppressive viruses and bacteria (f.i. retroviruses, HPVs, other kinds of herpesviruses, mycoplasmas etc.) infected the patient will also be treated.
The most frequent variants of the Herpes Simplex Viruses resonances are:
Herpes Simplex Virus-1: 290-294, 344-346 kHz
Herpes Simplex Virus-2: 352-365, 413, 425 kHz

5.2.4.1. Herpesvirus Hominis: Herpes Simplex-1 (HSV1) and Herpes Simplex-2 (HSV2)

The HSV1 and HSV2 are alphaherpesviruses, aiming at the mucoepithelia, causing blisters on the skin. They establish diverse relations with human beings. An acute disseminated primary infection (called gingivostomatitis), or chronic infections with continual viral shedding (named herpes keratitis and herpes labialis), being though a clinical reactivation, may also occur. The virus can infect the skin, the mucoepithelia, the macrophages and the lymphocytes. Once the epithelial cells are infected, a multiplication of the virus around the lesion will begin and will enter into the innervating neuron. The virus can proceed along the exocytotic pathway or can enter the cytoplasm and be released by cell lysis. It also appears to be able to pass through intercellular junctions, spreading thereby from cell to cell. Phagocytosis of the virions by susceptible cells, i.e. viropexis, precedes the digestion of viral envelopes and proteins. After the initiation of the infection, the absorption of the virus is completed within three hours. A new viral infectivity arises sharply from the sixth to the ninth hour, after which it calms down. The viral DNA enters the nucleus of the host

cells, where a new viral DNA is synthesized. The viral proteins are synthetized in the cytoplasm and migrate to the nucleus. This cyclic process is very important when determining the way of the RFR method.
HSV1 and HSV2 first infect the cells of the mucoepithelia or get entrance through wounds. The locus of the initial infection depends on the way in which the patient acquired the virus. The virus is found in the lesions on the skin but can also be present in a variety of body fluids, including saliva and vaginal secretions. Though the eruption subsides, the virus remains in an inactive latent state inside the ganglia in the nervous system, supplying the sensory nerves of the infected area A large proportion of the population is evidently HSV1-infected, judging by the presence of specific antibodies. The HSV2 normally spreads by sexual intercourse but is found in the anus, rectum and the upper alimentary tract too, as well as in the genital area. Moreover, even an infant can be infected at its birth by an infected mother. The infant can also get infected in utero, if the mother's infection spreads. Because of the infant's immature immune system, the resulting infection can be very serious and can sometimes even lead to death.
Symptoms: In case of the **primary herpetic gingivostomatitis,** the typically clear lesions develop first and are then becoming wounds. The infection begins often on the lips, spreads over to every part of the mouth and pharynx. The initial lesion is a clear vesicle, containing infectious viruses with a red (erythematous) lesion at the base of the vesicle. The infection recurring now and then, produces small, painful, fluid-filled blisters on the skin or on the mucous membranes. An **oral herpes** is the most frequent clinical picture of the infection. Recurring eruptions can be triggered due to overexposure to sunlight, or owing to the presence of another illness, by physical or emotional stress, by the weakening of the immune system, or due to certain foods and drugs, and/or by fatigue. The **herpes keratitis** is an infection of the eye and is primarily caused by HSV,1 can be recurrent and may lead to blindness. An **eczema herpeticum** can develop in case of atopic dermatitis of children, can spread over the skin in the area of the eczema lesions. The virus can then spread to other organs, such as the liver and the adrenals. The **HSV encephalitis** is usually the result of an HSV1 infection and is the most common sporadic viral encephalitis. It is a febrile disease and may result in the damage of one of the temporal lobes. The disease can be fatal.
The **genital herpes** is usually the result of HSV2 and, in about 10% of the cases, the result of HSV1. The primary infection is often asymptomatic, but many painful lesions can develop on the glans or on the shaft of the penis in men and on the vulva, vagina and cervix of women. In regard to both sexes, the urethra and the perianal region can be involved.
The **HSV proctitis** is an inflammation of the rectum and the anus. The **HSV Meningitis** can be the result of an HSV2 infection. The symptoms end spontaneously.
A HSV infection of neonates resulting from HSV2 can often be fatal, though such infections are rather rare. An infection is possible especially if the mother is shedding viruses at the time of her delivery. Because of the neonate's underdeveloped immune system, the virus can spread rapidly to many peripheral organs (e.g. lungs and liver) and can infect the central nervous system too.
Diagnosis: according to the symptoms or using PCR techniques, by serological methods, etc.
Treatment: by using local or systemic antiviral therapy
RFR method: detects and may eliminate the viruses.
The general frequencies of Herpes Simplex Virus-1 are: 290-294, 344-346 kHz
Its other frequencies are: 307, 328, 331-339, 346-350, 357-358, 370-372, 383, 396-403, 413, 418, 420, 431-433, 438, 447-458, 463, 476, 478, 480-490, 533 kHz
The general range of Herpes Simplex Virus-2 is: 352-365, 413, 425 kHz

Other frequencies of the Herpes Simplex Virus-2 are: 300-301, 310, 337-350, 366-368, 374-378, 380-381, 396, 403, 425, 432-434, 450-459, 474, 496, 540-552, 568 kHz
The resonant frequencies of other, non differentiated Herpes Simplex Virus species are: 290-294, 300-307, 310, 313, 318, 328, 331-340, 343-345, 360-375, 377-378, 380-383, 396-403, 413-425, 431-433, 449-450, 458-459, 463, 474-478, 483-492, 527, 533, 540-552, 568 kHz

5.2.4.2. Varicella Zoster Virus (VZV or HHV-3)

This virus is an alphaherpesvirus which causes two diseases, the chicken-pox (Varicella), usually affecting children, and the shingles, occuring later in life. The Shingles (Zoster) is an illness caused by the reactivation of an earlier varicella infection.

5.2.4.2.1. Varicella (Chickenpox)

Chickenpox is a highly contagious, infectious illness, which spreads by airborne droplets of moisture containing the varicella zoster virus. An infected person is most contagious just when the symptoms begin, and remains so until the last blisters are crusted. Isolation of an infected patient helps to prevent the spread of infection to others.
Symptoms: The infection occurs usually *among infants*. The first symptoms include a rash of small, flat, red areas appearing one after the other on the whole body and face, along with headache, moderate fever and a feeling of illness. The spots soon become raised, form an itchy, round, fluid-filled blister against a red background, and, finally, crust. The lymphnodes at the side of the neck may become enlarged and tender. *VZV pneumonia* is a serious complication that might occur, mostly in case of adults, newborn infants, or any person with an impaired immune system. The heart and the joints may be involved, heart murmur and joint pain can develop. The liver may become inflamed, though usually, there are no symptoms. A bacterial superinfection of the skin lesions may also come about, such as, pyoderma, erysipelas or impetigo. Encephalitis is a rare complication, causing headache, vomiting, unsteadiness in walking, confusion and seizures. The Chickenpox *regarding adults,* is often more severe than in case of children, more profuse rash, higher fever, and a greater incidence of pneumonia. The **Connatal Varicella syndrome** may develop, in case of infection in utero, during the first trimester. The connatal varicella syndrome leads to the scarring of the skin of the limbs, causes damage to the crystalline lens, retina and brain and can cause microphthalmia. In early pregnancy abortive complications might develop.
Prevention: can happen by vaccination. Antibodies against the varicella virus may be given to immunesupprimated people, who were not vaccinated and run thus a higher risk of getting complications.
Treatment: symptomatically and with antiviral drogs
RFR method: solely in case of an impaired immune system or encephalitis.
Its general range is: 416-421 kHz
Its other resonant frequencies are: 293, 310, 339-340, 372, 383, 396-398, 402, 405-410, 415-420, 451, 460, 467, 474, 477, 544, 555 kHz

5.2.4.2.2. Herpes Zoster (Shingles)

Shingles is caused by the same VZV that causes chickenpox. The first infection with the varicella zoster virus happens in the clinical form of the chickenpox, and ends with the entering of the virus via the nerves into the spinal or cranial ganglia of the area in which the virus multiplied. The virus remains latent there, and may never cause symptoms again, except in case of being reactivated again, many years later. The virus may be reactivated due to stress, or owing to some immune deficiencies.
Symptoms: Herpes zoster is characterized by the segmental inflammation of the spinal or cranial nerves and their ganglions, and by a painful, localized, vesicular eruption of the

skin responding to the involved nerve. The recurrence of a varicella is accompanied by a severe, radicular pain in the area, innervated by the nerve in which the reinfection had occurred. A few days later, chickenpox-like lesions appear in restricted areas (dermatome) which are innervated by that ganglion. The skin lesions are somehow different from those observed in case of chickenpox, being small and close together and maculopapulous with an erythematous base. The skin lesions heal usually in about two weeks time, but scarring may develop even in simple cases. In addition to the sensory disturbances, a temporary paralysis of the muscles, innervated by their intercostal, or cranial, or periferial nerves, can occur. Especially in case of elderly patients, the reactivation can lead to a chronic burning or itching pain, called post-herpetic neuralgia. The pain may last well after the rash got healed (even for months or years). An increased sensitivity to touch (hyperesthesia) is often associated with the post-herpetic neuralgia. In case of a severe immune deficiency, a generalized herpes zoster dissemination may develop, involving the visceral organs and resulting in death.
Prevention: vaccine is available
Diagnosis: according to the symptoms.
Treatment: with systemic and local antiviral agents as well as symptomatically
RFR method: detects and may eliminate the virus.
The general range of Herpes Zoster Virus is: 416-421, 544-555 kHz
Its other frequencies are: 293, 310, 339, 348, 372, 383, 396-398, 402, 409-410, 450, 460, 467, 474, 477 kHz

5.2.4.3. Epstein-Barr Virus (EBV or Human Herpes Virus-4)

The Epstein-Barr Virus is an ubiquitous gammaherpesvirus and the causative agent of the Infectious Mononucleosis in the west, the Burkitt's lymphoma in Africa, and the Nasal Pharyngeal Carcinoma in the orient. The virus only infects a small number of cell types, those f.i. which can express the receptor for complement C3d component (CR2 or CD21). These are certain *epithelial cells (of the oro- and naso-pharynx) and B lymphocytes.* This explains the cellular tropism of the virus.
The epithelial cells permit the complete lytic replication of the virus.
The B lymphocytes are only semi-permissive for the multiplication of the virus, the infection may either be latent, or the cells may be stimulated and transformed by the virus. In case of lymphocytes latently infected, the B-cell contains a few unintegrated copies (episomes) of the virus genome, which multiplicate every time the cell divides. In this case the early immediate genes are expressed, the EBV nuclear antigens included. In addition, two latent membrane proteins are also expressed. These membrane proteins are oncogenic.

5.2.4.3.1. Mononucleosis Infectiosa (Infectious Mononucleosis)

The Infectious mononucleosis is an acute and, usually, a benign infectious disease, caused by the Epstein-Barr Virus. Mostly affecting teenagers or young adults, the virus is transmitted by saliva and by close contact (kissing disease) or following intimate contact with someone infected with the EBV. The primary infection can be asymptomatic. A large proportion of the population is infected with EBV, these people, though usually free of symptoms, will shed the infectious virus from time to time throughout all their life.
Symptoms: In symptomatic cases of the infection the disease develops after 1-2 months of latency. It occurs generally among adolescents and young adults and is characterized by malaise, fatigue, fever, enlarged, generalized lymph nodes, sore throat with tonsillitis, enlarged spleen and liver. A maculopapulous rash can also be present. The fever usually persists for more than a week. An increased number of peripherial lymphocytes is present in the blood with a high proportion of atypical cells. The seriousness of the disease often depends on age. If younger patients are affected, the disease subsides more quickly (usually within 1 to 4 weeks). Complications may develop, including neurological

disorders, such as meningitis, encephalitis, myelitis and the Guillain-Barré syndrome. Secondary infections, autoimmune hemolytic anemia, thrombocytopenia, agranulocytosis, aplastic anemia may also occur. A chronic syndrome with symptoms similar to the chronic fatigue syndromes (i.e. headaches, low fever, fatigue, malaise and sore throat) may also develop and persist for months or even longer. EBV may play a role in the development of tumors forming in B-cells, affecting persons with an impaired immune system, e.g. if organ-transplanted or suffering from AIDS.
Profilaxis: vaccine is not yet available, but about to be developed
Diagnosis: The symptoms of the infectious mononucleosis are not specific and may resemble those of certain other infections (e.g. CMV, Rubella, Toxoplasma, Hepatitis etc.). Blood test can confirm the diagnosis of infectious mononucleosis just like laboratory examinations can detect specific antibodies.
Treatment: symptomatically, there are no drugs available yet to treat EBV infections.
RFR method: eliminates the Epstein-Barr Virus.
The general range of Epstein-Barr Virus is: 370-384 kHz
Its other frequencies are: 337-340, 342-347, 352, 372, 377-380, 389-397, 422, 424, 438, 491, 516, 518, 528, 560 kHz

5.2.4.3.2. Burkitt's Lymphoma

This lymphoma caused by EBV is endemic in equatorial Africa occuring rarely elsewhere. There is probably also a genetic reason for this, possibly involves an association with malaria. Persons who are resistant to malaria appear to be susceptible to develop this lymphoma. In case of infection, the virus is multiplied in pharyngeal epithelial cells, shed into the saliva, and is ingested by CD21+ B lymphocytes. This results in the inhibition of T-cell responses promoting the growth of the B-cells and the IgG secretion. Moreover, the virus stimulates also the cells to produce other cytokines, including IL-5 and IL-6. This lymphoma is the most common malignant tumor of childhood in Central Africa and one of the most aggressive ones of all cancers suffered by the mankind.
Its most frequent resonances are: 337, 339-347, 352, 372-382, 397-398, 403-404, 422, 424, 476, 491, 516, 518, 528, 560 kHz

5.2.4.3.3. Nasopharyngeal Carcinoma (Caused by EBV)

This disease, also associated with EBV occurs in south China, Alaska, Tunisia, and East-Africa. There may be a genetic predisposition to develop EBV cancers among these population or there might be an environmental cofactor involved.
Its most frequent resonances are: 372-383 kHz

5.2.4.3.4. EBV Infection-Related Malformations

The **Oral hairy leukoplakia** is an EBV-associated disease, causing lesions in the mouth and, being an opportunist infection, its frequency increases among HIV-infected patients.
EBV is a well-established risk-factor of the **Hodgkin lymphoma**, especially in certain cases of **lymphoma** among organ-transplanted patients or those infected by HIV.

5.2.4.4. Cytomegalovirus (CMV or Human Herpes Virus-5)

Cytomegalovirus is a betaherpesvirus and, appearing only as it would multiplicate only in human cells, *targeting epithelia, monocytes, and lymphocytes*. The virus spreads by contact or in a connatal way, though its transmission can happen due to blood transfusion or tissue transplantation too.
The CMV infection can be acquired already before birth via the placenta, or at any age thereafter.

The cytomegaloviral infection is found in a significant proportion of the population. The seropositivity increases with age. Infected people may harbor the virus in their secretum (like urine or saliva) for months, it may be present in breast milk, stool, cervical mucus and semen, so that it can be transmitted by sexual intercourse too. The virus first infects the upper respiratory tract and then the local lymphocytes. The circulating lymphocytes then spread the virus to other lymphocytes and monocytes in the spleen and the lymph nodes. The virus finally spreads to a variety of epithelial cells including those of the salivary glands, kidney tubules, testes, epididymides and cervix. *The infection is usually asymptomatic.* The virus elicits both the humoral antibodies and the cell-mediated immunity, the infection nevertheless does not seize. After being infected, the virus may be dormant for years but may become active at any time again, and able to cause a disease. During its latency period the virus is present in the monocytes, lymphocytes and possibly in other cells too. Clinical symptoms develop generally only in people having an impaired immune system. The CMV can inhibit the T-cell responses and can have an **immunosuppressive effect** on human beings.
Symptoms: Cytomegaloviral infection can cause severe **connatal diseases**. The infection happens via the placenta causing *damages in the liver or in other organs*, such as f.i. *the brain of the fetus*. The abnormalities include microcephaly, rash, brain calcification and hepatosplenomegaly. These again may result in defective hearing (bilateral or unilateral) as well as mental deficiency. This viral disease can cause the *death of newborn infants* too. Among **perinatally** infected infants, *pneumonia, hepatitis,* enlarged spleen and *retinitis* may develop.
In case of **immunosuppressed** adults, the disease develop as a localized infection e.g. *retinitis.* In addition, an *interstitial pneumonitis, esophagitis, encephalitis* and *colitis* can also be observed in some patients.
Concerning multiple infections, the CMV is frequently associated with bacterial, fungal as well as with various other types of herpesvirus infections, which may cause even more serious symptoms. Concomitant infections with pertussis, toxoplasmosis and Pneumocystis carinii are frequent among immunodeficient patients.
Diagnosis: can happen by antibody tests. By increasing the level of antibodies against the virus, which measured by blood tests taken on several different days, strongly indicates that this virus causes the infection.
Prevention: by restricting the contact between infected children and pregnant women. A vaccine is about to be developed.
Treatment: with antiviral drogs and symptomatically.
RFR method: detects and may eliminate the virus with repeated treatment.
The general range of the Cytomegalovirus is: 406-412 kHz
Its other frequencies are: 305, 327, 345-350, 512, 530-536, 539 kHz

5.2.4.5. Herpes Lymphotropic Virus (Human Herpes Virus-6)

HHV6 is a betaherpesvirus, targeting T-lymphocytes and other cells. It *replicates in B- and T-lymphocytes, megakaryocytes, glioblastoma cells and in the oropharyngeal epithelia.* It can set up a latent infection in the T-cells which can then be activated when the cells are stimulated to divide. The receptor of this virus is not known. It is found worldwide in the saliva of the majority of adults
It infects almost every child at the age of two and they remain infected all their life. The virus can be reactivated if the patient is in an immune-suppressive state.
Symptoms: HHV6 causes **Exanthema subitum**, otherwise known as roseola infantum. This is a common *disease of young children.* Its symptoms include fever and sometimes the infection of the upper respiratory tract and lymphadenomegaly. The symptoms last a few days long, following an incubation period of about 14 days. The fever finally subsides, leaving a maculopapular rash on the trunk and on the neck that lasts a few days longer.

Regarding adults, the primary infection occurs but seldom and is associated with mononucleosis. This virus was originally isolated from *patients with a lymphoproliferative disease*. It may co-infect HIV-infected T4 lymphocytes exacerbating the replication of HIV. HHV6 has been associated with a number of neurological disorders, including encephalitis and seizures. It has been postulated to play a role in the pathogenesis of Multiple Sclerosis and in the chronic fatigue immunodeficiency syndrome.
Treatment: symptomatically
RFR method: is needed only in immunesupprimated cases, detects and eliminates the virus
Its most frequent resonances are: 372, 402, 412-414, 440, 450, 464, 522-528 kHz

5.2.4.6. Human Herpes Virus-7

This virus is bound up with the CD4 antigen and replicates in (CD4+) T-cells and is found in the saliva of the majority of the adult population. Most people get the infection in their childhood without showing any remarkable symptoms, remaining infected all their lives.
Its most frequent resonances are: 290-293, 413, 438-446, 460-462, 473 kHz

5.2.4.7. Human Herpes Virus-8 (Kaposi's Sarcoma Associated Herpes Virus, KSHV)

The HHV-8 was formerly known as Kaposi's sarcoma-associated herpes virus and is found in the saliva of many patients attached by AIDS. The virus is a gammaherpesvirus which infects the peripherial blood lymphocytes. Following the infection,.the virus enters into the lymphocytes, where it remains in a latent state. In the United States and in northern Europe KSHV is an uncommon infection, homo- and bisexual men, however, run nethertheless a high risk of infection. In African countries, the infection is on the contrary spread in a non-sexual way. Young children can be infected, and infection can occur throughout any person's adulthood. Though being KSHV infected, most people are, usually, free from symptoms throughout their life. A healthy immune system will keep the virus in check. Kaposi's sarcoma usually occurs when someone already infected with HHV-8 becomes immunocompromitted due to AIDS, old age or due to medical treatment.
Symptoms: Kaposi's sarcoma is a cancer with nodular, red, purple, brown or black papular lesions causing abnormal tissue patches to grow in the skin, mouth, nose, throat, the gastrointestinal or respiratory tract. Kaposi's sarcoma arises as a *cancer of the lymphatic endothelium* and forms vascular channels with blood in it. Formally, before the *AIDS* epidemic, the lesions usually developed but slowly. Concerning AIDS patients, however, the disease spreads rapidly. A form of Kaposi's sarcoma attaching young African children due to this infection is almost uniformly and rapidly fatal, bleeding can be its emergent complication.
Treatment: Kaposi's sarcoma is usually a localized tumor that can be treated either surgically or by using local irradiation. Chemotherapy and antiviral therapy can be effective in case of AIDS patients.
RFR method: detects and eliminates the virus
The resonance frequencies of the HHV-8 are: 331, 426, 428, 508-509 kHz

5.2.5. Hepatitis B Virus – enveloped ds/ssDNA

Hepatitis B virus (HBV) is a member of the Hepadnavirus family and one of hundreds of unrelated viral species causing viral hepatitis. Originally known as „serum hepatitis", causing rife epidemics in different parts of Asia and Africa, it is found endemic in China and Asia. This virus is less easily transmitted than the Hepatitis A virus. It can be transmitted through contaminated blood or blood products. If proper precautions are taken to ensure a safe blood supply, blood transfusions very rarely transmit this virus.

Transmission occurs commonly only among drug users, among those who share hypodermic needles, as well as between sexual partners, so that it is undoubtedly a sexually transmitted disease. A pregnant woman infected with HBV can transmit the virus to her baby during its birth.
Symptoms: It is thought that most HBV infected people with are able to get rid of the infection without treatment, but there are certainly some who cannot. Among these latter patients an acute, fulminant hepatitis develops with malaise, vomiting and jaundice. Hepatitis B infected people usually get better of their own accord after a few months. However, there may occur a more serious chronic infection occur, causing liver cirrhosis. This condition can lead to a severe liver damage, and sometimes even to death. The liver cirrhosis, caused by HBV may lead to a fatal, primary hepatocellular carcinoma.
Prevention: an effective vaccina is already available
Treatment: with alpha interferon and antiviral drogs
RFR method: detects and may eliminate the virus.
The frequency resonances of Hepatitis B Virus are: 293, 340-341, 372, 384, 392-402, 414-420, 444-454, 488 kHz

5.2.6. Parvoviruses ssDNA

Parvoviruses which infect human being are as follows.
The *RA-1 Virus* is a member of the parvovirus genus, and is associated with rheumatoid arthritis.
The *Adeno-Associated Viruses* (AAV 1-5) belong to the dependo-virus genus which, probably do not cause any diseasesA host cell infection can only come about in case of a coinfection with some other virus (either an adenovirus or a herpes virus). Recently, great attention was turned towards the AAVs as possible vectors for gene-therapy.
The *B19 virus* is a member of the erythrovirus genus, which causes an extremely contagious, though mild childhood illness, named **Erythema infectiosum** (Fifth disease). The B19 viral transmission comes about by droplets or through direct contact with respiratory secretum. A second way of viral transmission is transplacental. About 30% of mothers primarily infected, pass the virus on to the fetus, but usually there does not happen any harm. The virus targets erythroid progenitor cells in the bone-marrow and the spleen, which, when infected, undergo a lysis, resulting in the decline in the number of erythrocytes, lymphocytes, granulocytes, and platelets.
Symptoms: After the period of incubation, a fever may start, following which, a few days later, presumably immune-mediated a rash might develop. It is facial and lace-like disseminated on the trunk and on the extremities disappearing within a few days. Nevertheless, reappearance of these rashes can be seen up to several weeks following the infection with the virus, in response to changes in stimuli, such as, temperature, sunlight and emotional stress. Asymptomatic infection with B19 has been reported in about 20% of cases. The infection is occuring worldwide and throughout whole the year, concerning people of all ages, either in epidemics or as sporadic cases. It is believed that transmission usually occurs during the incubation period, lasting cc. 20 days.
Treatment: usually symptomatically, in case of immunocompromised individuals or a chronic haemolytic anemia antibody therapy or IVIG can be carried out.
RFR method: solely in complicated cases needed. Detects and eliminates the virus.
Its resonant frequencies are: 313, 318, 326, 378, 386, 413-417, 499, 526, 537, 547 kHz

6. HUMAN PATHOGENIC BACTERIAL INFECTIONS

The Phylum of the Proteobacteria

All members of the Phylum of the Proteobacteriae are **Gram-negative, with an outer membrane mainly composed of lipopolysaccharides,** which is often referred to as an endotoxin. Gram-negative bacteria are generally more resistant to antibiotics than are gram-positive bacteria. They are characterized by the presence of a double membrane surrounding each bacterial cell. Gram-negative bacteria have a great facility for exchanging genetic material (DNA) among strains of the same species and even among different species. Most members are facultatively or obligately **anaerobic and heterotrophic.** The phylum is filed into five classes named alphabetically from alpha to epsilon.

The Class of the Alpha Proteobacteria

In the class of the alpha proteobacteriae there are two orders causing illnesses among human beings i.e. *the Order of the Rickettsiales and the Order of the Rhyzobiales.*
The Rickettsiales, also called rickettsiae, is an order of small proteobacteria. Their cultivating is very difficult, as they only grow in tissue- or in embryo-cultures. The members of this order survive only as endosymbionts of other cells. Some are notable human pathogens. The order is divided into two families: the *family of the Rickettsiaceae and the Ehrlichiaceae.*

6.1. The Order of the Rickettsiales

6.1.1. The Most Frequent Rickettsial Diseases Caused by the Members of the Rickettsia Family

The members of this family are obligate intracellular parasites about the size of bacteria. Microscopically they are usually seen as non-motile, Gram-negative, non-sporeforming pleomorphic coccobacilli. Formerly they were regarded as microorganisms, positioned somewhere between viruses and true bacteria. Rickettsiae, pathogenic for human beings maintain themselves in nature by a life-cycle which involves an insect vector as well as an animal (or a human) reservoir. The *Rickettsia genus* and the *Orientia genus* are both members of the Rickettsia family able to cause illnesses among people.
Rickettsia species are carried as parasites by many ticks, fleas and lice. Being bitten by these insects, a rickettsial infection of human beings can likely occur. The majority of Rickettsia bacteria are susceptible to the antibiotics of the Tetracyclin group. The illnesses caused by human rickettsial pathogens are generally filed into the typhus group and into the spotted fever group.

6.1.1.1. Illnesses Caused by the Rickettsia Genus

6.1.1.1.1. Typhus Exanthemicus

Rickettsia prowazekii can cause an epidemic, a recrudescent and a sporadic form of typhus. The classical epidemic form of typhus is a severe, febrile disease transmitted to human beings by body lice.

The Symptoms: are intense headache, continuous pyrexia lasting for about two weeks, a macular skin eruption, appearing approximately on the fifth febrile day, malaise, as well as vascular and neurological disturbances represent the principal clinical symptoms. Neurological symptoms from headache to extreme agitation, stupor and coma. Circulatory disturbances such as tachycardia, hypotension and cyanosis are almost as severe as in Rocky Mountain spotted fever. Ultimately, in untreated cases azotemia often reaches high levels as a result of some vascular and renal failure and death follows in the second week of illness. Furthermore, thrombosis of major blood vessels and cutaneous gangrene develop in a manner similar to that observed in some cases of the Rocky Mountain spotted fever.
The diagnosis is usually confirmed by a combination of clinical, epidemiological and serological testing (IgM and Weil Felix tests)
Treatment: with doxycyclin (or chloramphenicol) and symptomatically
RFR method: detects and eliminates the rickettsia

6.1.1.1.2. Murine (endemic) Typhus
Endemic typhus, also called „flea-borne typhus" and „murine typhus" or „rat flea typhus", is caused by *Rickettsia typhi mooserii*, transmitted by the fleas infesting rats. The rat fleas transmit this pathogen to human persons. Its occurence is sporadic, but worldwide met with; the incidence is insignificant, but higher in rat-infested areas.
Symptoms: The endemic typhus is clinically similar though milder, than the epidemic typhus. The illness is characterized by fever, lasting from nine to fourteen days, chills, headache, a macropapular rash appearing on the third to fifth day, extreme fatigue and myalgia. The triad consisting of headache, chills and pyrexia is usually followed a few hours later by nausea and vomiting. Prostration, malaise and weakness are suffice to enforce in adults to cease working, in contrast to children, whose illness is less grave. Mild symptoms make it occasionally difficult to define the actual onset of the disease. If untreated, the infection can be fatal, especially in those over the age of 50 or in cases of immunosuppressed people. Transient partial deafness occurs occasionally, but localized neuritis and hemiplegia are but seldom encountered. Though neurologic sequelae are usual. Children experience only minimal neurological changes.
Diagnosis: is usually confirmed by a combination of clinical, epidemiological and serological testing (IgM and Weil Felix tests).
Differential diagnosis: Rocky Mountain fever, scrub typhus, Q-fever and other Rickettsial infections.
Treatment: with doxycyclin (or chloramphenicol) and symptomatically.
RFR method: detects and may eliminate the rickettsia.
Its resonant frequencies are: 310, 323, 347, 368-371, 482, 528, 534, 543, 562 kHz

6.1.1.1.3. Rickettsialpox
The Rickettsialpox is a mild, febrile illness caused by *Rickettsia akari*, which is transmitted by mice or other rodents to human beings through a mite vector. Infected areas are found in the USA, Suoth Africa, Korea and the former Soviet Union.
Symptoms: begin about 7-10 days after being infected, as a painless, red, papular skin lesion 1 to 2 centimeter in diameter on the place of the bite of the mite. The lesion becomes later vesicular, surrounded by erythema, which, some days later, followed by sudden raging sets in fever with chills, headache and myalgia, lasting for a week. Regional lymphadenopathy may also be present. The maculopapulo-vesicular exanthema can be generalized, as being abundant, or scant and liable to involve the oral cavity (usually the palate). The disease is mild and healingby itself without leaving scars within about two weeks.

Diagnosis: is usually confirmed by a combination of clinical, epidemiological and serological testing
Differential diagnosis: chickenpox, smallpox, Hand, Foot and Mouth Disease and other rickettsial illnesses.
Treatment: using antibiotics hastens the healing process or symptomatically.
RFR method: detects and may eliminate the pathogen.

6.1.1.1.4. Tick-borne Lymphadenopathy (TIBOLA)

The mild illness is caused by the *Rickettsia slovaca* species characterized by a single lesion (eschar) in the locus of the inoculation (usually the scalp) and by enlarged cervical lymph nodes. The vector of this illness is the Dermacentor tick experienced in Europe. The infections most likely occur among children under 10 years of age and patients bitten in the colder months of the year. Fever, rash asthenia and localized alopecia can also occur.
Treatment: using antibiotics
RFR method: detects and may eliminate the pathogen.
The restriction fragment length polymorphism (RFLP) analysis of the polymerase chain reaction (PCR)-amplified gene fragments can be used for the differentiation of the isolated species of the *spotted fever group (SFG) of rickettsiae.* The most important species of SFG are the Rickettsia rickettsii, the R. conorii, the R. australis, the R. japonica, the R. africae, the R. sibirica, the R. parkeri and the Orientia tsutsugamushi. The infections they cause show similar clinical symptoms, though with certain differencies.
The most frequen resonance: 319-325, 369-371, 470-485, 528-538 kHz

6.1.1.1.5. Rocky Montain Spotted Fever

The Rocky Mountain spotted fever (synonyms include „tick typhus", „Tobia fever", „Sao Paulo fever", „febre maculosa", „fiebre manchada") is the most severe and most frequently reported rickettsial illness in America, remaining also nowadays a serious and potentially life-threatening infectious disease.
Like every as all rickettsial infection, the Rocky Mountain spotted fever is also a zoonotic infection. It is caused by *Rickettsia rickettsii*, transmitted by ticks (mostly by Dermacentor variabilis, the American dog tick or Dermacentor andersoni, the Rocky mountain wood tick) throught their saliva while feeding. The disease can be difficult to diagnose in its early stage, without a prompt and appropriate treatment the disease can be fatal. The pathogen infects the endothel cells, lining the blood vessels of the whole body. The illness is characterized by an increased vascular permeability, which causes oedema, hypovolaemia and hypalbuminaemia.
Symptoms: About two weeks after being infected acute symptoms appear, as severe headache, nausea, fever, and muscle pain. On about the fourth day of having fever, maculopapular and petechial rashes develop on the wrists, ankles, palms, soles and forearms, rapidly extending over the whole body. The vascular injuries of the skin appear initially as blanchable erythematous macules (1-5 mm in diameter). This classical petechial rash does occur usually, though in case of 10-15% of the patients no dermatological involvement can be experiencod (Rocky Mountain spotless fever). The gastrointestinal endothelial cell injury causes abdominal pain, vomiting, diarrhoea and guaiac-positive stools. A massive gastrointestinal bleeding can lead to severe anaemia and even to death. The pulmonary involvement may cause noncardiogenic pulmonary oedema, interstitial pneumonia and an adult respiratory distress syndrome. The CNS manifestations include encephalitis and meningoencephalitis secondary to a vascular injury. Seizures, cranial nerve damage, hemiplegia and peripheral neuritis might sometimes follow.
Delirium, shock, renal failure and even sudden death can in severe cases come about.

The **Diagnosis:** must be decided upon as soon as possible on the basis of the findings both clinical and epidemiological. Treatment should not be delayed by waiting for serological affirmation.
Differential diagnosis: meningococcemia, measles, or some other rickettsial or viral infections.
Treatment: An appropriate antibiotic treatment, using Doxycyclin (or Chloramphenicol) must be initiated, immediately after suspicion of Rocky Mountain spotted fever had arisen.
RFR method: detects and may eliminate the rickettsia.
Its resonant frequencies are: 314-315, 384-390, 402-405, 440-442, 478-482 kHz

6.1.1.1.6. Spotted Fever Diseases Caused by Other Rickettsia Specieses

The disease called **Boutonneuse fever**, (or Mediterranean Spotted Fever, or Marseilles Fever) is caused by the *Rickettsia conorii*. The **Siberian tick typhus** is caused by the *Rickettsia sibirica*. The Astrakhan spotted fever is caused by *R. conorii subsp. Caspia*. The **Australian tick typhus** (or Queensland Tick Typhus) is caused by *Rickettsia australis*. **The Oriental spotted fever** (or Japanese spotted fever) is caused by *Rickettsia japonica*. Their name shows the geographical area, where they are endemic. The target cell of these Rickettsias is the endothelial cell, proliferating within the endothelial cells of small blood vessels causing vasculitis. Their vectors are ticks, the illnesses they cause show all the same symptoms. Their symptoms are mild and in most cases healiIng by itself.
Symptoms: After an incubation period of around 7 days, the diseases begin abruptly with chills, high fever, serious headache, muscular and articular pains and photophobia. A stiff neck, nausea, vomiting and mental confusion are possible too. At the spot of the bite a lesion of the inoculation *(eschar)* 2-5 mm in diameter, is present in most of the cases. It finally forms a black crust (tache noire). Around the 5-6th day of the illness, a red spotted (macular), or raised (papular), or blistering (vesicular), or sometimes petechial rash may occur on the trunk, then later on the face, palms and soles. Conjunctivitis, pharyngitis, dry cough, abdominal pain, and neurological involvemens may be encountered. Untreated, the fever lasts for one or two weeks. The malignant form of the disease occur by patients having thrombopenia (less than 100 g/l), renal failure (creatinine level >150 mmol/l), hyponatremia, hypocalcemia and hypoxemia.
The diagnosis is usually confirmed by a combination of clinical, epidemiological, and serological testing (IgM and Weil Felix tests)
Treatment: tetracyclines are usually rapidly effective, symptomatically
RFR method: detects and may eliminate the rickettsia.
The summarized resonant frequencies of the Spotted fever group are: 295-296, 310-315, 319, 323, 344, 354, 360, 368-372, 384-388, 390, 403, 407-408, 427, 432-433, 441, 452, 470-479, 482-483, 489, 515, 524, 532-535, 540-543 kHz

6.1.1.2. Scrub Typhus – Caused by Genus Orientia

Orientia tsutsugamushi, the causative agent of scrub typhus, formerly known as Rickettsia tsutsugamushi, has been reclassified into the genus Orientia. The members of this genus are similar to those of Rickettsias being obligate intracellular, Gram-negative bacteria, transmitted to mammalia and found in insect vectors. They are spread by the bites or the feces of infected mites, belonging to the species Leptotrombidium akamushi and Leptotrombidium deliens, typically occuring in Southeast Asia, Japan, India, northern Australia and the adjacent islands.
Symptoms: The disease is characterized by an erythematous vesicular lesion developing about the fifth day at the spot of the bite, being followed in about 8 to 12 days by shaking chills, fever, severe headache, conjunctival infection, anorexia and general apathy. A generalized moderate lymphadenopathy, most prominently in the nodes, draining the area of the primary lesion is also common. The vesicule becomes ulcerated and after the 5th day

of the infection a dull red rash may appear all over the body, starting on the trunk and extending to the extremities. Additional symptoms include splenomegaly, cough and delirium. Pneumonitis or encephalitis may develop during the second week. In severe cases, the patient's pulse rate increases while the blood pressure decreases. The patients may become delirious and lose their consciousness. Other complications, such as muscle twitching, or interstitial myocarditis, more prominent than in other rickettsial cases, may develop. If not treated, symptoms can last for more than 2 weeks; with proper treatment, the patient recovers within 36 hours.

Diagnosis: is confirmed by a combination of clinical, epidemiological and specific antibody testing

Differential diagnosis: typhus, spotted fever, measles and typhoid fever.

Common diagnostic methods in case of SFG diseases: Serologic assays in order to detect anti–R. rickettsii immunoglobulin G (IgG) antibodies are usually performed for the definitive diagnosis. Testing of acute- and convalescent-phase sera is recommended to demonstrate a 4-fold or even higher increase in the titre.

Enzyme immunoassays and immunoglobulin M (IgM) antibody-capture immunoassays are new serologic tests which potentially allow for an early diagnosis.

In research laboratories, the isolation of rickettsiae from tissues or a direct detection of rickettsiae in tissues by means of direct immunofluorescence is used to confirm the diagnosis.

Polymerase chain reactions have been developed but are not widely available.

Differential diagnosis: the rash of meningococcemia resembles the Rocky Mountain spotted fever in certain aspects, being macular, maculopapular, or petechial in its chronic form, and petechial, confluent, or ecchymotic in its fulminant type. The meningococcal skin lesion is tender and develops with extreme rapidity in its fulminant form, whereas the rickettsial rash does only develop on about the fourth day of the disease and gradually becoming petechial. Spotted fever is often confused with measles. The exanthem of rubeola rapidly becomes confluent, while that of rubella usually remains discrete.

Treatment: administering Doxycycline or Chloramphenicol.

RFR method: detects and may eliminate the rickettsial pathogen. Together with antibiotics these frequencies should be used.

The most frequent resonances of the Spotted fever group are: 295-296, 310-315, 319, 323, 344, 354, 360, 368-372, 384-388, 390, 403, 407-408, 427, 432-433, 441, 452, 470-479, 482-483, 489, 515, 524, 532-535, 540-543 kHz

6.1.2. Human Diseases Caused by the Ehrlichiaceae Family

The Ehrlichiaceae is a family of bacteria sorted to the order of Rickettsiales. The human pathogen members of the family include Ehrlichia, Anaplasma and Neorickettsia genera. These bacteria are obligate intracellular coccobacilli, being recognized historically as veterinary pathogens, usually with ticks as vectors. Human ehrlichiosis exists as being 2 clinically similar illnesses:

Human Monocytic Ehrlichiosis (HME), caused by *Ehrlichia chaffeensis* (tick vector: Ambliomma americanum), and

Human Granulocytic Anaplasmosis (HGA), formally known as human granulocytic ehrlichiosis (HGE), is caused by the *Anaplasma phagocytophilum* and *Ehrlichia equi* (tick vectors: Ixodes scapularis, Ixodes pacificus). The *Ehrlichia ewingii* differs from the E.chaffeensis solely in some molecules and can be separated by using molecular techniques. It is known as the pathogen of some cases of HGA in Missouri. The primary target cell of the HGA is the neutrophil granulocyte, while the target cell of the HME is the macrophage. The evidence of human ehrlichiosis has been reported among the residents of USA, Japan, Europe, Scandinavia, and Africa. Ehrlichiosis is a serious disease, its patients often require hospitalization.

Symptoms begin 4-10 days following the tick bite. The onset is abrupt or subacute. The symptoms are nonspecific, resembling those of the Rocky Mountain spotted fever. Fever, headache, myalgia, arthalgia, anorexia and nausea are most typical. A maculo-papular and/or petechial, generalized rash is far less common in case of ehrlichiosis than in case of the Rocky Mountain spotted fever. The laboratory findings include leukopenia, thrombocytopenia, and elevated liver enzymes. Serious manifestations of the disease may include prolonged fever, renal failure, disseminated intravascular coagulopathy, meningoencephalitis, an adult respiratory distress syndrome, seizures and even coma. The infection caused by Ehrlichia chaffeensis may become even more serious than any other ehrlichial infections. An untreated infection can be fatal.
Diagnosis: is confirmed by a combination of clinical, epidemiological and specific antibody testing
Treatment: should begin as soon as possible, should not be delayed waiting for laboratory confirmation. Doxycyclin and Tetracyclin proved to be effective in every case.
RFR method: in combination with antibiotic treatment.
The most frequent resonances are: 307-311, 335-337, 384-389, 414-425 490-491, 534-535 kHz

6.2. The Order of the Rhizobiales

There are two families belonging to the order of the Rhizobiales which are able to cause illnesses among human beings i.e.: the family of the Bartonellacea and the family of the Brucellaceae.

6.2.1. The Most Common Human Illnesses Caused by the Bartonellacea Family

The Bartonellaceae family contains the genus Bartonella, which has eight species of bacteria pathogenic to human beings. They are Gram-negative and facultative intracellular parasites. Apart from the fact that the Bartonella species can infect healthy people, they are *especially important opportunist pathogens*. The bacteria are transmitted by blood-sucking vectors, such as ticks, fleas, sand flies, lice and mosquitoes, the reservoir hosts being mammals. Immediately after infection, the bacteria colonize in the cells of the endothelium. Every five days, a part of the Bartonella in these cells are released into the blood stream, where they infect the erythrocytes. Inside the erythrocytes, the bacteria multiply until they reach a critical population density. At this point, the Bartonella has simply to wait until it is taken within the erythrocytes by a blood-sucking arthropod.

6.2.1.1. Bartonella Bacilliformis

The Bartonella bacilliformis is the causative agent of the **Bartonellosis (also known as Carrion's Disease, Oroya Fever, Verruga Peruana).** The illness is a rare infectious disease found in certain regions of the Andes of South America, and endemic in some areas of Peru. The bacterium is transmitted by sandflies of the genus Lutzomyia. The illness can be characterized by an acute stage with primarily vascular effects and a chronic stage associated with skin symptoms.
Symptoms: The acute stage (also known as *Fiebre de la Oroya*) typically lasts from two-to four weeks, and is a life-threatening illness associated with sudden fever and hemolytic anemia. The peripheral blood smears show anisomacrocytosis with many bacilli adherent to red blood cells. Thrombocytopenia can also be present. A case of neurologic involvement *(neurobartonellosis)* with spinal meningitis or paralysis has a bad prognosis.
The chronic stage (the *Verruga Peruana*) is characterized by a benign skin eruption with reddish-purple nodules (angiomatous tumours). An untreated bartonellosis often leads to death.

RFR method: detects and eliminates the bacteria
Its most frequent resonances are: 308-310, 364-390, 478, 480-492, 548, 558-566 kHz

6.2.1.2. Bartonella Quintana

Bartonella quintana (also called Rochalimaea quintana) is the causative microorganism of the disease called **Trench fever (also known as Shinbone fever, Volhynia fever, His-Werner disease** and **Quintana fever).** The vector is the human body louse, the infected feces of which transmits the microba to people, by rubbing it into the abraded skin or into the conjunctivae. The infection results in a perivascular inflammation.
The disease was first recognized in the trenches of the World-War I, when more than one million people in Russia and on the fronts in Europe were infected. The Trench fever was again a major problem in the military in the World-War II and is endemic in Mexico, Africa, E. Europe, etc.
The **Urban trench fever** occurs nowadays among homeless people, drog users and alcohol addicts and causes bacteremia associated with non-specific symptoms, endocarditis, or no symptoms at all.
Symptoms: The onset of symptoms is sudden, characterized by high fever, splitting headache, muscle pain, bone pain and joint pain of the extremities and the back, as well as the development of a fleeting rash. The macules on the skin show a non-specific perivascular infiltration without involving the vessel walls (in contrast to typhus cases). In most of these cases the symptoms assume a relapse of the illness. The recovery takes a month or more. Relapses are common, characteristically occuring in intervals of 4-5 days. The disease is marked by persistent bacteremia, being present at the initial attack, during the relapses and throughout the asymptomatic periods for even ten years after the onset of the infection.
B. quintana has been found responsible for the disease called **Bacillary angiomatosis** in case of people infected with HIV, for the infection of the heart and the great vessels (endocarditis) with bacteremia. The full spectrum of diseases caused by Bartonella quintana is still uncomplete.
Prevention: by the elimination of lice and with the improvement of the living conditions, the provision for frequent bathing and washing of the articles of clothing.
Diagnosis: can happen by using complement tests or special bacterial culture methods. Bartonella infection is difficult to diagnose in laboratories. One has to inform the laboratory about the suspicion of the B quintana infection, in order that the personnel can perform the special bacterial culture methods required for isolation.
Differential diagnosis: typhoid fever, typhus, dengue, relapsing fever, influenza, etc.
Treatment: with antibiotics such as Doxycyclin etc.
RFR method: detects and may eliminate the bacteria, by using it together with antibiotic treatment.
Its most frequent resonances are: 554-564 kHz
Having eliminated these frequencies, rechecking is necessary over a long period of time.

6.2.1.3. Bartonella Henselae

Bartonella henselae can cause **bacteremia, endocarditis, bacillary angiomatosis** and **peliosis hepatis**. Peliosis is an uncommon vascular alteration, characterized by multiple blood-filled cavities (2-30 mm diameter in size) throughout the liver and/or the spleen. Patients with peliosis hepatitis have gastrointestinal symptoms, fever, chills, lymphadenosis and an enlarged liver and enlarged spleen. This systemic disease is observable in patients infected with HIV or in case of those, immuno-compromised in an other way.
Bartonella henselae is also the causative agent of the **Cat-scratch disease**, which occurs after the bite or scratch of a cat. (The cat usually does not show any signs of illness). The

disease is characterized by a red, crusted cutaneous lesion at the spot of the inoculation, and is followed by fever and an indolent, sometimes suppurative regional lymphadenitis, secondarily accompanied by headache. The histopathological finding of the swollen lymphnode shows cell hyperplasia at the beginning and later on granuloma formations and microabcesses.
Diagnosis: can happen by carrying out specific antibody-tests.
Treatment: by giving effective antibiotics.
RFR method: detects and may eliminate the bacteria!
Its most frequent resonances are: 324, 330-334, 356, 366, 372, 388, 402, 429-435, 438, 440, 495 kHz

6.2.1.4. Other Members of the Bartonella Genus

These members of the Bartonella genus (f.i. B. clarridgeiae, B. elizabethae, B. grahamii, B. washoensis), cause only incidentally illnesses among human beings, mostly in cases of immune damages. The transmission of the bacteria happens by an incidental biting or scratching etc. of an infected domestic cat, or a rat, mouse, squirrel, etc. The symptoms are typically those of the cat-scratch disease, the bacillary endocarditis, the bacillary angiomatosis, the bacillary neuroretinitis or the symptoms of a bacteremia.
Its most frequen resonances are: 380-389, 429-438, 510 kHz

6.2.2. Human Illnesses Caused by the Family of the Brucellacea

Only the brucella genus of this family can cause illnesses among human beings. The members of this genus are Gram-negative, small, non-motil coccobacilli. The Brucella causes the brucellosis. This illness is **a true zoonosis**, the bacteria infect animals, the transmission to man can occur either due to the ingestion of infected food, by direct contact with an infected animal, or by inhaling infected aerosols. It is primarily a world-wide disease of domestic animals (goats, pigs, cattle, dogs, etc) and human beings. **Human brucellosis** primarily occurs owing to occupational exposure to cattle, sheep, pigs, goats, etc. and also by consumption of unpasteurized milk products (milk or cheese). The causative species are mostly the **Brucella melitensis, the Brucella abortus and the Brucella suis**. Brucella melitensis causes illnesses of the reproductive organs, f.i. orchitis, epididymitis, mastitis and abortions. This zoonosis can cause Malta fever or localized brucellosis among human beings too.
Symptoms: The disease begins suddenly with chills and fever, headache, abdominal pain, joint pain, and, occasionally, diarrhea. *In the first phase* of the disease, *septicaemia* occurs and leads to the classic triad of *undulant fever, sweating* (often with a characteristic smell, likened to wet hay), *migratory arthalgia and myalgia*. Following the invasion of the body by brucellae, the microorganisms tend to become localized in the tissues of the reticuloendothelial system, such as the bone marrow, lymph nodes, liver, spleen and the kidneys as well. A characteristic, but nonspecific reaction of these tissues to the brucellae is the appearance of epitheloid cells, giant cells of Langhans' type, lymphocytes, and plasma cells. Necrosis and caseation rarely occur in these granulomatous areas. The *granulomas* are similar to those observable in case of sarcoidosis and tuberculosis. Caseation is usually found in illnesses caused by Brucella suis. Other, less frequent localization of Brucella organisms are the bones, the endocardium, and the testes. After the initial phase, the symptoms usually include severe constipation, appetite loss, weight loss, sweating, headache, backache, muscular pain, weakness, irritability, insomnia, depression, and emotional instability, without any abnormal physical finding. Leukopenia, anemia might often develop. If untreated, the disease can lead to focalization or become chronic. The *focalizations* of brucellosis are usually in the bones and joints, and *spondylodisciitis* of the lumbar spine, accompanied by *sacroileitis* is characteristic of this disease. The duration

of the disease can vary from a few weeks to many months or even years. Some people develop a *chronic brucellosis* and experience repeated waves of *fever, sweating, fatigue, mental depression* and evenremission over months or years. The sequele of the disease may include *granulomatous hepatitis, arthritis, meningitis, uveitis, optic neuritis and endocarditis, anemia, leukopenia and thrombocytopenia.*
Diagnosis: by using antibody-tests, liver-biopsy, radiological finding, blood cultures
Differential diagnosis: other acute febrile illnesses, typhoid fever, sarcoidosis, several types of nervous disorders etc.
Treatment: with antibiotics (Doxycyclin, Rifampicin), aminoglycosides (Streptomycin, and Gentamycin) administered for several weeks.
Prevention: by vaccination of the animals.
RFR method detects and eliminates the bacteria.
The most frequent resonances of Brucella abortus are: 455 kHz
The most frequent resonances of Brucella melitensis are: 329, 355, 382 kHz

The Class of the Beta Proteobacteria

Some members of the Neisseriaceae, some species of the Burkholderia genus, and one species of the Spirillum genus of the order of Nitrosomonadales belonging to this class, are human pathogens. The *Spirillum minus* species can cause **rat-bite fever** *mostly in countries of Asia and Africa.* (Rat-bite fever caused by *Streptobacillus moniliformis* see the Phylum of Fusobacteria*)*. The illness, named also sodoku, is characterized by a prolonged incubation period followed by relapsing fever, local lymphadenitis and a suppurative inflammation of the skin area bitten by a rodent.

6.3. The Order of the Neisseriales

The genus Neisseria of this order includes 2 important human pathogenic species. One of them is responsible for gonorrhoea, the other for many cases of meningitis. Being members of the Neisseriaceae family, they are strictly aerobic, Gram-negative, occuring in pairs (diplococci).

6.3.1. Neisseria Gonorrhoeae (Gonococcus)

The diplococcus Neisseria gonorrhoeae is responsible for Gonorrhoea, which is a. sexually transmitted disease (STD). These cocci are facultative intracellular, appearing typically in pairs, their natural host being solely human beings. The disease prevails usually among heterosexual young people, repeatedly changing their sexual partners. Neisseria gonorrhoea infects mainly the urethra and the vagina of women, the urethra, and the prostate of men. It can infect the anus or the conjunctiva too. An oral sexual intercourse with an infected partner may result in gonorrhoeal pharyngitis. If not treated, the infection might spread. The arthritis caused by gonococcus is probably the most common form of acute arthritis in young adults. Local ascensive complications include endometritis, salpingitis, peritonitis in case of females, and periurethral abcesses and epididymitis in case of males.
Symptoms include a purulent discharge from the genitals, a foul smelliness, a burning sensation when urinating, and, if infected fluids get into the eyes, purulent conjunctivitis may develop. If not treated, complications will develop either by ascending and infecting the inner genital organs, or by spreading via bloodstream into the joints, usually causing swollen, tender and extremely painful monoarthritis. In more complicated cases fever, a general feeling of illness, or pain spreading from joint to joint and septic gonococcal pus-filled spotted dermatitis (called gonococcal arthritis-dermatitis syndrome) can come into being. Systemic manifestations of gonococcemia resulting in endocarditis, pericarditis, perihepatitis, are rare complications of this disease. Meningitis caused by gonococcus is

very rare. Conjunctivitis can occur in neonates infected at their birth. The sexual partners should be tested for chlamydia trachomatis infection, since co-infection can be frequently found. In some cases, mostly regarding women, the infection can be asymptomatic.
Diagnosis: happens by identification of the Gonococcus, using bacterial culture procedures.
Treatment: by administrating adequate antibiotics
RFR method: beside giving adequate antibiotics, RFR method can also be used for the detection and the elimination of the bacteria.
The general range of Neisseria gonorrhoeae is: 332-337 kHz
Its other frequency resonances are: 298, 307, 330-338, 364, 384, 455, 474-475, 477, 480, 532 kHz

6.3.2. Neisseria Meningitidis (Meningococcus)

The Neisseria meningitidis is also a Gram-negative diplococcus, causing meningitis. It infects only human beings. This is the only bacterial meningitis form, causing epidemics. The most important strains of meningococcus are the A, B, C, Y and W135 strains:
Infection, caused by *Strain A* - is mostly present in Sub-Sahara Africa.
Strain B –did also cause cases of lethal meningitis in the USA, before a specific vaccination was given against it. The changing character of group B is prevented by using a general B vaccine in the UK. A vaccine against a specific strain of group B meningococcus was developed to control an epidemic in New Zealand.
Strain C - caused a lot of cases in the UK before a successful vaccination program for infants with a conjugated form of vaccine had been introduced.
Strain W135 – could be a problem for pilgrims going every year toMecca, so that they are not allowed to enter the state without being specific vaccinated beforehand.
Serogroup X –caused a large outbreak of meningitis in Niger in 2006. There is still no available vaccine for this strain.
Serogroup Y caused diseases in Northern America.
Other strains include 29-E, H, I, K, L, X, and Z.
Meningococci cause both epidemic or sporadic diseases.
The human nasopharynx is the only known reservoir of the meningococcal infection. Meningococci spread from person to person by airborne droplets of infected nasopharyngeal secretions but unlikely by contact with contaminated fomites. The pathogens attach themselves to mucosal surfaces, producing there but few symptoms. A concommitant viral respiratory infection, particularly an infection caused by influenza viruses, appears to enhance the likelyhood of the nasopharyngeal carriing after exposure to meningococci and furthers the spread of the meningococcal infection.
Meningococcal infection of the nasopharynx is usually subclinical. Asymptomatic nasopharyngeal carriing of meningococci is transient and resolves within several weeks. In some individuals, the bacteria invade the circulation and cause clinical diseases. The clinical disease caused by this microorganism can be classified into 3 arbitraryforms:

(1) it can be an uncomplicated bacteremic process,
(2) a metastatic infection that commonly involves the meninges and
(3) an overwhelming systemic infection with circulatory collapse and with the evidence of disseminated intravascular coagulation (DIC).

The fundamental pathologic change in meningococcemia is the widespread vascular injury, characterized by endothelial necrosis, intraluminal thrombosis and perivascular hemorrhage. The meningococcal endotoxin may be responsible for the hypotension and the vascular collapse, observed in case of a fulminant meningococcemia, and may also play a role in the pathogenesis of the purpura and the visceral hemorrhages, associated with meningococcal bacteremia. Skin lesions usually contain numerous meningococci, undergoing phagocytosis by neutrophils. Occlusive thrombi composed of platelets, red

blood cells, and fibrin are most prominent in vessels deep in the dermis. Other organs can have the same vascular injury, although bacteria are difficult to find in tissues other than the skin.
Among patients with a fulminant meningococcemia, thrombosis and haemorrhage can develop in the skin, the mucous membranes, the serosal membranes, adrenal sinusoids, and renal glomeruli. The adrenal haemorrhage is seldom extensive. The thrombosis of the glomerular capillaries may cause renal cortical necrosis, which is the most characteristic feature of the generalized Shwartzman reaction. Thrombi containing numerous leukocytes and an extensive intra-alveolar haemorrhage are occasionally observed in the lungs. Myocarditis can be observed in case of fatal meningococcal infections in adults.
Symptoms: The onset of the clinical illness may be abrupt, the patients usually have nonspecific prodomal symptoms such as *cough, headache, and a sore throat,* followed by a sudden development of *raging fever, chills, arthralgia, and muscle pains,* which may be particularly severe in the lower extremities and in the back. In addition to high fever, *vomiting, headache, tachycardia, and tachypnea, mild hypotension* may also be present. The dissemination of the meningococci occurs from the nasopharynx via the bloodstream, and is followed by the clinical manifestation of the meningococcal disease. A *purulent meningitis, causing neck stiffness* is the most common form of the metastatic infection, and is either associated with signs and symptoms of the meningococcemia or constitutes the predominant clinical expression of the illness, which, in some cases rapidly leads to *coma and death.* People with an impaired immunity are particularly in danger of getting a meningococcal disease. The suspicion of having meningitis is a medical emergency and an immediate medical assessment (like iv. antibiotics) and hospitalization is needed. *Septicaemia* caused by Neisseria meningitidis usually ends with the infant's death. The meningococcal septicaemia causes typically a *purpuric „non-blanching" rash* and does not show the classical symptoms of meningitis, the rash is thus solely a warning signal. The mortality rate of septicaemia is an approximatly by 50%. Death ensues within a few hours. It is advised that anyone who has a non-blanching rash should go to a hospital emergency room as soon as possible. The fulminant form of meningococcemy, called *Waterhouse-Friderichsen syndrome* is characterized by a massive, often bilateral hemorrhage into the adrenal glands, and is associated with a vasomotor collapse and shock. Chronic meningococcemy is a rare form of themeningococcal infection, which lasts for weeks or months, and is characterized by fever, rash, and arthritis or arthralgia. Meningococcus can only seldom be the cause of a *purulent conjunctivitis*, sinusitis, or pneumonia. *Meningococcal endocarditis* is a rather rare illness. A high percentage of patients dying of meningococcal infections suffer from myocarditis.
Prevention: by using strain-specific vaccinations, if available
Diagnosis: by sending immediately a blood sample and a cerebrospinal fluid speciment to the laboratory for the identification of the diplococcus.
Therapy: iv. Benzylpenicillin, Ceftriaxon or any other effective antibiotic therapy must be given as soon as possible.
RFR method: the first step is the antibiotic therapy, after which RFR method can be used in order to detect and eliminate the bacteria.
Its most frequent resonances are: 307, 320, 330-347, 359-360, 364-368, 372, 402, 410, 425, 450, 476-477, 518-519, 548, 550 kHz
The list is not complete yet.

6.4. The Order of the Burkholderiales

The **Burkholderiales** is an order of the Beta Proteobacteria. Two families of this order, i.e. the Burkholderiaceae and the Alkaligenaceae include several pathogenic species sortable to the genus of Burkholderia and Bordetella.

6.4.1. The Burkholderia Genus

The Burkholderia genus (previously sorted to the Pseudomonas genus) refers to a group of virtually ubiquitous gram-negative, motile, obligately aerobic bacteria. Its pathogenic members are the *Burkholderia mallei*, which is the causative agent of the glanders, infecting mainly horses; the *Burkholderia pseudomallei*, being the pathogen agent of melioidosis and the *Burkholderia cepacia*, which can cause pulmonary infections among people with cystic fibrosis.

6.4.1.1. Burkholderia Mallei (Glanders or Malleus)

Burkholderia mallei (formerly: Acinetobacter mallei, Pseudomonas mallei, Loefferella mallei, etc.) is a gram-negative, non-motile, bipolar, aerobic, pathogenic bacillus, infecting animals and human beings. The glanders is a serious infection of equine animals caused by B. mallei. The pathogen is transmitted occasionally to other domestic animals and also to human beings. The acute lesion is characterized by nodules, consisting of polymorphonuclear leucocytes, surrounded by a zone of congestion. Giant cells, or epitheloid cells are present in the area of the lesion, forming a central necrosis.

The **Symptoms,** which may frequently be overlapping, can be characteristic of an acute, localized suppurative infection, an acute pulmonary infection, an acute septicemic infection, and of a chronic suppurative infection.

Diagnosis: serologically, and/or using bacterial culture, complement examinations.

Differential diagnosis: any other febrile illness.

Treatment: with effective antibiotics, such as Doxycyclin, Tobramycin etc.

Prevention: active immunization

RFR method: used in conjunction with antibiotic treatment, it detects and eliminates the bacteria.

The most frequent resonances of this genus are: 291-298, 325, 333, 351, 356-396, 408, 438-450, 580 kHz

Those of Burkholderia mallei are: 325, 351, 377, 380, 396, 438, 448, 504, 513 kHz

This list is not complete yet.

6.4.1.2. Burkholderia Pseudomallei (Melioidosis)

Burkholderia pseudomallei is a Gram-negative, bipolar, aerobic, rod-shaped human and animal pathogen bacillus, causing melioidosis. The bacteria are motile, using flagellae and producing both exo- and endotoxins. It affects human beings as well as animals such as goats, sheep, horses and cattle. The infection can happen either due to inoculation through a chapped skin, or through inhalation of aerosolized B. pseudomallei.

The melioidosis is endemic in certain parts of South-East Asia (including Thailand, Singapore, Malaysia, Burma and Vietnam) as well as in northern Australia. Many a case has been experienced and described also in southern China and Hong Kong, Brunei, Taiwan, India and Laos. In Central and South America, the Middle East, Bangladesh, the Pacific and several African countries cases can only be found sporadically. The disease exists both in acute and in chronic forms. In case of inoculation, the mean incubation period used to be 9 days. Patients may remain asymptomatic for decades. There is a wide range as regards the severity of the disease, in chronic cases the symptoms may be present for months, while in fulminant cases the serious symptoms may be present very soon after being infected. Patients suffering **risk factors** like diabetes, thalassemia, alcohol abusus or a renal disease, are all prone to get melioidosis and people frequently give their history of occupational or recreational exposure to mud or pooled surface water. Even otherwise healthy persons, including children, may get melioidosis.

Symptoms of the acute form: An infection via inoculation owing to a chapped skin usually results in a nodule and a regional *acute lymphangitis and lymphadenitis*. This form of infection may rapidly progress to an acute septicemic form. In case of acute infections, the majority of the lesions develops in the lung, the rest of them produce occasional abscesses in other organs. The acute *pulmonary* infection can vary in severity from a mild bronchitis to an overwhelming necrotizing pneumonia. Symptoms include *headache, fever, anorexia, and a generalized myalgia*. Cough, with or without sputum is common. Acute *septicemic* infection may be fatal. The latter is characterized by high fever, extreme *tachypnea*, a flushed skin and cyanosis. Muscle tenderness may be striking. The course of this septicemic form of the disease is usually rapidly progressive and fatal and is in some instances too fulminant to be alterable by therapy.
In case of subacute infections, the *lung abscesses* tend to be more extensive, and the *abscesses might be found throughout the whole body*: in the skin, in the subcutaneous tissue, in the meninges, the brain, the eyes, the heart, the liver, the kidney, the spleen, the bones, the prostate and in the lymph nodes. The abscesses are characterized by an outer edgea of hemorrhage, a medial zone, heavily infiltrated with polimorphonuclear leucocytes, and an inner core of necrotic debris containing large histiocytes with two or three nuclei, termed giant cells. The most common form of melioidosis is the pulmonary infection.
Chronic melioidosis is usually definable by symptoms longer than 2 months and that it effects almost. 10% of the patients. Its clinical picture is protean, including such injuries like chronic skin infections, skin ulcers and lung nodules or chronic pneumonia, closely mimicking that of tuberculosis.
Diagnosis: A definite diagnosis can be made by culturing the organism from any clinical sample. Blood culture, sputum culture, urine culture, throat swab and culture of any aspirated pus should be performed concerning every patient with suspected melioidosis. Imaging of the abdomen using CT scans or ultrasound is recommended as routine work, the abscesses are sometimes not clinically apparent but may nevertheless, be present in connection with this disease.
Prevention: A vaccine is not yet available.
The **Treatment** of melioidosis can be divided into two stages, an intravenous high-intensity stage (iv. Ceftazidime, Meropenem, Imipenem, Amoxicillin-clavulanate) administered for 10 to14 days and an oral maintenance stage, to prevent its recurrence (f.i. Co-trimoxazole, Doxycycline) given for 12-20 weeks. Surgical drainage is usually indicated in case of prostatic abscesses and septic arthritis, and may be advisable against parotid abscesses but usually not against hepatosplenic abscesses.
RFR method: used in conjunction with antibiotic treatment, it detects and eliminates the bacteria.
Its most frequent resonances are: 325, 351, 377, 380, 396, 438, 448, 504, 513 kHz

6.4.1.3. Burkholderia Cepacia Complex

Burkholderia Cepacia Complex (BCC) is a group of Gram-negative bacteria, composed of at least nine different species. It is an important human pathogen which very often causes pneumonia among immunocompromised patients, suffering from any lung disease (e.g. cystic fibrosis, chronic granulomatous lung disease). BCC organisms are typically found in water and soil and can survive for prolonged periods in any moist environment. It spreads from person-to-person, as documented. BCC infection can lead to a rapid decline of the lung functions and can even result in death.
Diagnosis of BCC involves the isolation of bacteria from sputum cultures.
Therapy: with antibiotics, such as doxycyclin, ceftazidime, co-trimoxazole etc.
RFR method: detects and may eliminate the bacteria.

6.4.2. The Bordetella Genus

The Bordetella genus belongs to the Alkaligenacea family of the Order of the Burkholderiales.

There are three humanpathogenic species among them: the Bordetella pertussis, the Bordetella parapertussis and the Bordetella bronchiseptica. They are small, obligate aerobes and highly fastidious Gram-negative coccobacilli. The latter is motile as well.

Bordetella pertussis and, occasionally, *B. parapertussis* cause the illness called Pertussis or whooping cough among human beings. Bordetella pertussis strains can colonize sheep too. *Bordetella bronchiseptica* infect chiefly immunocompromised patients.

6.4.2.1. Bordetella Pertussis (Pertussis)

The illness is characterized by an inflammation of the respiratory tract causing a paroxysmal cough and a typical inspiratory stridor or whoop among unimmunized children. The transmission of the pathogens occurs by direct contact, through respiratory aerosol droplets or fomites. The bacteria adhere first to ciliated epithelial cells in the nasopharynx. The secretion of toxins (haemagglutinin, pertussis toxin, tracheal cytotoxin etc.) causes ciliostasis and facilitates the entry of the bacteria into the tracheal/bronchial cells. The hyperplasia of the peribronchial and tracheobronchial lymphoid tissue tends to develop. The upper respiratory tract will get soon involved in a necrotizing inflammatory reaction.

Symptoms: The first clinical manifestations are a slight nasal discharge, conjunctivitis, and a mild cough. The later phase of pertussis is characterized by paroxysms of coughing, ending in a loud, crowing inspiratory noise, the expulsion of varying quantities of thick, mucoid sputum from the respiratory tract and vomiting. But no fever unless coinfections are present. Spasm, ulcer, or edema of the glottis can develop in some cases. Severe vomiting and inability to retain food, serious inanition, wasting, and tetany may appear.

Hemoptysis, epitaxis, purpura, and subconjunctival or intestinal haemorrhages are usually of little clinical significance.

Associated infections (due to Streptococcus pyogenes, Diplococcus pneumoniae, Staphylococcus aureus, Hemophilus influenzae, Bordatella parapertussis, Bordatella bronchiseptica, Adenovirus, cold virus etc.) are apt to cause complications. A pneumonitis appearing during the course of antibiotic treatment develops most often due to Escherichia coli, Proteus strains, Klebsiella groups or Pseudomonas aeruginosa. Another important complication is the atelectasia, developing with small areas of collapse. Spontaneous pneumothorax is rare.

Other complications, as hyperreflexia, nuchal rigidity, cranial nerve palsies, flaccid hemiplegia, areflexia, spasticity of the extremites, nystagmus, blindness and strabismus may occur.

Diagnosis: the typical coughing and whooping is of diagnostic value; through identification of the bacteria.

Prevention: active immunization is available and used obligatory in most countries.

Treatment: with Doxycyclin, Erythromycin and Chloramphenicol.

RFR method: used together with antibiotic treatment, it detects and eliminates the bacteria.

Its general range is: 329-333 kHz

Its other frequency resonances are: 315-316, 334-335, 340, 356, 372, 374, 384, 391, 397, 402-410, 425, 450-463, 490, 494, 538, 564, 580 kHz

The Class of the Gamma Proteobacteria

The Class of the Gamma proteobacteria comprises several human pathogenic bacteria.

6.5. The Order of the Enterobacteriales

The **Enterobacteriaceae** is a large family of bacteria which can cause infections of the gastrointestinal tract or of other organs of the body. The family includes pathogens infecting human beings such as Enterobacter, Escherichia, Klebsiella, Salmonella, Shigella, Serratia, Proteus, Morganella, Providentia and Yersinia. These bacteria can cause both acute and chronic serious diseases, resulting in the fatigue syndrome or the immunodepression and in the immunosuppression of organs.

Enterobacteriales are the microorganisms most commonly responsible for Gram-negative bacteraemia. In case the bacteria invade the bloodstream, a component of Gram-negative bacterial cell walls, the **endotoxin**, apparently triggers a cascade of inflammatory responses of the host, leading to major detrimental effects. Gram-negative bacteria can cause *endotoxin-induced sepsis*, resulting in a *Systemic Inflammatory Response Syndrome (SIRS)*. SIRS has a mortality rate of 20-50%. Although other organisms can trigger similar responses, it is useful to consider that the gram-negative bacteremia is a distinct entity because of its characteristic epidemiology, pathogenesis, pathophysiology, and treatment.

Enterobacteriaceae may posses colonization factors, which are supposed to play a role in the development of cancers. The antigens of the Enterobacteria and/or of other parasites (such as amoebae, fungi, protozoa) always play a role as cofactors in the etiology of the Crohn's disease. The members of the Enterobacteriaceae are Gram-negative, non-spore forming, facultative anaerobes. Most of them have many flagella, to move about, but some of their genus are non-motile. Many members of this family are normal parts of the gut flora in the intestines of human beings and animals, while others are found in water or soil, or are parasites on a variety of different animals and plants. The human pathogenic genera of this large family are, as follows:

6.5.1. The Citrobacter Genus

There are two species in this genus which can cause illness among human beings. Although the members of this genus can be found almost in every soil, water, waste-water etc., some members can also be found in the human intestine. They can infect the *urinary tract* and can cause *meningitis, when infecting infants*. Bactereemia due to *Citrobacter diversus* and *Citrobacter freundii* usually develop among hospitalized elderly patients, causing high mortality. In most cases of bacteremia, caused by the species *C. diversus*, the initial locus of the infection is very often the urinary tract, while in cases of bacteremia caused by *C. freundii*, the first focus is usually a gallbladder disease. Citrobacter spp. cause neonatal meningitis, and are unique because of their frequent association with brain abscess formations.

6.5.2. The Enterobacter Genus

Some strains of these bacteria (such as *Enterobacter aerogenes* and *Enterobacter cloacae)* can be pathogenic and apt to cause *opportunistic infections* in cases of immunocompromised, hospitalized hosts, usually resulting from venous catheter insertions or surgical procedures. The *urinary and the respiratory* tract are the most common loci of the infection. Species of the *Enterobacter aerogenes* are generally found in the human intestinal tract and are not causing any disease among healthy persons.

The most frequent resonances of the Enterobacter aerogenes are: 370-376 kHz
The most frequent resonances of the Serratia marcesceus are: 348-354 kHz
The most frequent resonances of the Morganella are: 370-374, 398-406, 505 kHz

6.5.3. The Escherichia Genus

Most of the members of this Gram-negative, facultatively anaerobic genus are normal inhabitants of the gastrointestinal tracts of warm-blooded animals. The Escherichia species provide their host with a portion of the microbially-derived Vitamin K2 and are the most numerous aerobic *commensal inhabitants* of the large intestine of the human body, and prevent the establishment of pathogenic bacteria in the intestine.
While many Escherichia species are harmless commensals, certain other strains of Escherichia coli are nevertheless pathogenic.
Symptoms of the enteric E. coli bacteria are like those of a *gastrointestinal infection,* with inflammatory (bloody and/or mucoid stools) or not inflammatory (watery, dysentery-like stools) diarrhea, abdominal cramps, and little or no fever. The majority of the infections abate completely. The virulent strains can cause serious illnesses or even death in elderly, very young or immunocompromised patients. On the basis of their virulence properties, these bacteria are classified into the following groups: *enterotoxigenic, enteropathogenic, enterohaemorrhagic and enteroaggregative E. coli bacteria.* The shiga-like toxins, produced by the enterohemorrhagic E. coli serotype O157: H7, are the most common cause of the *hemolytic uremic syndrome* (microangiopathic hemolytic anemia, thrombocytopenia, and acute renal failure) among children under 5 years of age. The illness is a foodborne disease, associated with eating or drinking undercooked, contaminated food, milk, or water. The bacteria can also be spread by person-to-person contact.
Some serotypes of E. coli are known as the most common cause of *urinary tract infections*, they *can infect the gallbladder*, *the lungs*, and *the peritoneum*. E. Coli bacteremia, developing to sepsis and meningitis harm mostly premature newborns.
Diagnosis: using PCR techniques
Therapy: by giving antibiotics, - but only in severe cases
RFR method: dertects and eliminates the bacterium. After ending the therapy the administration of probioticum-complex is needed.
Its general range is: 356, 390-394 kHz
Its other resonant frequencies are: 317-320, 323-328, 337, 343-344, 356-358, 390-398, 408-412, 422, 435, 443, 454-458, 478, 560-572, 576-588 kHz

6.5.4. The Klebsiella Genus

The members of this genus are non-motile, Gram-negative bacteria. They are ubiquitous in nature, and also frequent pathogens, causing *pneumonia, urinary tract infections, septicemy, ankylosing spondylitis and soft tissue infections.* In case of human beings, they may colonize the skin, the pharynx, or the gastrointestinal tract. They may also colonize sterile wounds and the urine. Klebsiellae may be regarded as normal flora in many parts of the colon, the intestinal tract and the biliary tract as well. They are opportunistic pathogens found in the environment and on the mucosal surfaces. The principal pathogenic reservoirs of the infection are the gastrointestinal tract of the patients and the hands of the hospital staff. The organisms can spread rapidly, and often lead to nosocomial outbreaks.
The most common human pathogen Klebsiella species is the *Klebsiella pneumoniae.* The bacteria overcome the host's innate immunity by several means. They possess a polysaccharide capsule, which is the main determinant of their pathogenic effect.
Symptoms: The infection occurs *in the lungs*, where they cause destructive changes of their illness such as necrosis, inflammation, and hemorrhage, producing sometimes a *thick, bloody, mucoid sputum.* The patients usually suffer an acute onset of high fever and *chills; have flulike symptoms; and a productive cough.* There exists an increased tendency to get abscesses, cavities, empyema, and pleural adhesions. The illness typically affects middle-aged and older men with an impaired respiratory defense potencial.
K. pneumoniae and *K. oxytoca* are often responsible for *nosocomial infections*. Common loci are the urinary tract, the lower respiratory tract, the biliary tract, and surgical wounds.

Clinical syndromes are *pneumonia, bacteremia, thrombophlebitis, urinary tract infections (UTIs), cholecystitis, diarrhea, upper respiratory tract infections, wound infection, osteomyelitis, and meningitis.* The presence of invasive devices, the contamination of respiratory support equipment, the use of urinary catheters, and the use of antibiotics are factors that increase the likelihood of nosocomial infections with the *Klebsiella* species. *Sepsis and septic shock* may follow the entry of the organisms from a focal source into the blood.
The *Rhinoscleroma* and the *ozena* are rare infections caused by *Klebsiella species.* The Rhinoscleroma is a chronic granulomatous inflammatory process involving the nasopharynx, whereas the ozena is a chronic atrophic rhinitis, characterized by nasal crusting, discharge, and a very bad smell. *K. oxytoca* is usually implicated in the *neonatal bacteremia.*
RFR method: detects and eliminates the bacteria
Its general ranges are: 398-402, 414-419 kHz
Its other resonance frequencies are: 319, 325, 332, 381, 391-392, 397-405, 410-430, 508 kHz

6.5.5. The Proteus Genus

The Proteus bacteria possess an extracytoplasmic outer membrane, a feature shared with other Gram-negative bacteria. The outer membrane contains a lipid bilayer, lipoproteins, polysaccharides, and lipopolysaccharides. Various components of the membrane interplay with the host to determine virulence. Also this genus can cause deep infections, particularly in the urinary tract. Three species are opportunistic human pathogens such as the *Proteus vulgaris*, the *Proteus mirabilis* and the *Proteus penneri* (formerly called Proteus vulgaris biogroup-1 or P.v. indole negative). Some strains of P. vulgaris share a common antigen with certain rickettsia species, accounting for the appearance of antibodies against Proteus in typhus, scrub typhus, and Rocky Mountain spotted fever.
The species of the Proteus genus are normally found in soil, sewage and are part of the normal fecal flora. They are sometimes a cause of *epidemic diarrhea* in infants. Proteus bacteria are often localized in damaged tissues, where they produce a typical, exudative, purulent inflammatory reaction. They are rarely primer invaders, but can produce local diseases in places previously infected by other organisms, such as Shigella, E. coli, Staphylococci, and Pseudomonas. Their locations include the skin, ears, mastoid sinuses, eyes, peritoneal cavity, bones, connective tissues, heart, urinary tract, meninges, lung and bloodstream. Proteus organisms are frequently found with other bacteria in isolated burns, varicous ulcers and decubitus.
These facultatively anaerobic, Gram-negative bacteria have the ability to produce urease. This enzym hydrolyzes the urea making thus the urine alkaline. The increased alkalinity can lead to the *formation of crystals* (containing ammonia; magnesium; phosphoric acid; hexahydrate, calciumcarbonate and/or apatite). The Proteus species is rarely pathogen in case of anatomically normal urinary tracts, except occasionally among patients with diabetes mellitus or immunosuppressive states. The ability of the bacteria to urease production and to bacterial motility with fimbriae may together favor the development of an upper urinary tract infection. The chance of a developing infection is hightened by a longer duration of catheterization, faulty catheter care, lack of systemic antibiotic therapy and if the patient is a female. The infection occurs either by migration of the bacteria up the catheter along the mucosal sheath, or by migration from the infected urine up in the catheter lumen.
The bloodstream invasion is the most serious manifestation of infections caused by Proteus bacteria. The symptoms include high fever, chills, shock, metastatic abscesses, leucocytosis and seldom thrombocytopenia. As Proteus (and Pseudomonas) organisms are Gram-negative bacteria, they can cause *Gram-negative endotoxin-induced sepsis*, resulting

in Systemic Inflammatory Response Syndrome (SIRS). The presence of the sepsis syndrome associated with an urinary tract infection (UTI) should raise the possibility of urinary tract obstruction.
Proteus species may be involved also in *synergistic nonclostridial anaerobic myonecrosis*, which affects the subcutaneous tissue, fascia, and muscles. This destruction is caused by Proteus species together with other, aerobic, Gram-negative bacteria (e.g. E. coli, Klebsiella and Enterobacter species) and anaerobes.

6.5.5.1. Proteus Mirabilis

The **Symptoms** of the infection of this species manifest themselves in the urinary tract. Once *kidney stones are developed* in this urinary tract infection, they may grow to be large enough to cause *obstruction* or to lead to *renal failures*. In other cases, this Proteus species can also cause *wound infections, septicemia and pneumonia,* mostly in case of hospitalized patients.
The general frequency resonances of P. mirabilis are: 320-326, 345-352, 411, 516 kHz

6.5.5.2. Proteus Vulgaris

Besides its ability to *infect the urinary tract and wounds, Proteus vulgaris* is a common cause of *maxillary sinus and respiratory infections*, especially in South East Asia. In case of a typical human sinusitis and/ or an infection of the respiratory tract caused by P. vulgaris, its eradication can take weeks or even months, even if using those few antibiotics that the P. vulgaris pathogen is sensitive to. In case of *otitis media and mastoiditis,* coinfected with Proteus species, the infection often result in extensive destruction of the middle ear and the mastoid sinuses. The paralysis of the facial nerve can be an occasional complication. The danger of these infections lies in the intracranial extension, leading to thrombosis of the lateral sinus, meningitis, brain abscess, and bacteraemia. If left untreated or undertreated with antibiotics that have but an intermediate effect on P. vulgaris, the infections can lead to death. (In case of a sinusitis or a respiratory tract infection caused by Proteus vulgaris, antibiotics should be given in much higher doses and for much longer than usually.)
Following a trauma to the eye, Proteus may *infect the cornea causing ulcers*, which process may terminate in panophthalmitis and destruction of the eye.
In case of *coinfections* of Proteus species with other bacteria and/or viruses, such as Streptococcus or the Coxsackie virus, a cardiac inflammatory disease may occur.
Treatment: by giving higher dosis of effective antibiotica and for a longer time, than usually.
RFR method: detects and may eliminate the bacteria.
The general frequency resonances of P. vulgaris are: 327-329, 333-339, 408-416, 426, 522-529, 535 kHz

6.5.6. The Providentia Genus

The members of this genus are opportunistic pathogens in case of infections among people, which can cause urinary tract infections, mostly among patients using urinary catheters for a long time, or have severe burns.
Treatment: with antibiotics
RFR method detects and eliminates the bacteria.

6.5.7. The Salmonella Genus

The members of this genus are Gram-negative, motile enterobacteria that can cause typhoid fever, paratyphoid fever and foodborne illnesses. There are two species in this genus: *Salmonella bongori* (previously called subspecies V) and *Salmonella enterica*

(formerly called *S. choleraesuis*). There are over 2500 serovars within both species, the vast majority of the human pathogenic isolates belong to the subspecies of *Salmonella enterica*, classified according to serology (somatic O antigen and flagellar H antigen).

6.5.7.1. Salmonellosis

Salmonellosis is a foodborne illness, caused by one of the numerous serovars of *Salmonella enterica* which can develop due to infected meat (poultry and cattle), raw milk, eggs, and raw egg products or, more generally, are caused by food cooked or frozen, not eaten straight away. Other sources include contaminated pets (reptiles and rodents)
The **symptoms** begin 6 to 72 hours after being infected and are characterized by certain *gastrointestinal* upsets, causing nausea, vomiting, fever, abdominal cramps, followed by diarrhea. In most cases, the illness lasts from 3 to 7 days, the affected persons recover without treatment. In case of some persons the diarrhea may be so severe that the patient becomes *dehydrated* and must be taken to hospital. Those suffering from an impaired immune system are more likely to be seriously ill. Some people, afflicted with Salmonellosis, can later experience a long-lasting reactive arthritis. Some infected people are carriers without having any symptoms.
The **Prevention**: involves an effective sanitizing of food-contacted surfaces. Food containing raw eggs should be thoroughly cooked or frozen before being eaten.

6.5.7.2. Enteric Fever

Enteric fever is the common name of the infections named typhoid and paratyphoid fever, caused by *Salmonella typhi* and *Salmonella paratyphi.*

6.5.7.2.1. Typhoid Fever

The infections of S. typhi are only suffered by human beings and are usually contracted through direct contact with the faecal matter of an infected person. Infections mainly occur in countries with no proper systems for the handling of human waste.
Symptoms: Typhoid fever, characterized by a *sustained fever as high as 40 °C (104 °F),* profuse sweating, bradycardia, malaise, headache, *abdominal pain,* gastroenteritis and *diarrhea,* (comparable to pea-soup) with six to eight stools per day. However, constipation is also frequent. *Spleen and liver are enlarged*, elevation of liver transaminases can be observed. A *rash*, like red coloured spots on the lower chest and on the abdomen may appear. *Delirium* is frequent. The patients are sometimes calm, sometimes agitated. On the third week of suffering typhoid fever, a number of complications, such as *intestinal hemorrhage* and *intestinal perforation* in the distal part of the ileum can occur, causing peritonitis or/and septicaemia. Complications, such as *dehydration,* encephalitis, abscesses, cholecystitis, endocarditis, osteitis etc. may also develop. The fever is permanently high, but by the end of the third week it slowly abates.
The most frequent resonances are:
Salmonella enterica: 329, 417, 524 kHz
Salmonella paratyphi: 365-370 kHz
Salmonella paratyphi B: 329, 337, 361, 364, 367-368, 376, 385, 398, 434, 483, 497 kHz
Salmonella paratyphi HC: 293, 466 kHz
Salmonella typhimurium: 382-387 kHz
Other frequencies of Salmonella typhimurium are: 339, 354, 386, 390, 395, 430, 553 kHz
Salmonella typhimurium B: 522, 559 kHz
Salmonella typhimurium HC: 306-307, 488-489 kHz

6.5.7.2.2. Paratyphoid Fever

This illness can be transmitted by animals or animal products to human beings or from person-to-person.
Symptoms: Paratyphoid fever is marked by *high fever, headache, loss of appetite, vomiting, and constipation or diarrhea.* The patient typically develops an enlarged spleen. Patients may have rosy spots on the front of their chest during the first week of the illness. The spots develop small haemorrhages. Patients with intestinal complications have symptoms resembling those of appendicitis. Most patients with paratyphoid fever recover completely, but in serious cases the intestinal complications can result in death.
Diagnosis: by using baterial culture-methods.
Prevention: by hygienic measures and vaccination
Treatment: with antibiotics
RFR method: detects and may eliminate the bacteria.
Its most frequent resonances are: 365-370 kHz

6.5.8. The Serratia Genus

The most common species of this genus is the *Serratia marcescens*, which is a facultatively anaerobic, Gram-negative bacterium. It is the only human pathogen bacterium of this genus, causing seldom occuring nosocomial infections of the bloodstream, the lower respiratory tract, the urinary tract, of surgical wounds, skin and soft tissues of adult patients. *Serratia marcescens* infection can cause keratitis, conjunctivitis, endophthalmitis and the inflammation of the tear ducts. It can be present in the respiratory and urinary tract of adults and the gastrointestinal tract of children, without causing any symptoms.
Its most frequent resonances are: 348-354, 445, 558 kHz

6.5.9. The Shigella Genus (Dysentery)

The members of this genus *(Shigella dysenteriae, Shigella flexneri, Shigella boydii* and *Shigella sonnei)* are non-motile, Gram-negative bacteria and the causative agent of shigellosis among human beings and other primates. Shigella infections happen by fecal-oral means, contaminated water or food, resulting in the destruction of the epithelial cells of the intestinal mucosa in the cecum and rectum. Some strains produce enterotoxin and shigatoxin, which latter is associated with causing the haemolytic uremic syndrome. Epidemics are most frequent in overcrowded populations with inadequate sanitation.
Symptoms: *severe diarrhea, fever, nausea, vomiting, stomach cramps,* lasting for several days. The stool may contain blood, mucus, or pus. Shigellosis may cause *delirium, convulsions*, and coma in case of *dehydrated* patients. Ulcers in the intestine resulting from shigellosis can lead to a severe loss of blood. The toxin of the bacteria causes *depression.* Uncommon complications include the damage of the nerves and of the heart, and sometimes even the perforation of the intestine. A permanent loss of the bowel control can also occur. *Shigella dysenteriae) type 1*, causes deadly epidemics in regions of many developing countries.
Shigella is held to be one of the pathogenic causes of *reactive arthritis.*
Treatment: with antibiotics
The most frequent resonances of Shigella flexneri are: 313, 318, 369, 388-396, 403-410, 423-425, 499 kHz
The most frequent resonances of Shigella dysenteriae are: 310, 315-321, 388-398, 410, 425, 496 kHz
The most frequent resonances of Shigella sonnei are: 318, 403, 506 kHz

6.5.10. The Yersinia Genus

Some members of the Yersinia genus are pathogenic concerning human beings. Its natural reservoirs are rodents. Infection may occur either through blood (e.g. in case of Y.pestis) or

by food products (vegetables, milk-derived products, meat etc.) contaminated with infected urine or feces.

6.5.10.1. Yersiniosis (Yersinia enterocolica)

Yersiniosis is a disease caused by Yersinia enterocolica. In the acute phase of the infection **the symptoms** are: *severe diarrhea, including fever and lymphadenopathy.* In its chronic phase, a cutanous-subcutanous, nonspecific vasculitis (erythema nodosum) may develop. The enterocolitis is sometimes followed by *reactive arthritis.*
The treatment: happens with a combination of antibiotics.
RFR method: detect and eliminates the bacteria.

6.5.10.2. Yersinia Pseudotuberculosis

This bacterium infects human beeings zoonotically, most often through a food-borne route. Its **symptoms** are similar to those of yersiniosis, but the diarrheal component is often absent. Without treatment it lasts usually for 1-3 weeks. *Fever and abdominal pain*, in the area of the appendix (mimicing appendicitis) are the most common signs of the infection. *Erythema nodosum, reactive arthritis or bateremia* can be seldom occuring complications caused by this bacterium.
Treatment: antibiotics may be necessary in complicated cases
RFR method: detects and may eliminate the bacteria.

6.5.10.3. Yersinia Pestis (Plague or Black Death)

Yersinia pestis (originally named Pasteurella pestis) is a Gram-negative, facultative anaerobic bacterium. It is the causative agent of all (such as the *bubonic*, the *pulmonic* and the *septicemic*) forms of the plague. These three forms of the so called Black Death were responsible for the epidemics with high mortality rate in the Middle Ages in Europe. The bacterium infects primarily wild rodents (such as rats, mice, squirrels) and prairie dogs. The infection can be transmitted from these animals to men through the bite of infected ectoparasites (usually fleas), or can also by inhaling infected droplets. In case of the more common bubonic form of the disease, Y. pestis gains entry into the human host through the bite of an infected flea. In most cases, the infective bacteria are carried to the local lymphatics, where, they cause a haemorrhagic inflammation, and thus get into the bloodstream and, finally, into the lymphatic system. Plague bacteria secrete several kinds of toxins.
The symptoms of the Bubonic plague usually appear within three days after the exposure to the bacterium. The patient is most likely becoming *restless, delirious and confused.* Liver and spleen may swell considerably. A hemorrhagic, oedematic zone surrounds the inflamed and suppurating group of the regional lymph nodes. The lymph glands are hyperplastic and show numerous areas of necrosis, swarming with bacteria. The name of this alteration is *bubo*. Metastatic lesions might sometimes develop in other lymphatics or in the viscera. Particularly likely is the occurrence of *secondary pneumonia*, which constitutes a potential source for the spreading of pneumonia. The **Septicemic plague** is a form of the bubonic plague, in which the infection spreads into the blood. This clinical form of the disease is referred to as „bubonic plague" because of the presence of enlarged suppurating lymph nodes, or buboes. *Bleeding into the skin and into other organs will occur, creating black patches in the skin. A secondary pneumonia* may develop and lead to the direct respiratory transmission of the illness by infectious aerosols from person-to-person. This primary pulmonic infection of the lungs is called **Pneumonic plague**. Its symptoms begin either at once or within two days after exposure to the bacteria via infected droplets, causing *high fever, lympadenopathy, chills, headache and caughing with haemoptysis.* This form of the human disease is highly fatal.

In case of this plague haemorrhages in the skin and other organs are numerous, most likely as the result of a toxin produced by Y. pestis. Even if the patients get the required chemotherapy or RFR method extensively enough, so that the bacilli are no more present in any organ, it is not unusual that they die due to toxemia. Pestis minor is the mild form of this plague.
Prevention: by vaccination, in case of persons working and residing in enzootic or epidemic areas where the avoidance of rodents and fleas is impossible.
Treatment Early treatment is expedient to prevent death. The treatment should be guided by antibiotic sensitivities, if available. Solely an antibiotic treatment is insufficient for patients, requiring circulatory, ventilative, or renal support.
RFR method: should begin as soon as possible, detects and eliminates the bacteria
Its general range is: 344-346 kHz
Its other frequency resonances are: 320, 327, 336, 340, 346, 372-376, 402, 409, 428-440, 442, 450-452, 503-507, 510-524 kHz

6.6. The Order of the Legionellales

The **Legionellales** are an order of Gammaproteobacteria comprising two families, typified by two humanpathogenic genus: i.e. the Legionella genus and the Coxiella genus.

6.6.1. The Legionella Genus (Legionellosis)

The Legionella genus includes at least 50 species and 70 serogroup. The human pathogenic Legionella species are the causative agents of the Legionnaires disease and the Pontiac fever. The transmission of the infection happens by inhalation of mist droplets containing the bacteria. Common sources are the cooling towers, central air conditioning systems, domestic hot-water systems, fountains, freshwater ponds and creeks.
Legionella pneumophila is the most common causative agent of legionellosis. This bacterium is a facultative intracellular parasite and can invade and replicate itself inside amoebae and in the macrophages of human beings. The infection can cause harmful symptoms solely regarding people with a compromised immune system.
The symptoms of Legionellosis takes two distinct forms. The **Legionnaires' disease** is the more severe form, in which, the incubation time is up to 2 weeks. After having *fever, chills and a dry cough, a severe form of pneumonia* will develop. The most severe forms attach the gastrointestinal tract and the nervous system. The **Pontiac fever** is caused by the same bacterium, but the patients have a *milder respiratory infection*, resembling influenza without pneumonia, *fever and muscle aches*. Some people can be infected with this bacterium and have only mild symptoms or suffer no illness at all.
Diagnosis: It is difficult to establish this illness solely by the symptoms of pneumonia or the radiologic finding, serological tests are needed.
Treatment of legionellosis happens with appropriate antibiotics. This Pontiac fever needs no treatment.
RFR method: detects and may eliminate these bacteria.
The most frequent resonances are: 350-355, 369-372, 402, 449-459, 499, 519, 566 kHz

6.6.2. The Coxiella Genus (Q-fever)

There is only one humanpathogenic species in this genus, the *Coxiella burnetii.* This small intracellular pathogen is the causative agent of Q-fever. The transmission of the infection occur by inhalation of infected dust, by handling infected materials, and possibly by drinking milk contaminated with C. burnetii. A number of cases have occurred among laboratory workers engaged in studies on C. burnetii. The disease can be not transmitted from person to person. Q-fever is an acute infectious disease.

Symptoms are characterized by a sudden onset of fever, malaise, headache, weakness with myalgia, anorexia, and after cc. 4 days an interstitial pneumonitis develop folloing an initial phase with dry cough and chest pain. The infection can be longlasting chronic.
Complications: hepatitis, hepatomegaly, and endocarditis
Diagnosis: serology and other laboritory examinations.
Differential diagnosis: other causes of acute pneumonitis
Prevention: immunization with specific vaccines.
Treatment: only if needed in severe casesantibiotics.
RFR method: in severe cases used simultanously with antibiotics treatment, detects and eliminates the C. burnetii bacteria.
The resonance frequencies of Q-fever are: 323, 347, 368-371, 482, 528, 534, 543, 562 kHz

6.7. The Order of Pasteurellales

The Pasteurellaceae are a family of Gammaproteobacteria with Gram-negative stains, given their own order. They are facultative anaerob bacteria. The human pathogenic genus of this family is the genus of Pasteurella and the genus of Haemophylus. The members of these geni cause only some few diseases among human beings. The most notable is the Haemophilus influenzae. Other human pathogenic Pasteurellaceae can cause gingivitis and chancroids.

6.7.1. The Pasteurella Genus

The members of this genus are non-motile pleomorphic bacteria. They are zoonotic pathogens, people can only get this infection if bitten by domestic pets. The species *Pasteurella multocida* is the most common cause of such infections.
The common **symptoms** of Pasteurella infections in man include *a swelling cellulitis* and *a bloody drainage* in the areas round the bite. In case of an immunocompromised person haemorrhagic bacteremia may develop.
Treatment: with antibiotics
RFR method: detects and may eliminate the bacteria.
The most frequent resonances are: 330, 355, 467 kHz

6.7.2. The Haemophilus Genus

The members of this genus are Gram-negative, pleiomorphic, small coccobacilli and include commensal species and some pathogenic ones too. The members are either aerobic or facultatively anaerobic.

6.7.2.1. Haemophilus Ducreyi (Chancroid)

This bacterium causes a sexually transmitted disease named chancroid.
Symptoms are characterized by one or more painful, sharply defined, genital ulcers (most often on the coronal sulcus by men and the labia majora by women) with diameter of 3-50 mm. The ulcers have irregular borders, and their bottom is covered with a yellowish-gray material that bleeds easily if hurted. The patients have regional lymphadenopathy, it can suppurate, if not treated.
Treatment: with antibiotics
RFR method: detects and may eliminate the bacterium.

6.7.2.2. Haemophilus Influenzae (Pfeiffer's bacillus)

This Gram-negative non-motile coccobacillus was considered to be the cause of the common flu quite until 1933, when the viral etology became known. This bacterium has six recognized serotypes, called *H. influenzae type A, B, C, D, E and F.* Most of its strains

are opportunistic pathogens, as they can only cause disease in certain circumstances: that is, in case of coinfection with a viral infection, reduced immune functionating etc. The characteristic response of the host to H. influenzae is an acute, suppurative inflammation in the tissues. The infections of the larynx, trachea, and bronchial tree are characterized by oedema of the mucosa and thick exudates. The bacterial invasion into the lungs causes *bronchopneumonia*. A severe, *diffuse bronchiolitis* may develop chiefly in case of young children.
In case of a severe H. influenzae *meningitis*, the brain is covered with thick greenish-yellow exudates. Hemophilus bacteria can grow in the upper airways of both children and adults but only rarely cause an illness.
Symptoms: Haemophilus influenzae type B (HIB) can cause *bacteremia* and acute *bacterial meningitis among infants* and young children. In some cases it can cause cellulitis, osteomyelitis, epiglottitis and arthritis. This bacterium is the major cause of *lower respiratory tract infections* among infants and children in countries, where no vaccine is used. Non-B-type Hemophilus influenzae strains usually cause otitis media, conjunctivitis and sinusitis among children and in some cases is associated with pneumonia.
Prevention: by vaccination (one must attend the data which point out that the Haemophilus influenzae B (HIB) infection or vaccination may be a risk factor for childhood (insulin-dependent) diabetes mellitus type 1.
Treatment: with antibiotics, such as Doxycyclin.
RFR method: detects and may eliminate the bacterium.
Its general range is: 335-337 kHz
The resonance frequencies of the HIB are: 335-338, 374-382, 424-426, 452, 482-494, 534-536, 564 kHz

6.8. The Order of the Pseudomonadales

There are only a few human pathogen bacteria in this order. They are all opportunistic pathogens, so also the species of Pseudomonas aeruginosa, Acinetobacter and Moraxella.

6.8.1. Pseudomonas Aeruginosa

The species *P. aeruginosa* (formerly *P. pyocyanea*) a member of the Pseudomonas genus can cause chronic opportunistic infections among immunocompromised patients and the ageing population. The bacterium infects typically the pulmonary tract, urinary tract, burns, wounds and can cause blood infections too. It is the most frequent colonizer of medical devices (e.g. catheters, ventilators etc.).
Serious Pseudomonas infections occur most frequently in hospitals. Patients suffering cystic fibrosis are predisposed to *P. aeruginosa* infections of the lung. The most common cause of all burn infections is *P. aeruginosa*. The malignant external otitis, an ear infection, can cause severe pain in the ear and also nerve damage, and is most common among people with diabetes or an immune-system damage. When *P. aeruginosa* is isolated from a normally sterile locus (f.i. blood vessel, bone, etc.), it should be taken seriously as it always requires some treatment. The bacterium is resistant to a large range of antibiotics and chronic infections can not be easily eradicated.
Treatment: antibiotic, choosed by antibiogram.
RFR method: detects and may eliminate the pseudomonas group.
Its general range is: 331-335 kHz
Its other frequency resonances are: 312, 323-326, 330-340, 348, 351-361, 364-367, 372-374, 377-380, 388-397, 401, 414, 422, 425-428, 438, 446-448, 453, 492-496, 504, 507, 512-513, 530, 547, 558, 579 kHz

Those of Burkholderia (Pseudomonas) mallei are: 325, 351, 377, 380, 396, 438, 448, 504, 513 kHz
Those of Pseudomonas aeruginosa (pyocyanea) are: 447 kHz
This list is not complete yet; there are more subspecies which have different radiofrequency resonances.

6.8.2. The Acinetobacter Genus

The members of this genus belonging to the family Moraxellaceae are able to survive on various surfaces (both moist and dry) in the hospital environment, thereby being important sources of infection in debilitated patients. The bacteria are generally considered nonpathogenic to healthy individuals, but in hospital environments they can cause severe, life-threatening infections in compromised patients, f.i. in intensive care units, causing nosocomial pneumonia or other infections of the skin or wounds. Bacteremia and meningitis can also develop. These bacteria are innately resistant to many classes of antibiotics, so that infections are often fatal.

6.8.3. The Moraxella Genus

Moraxella catarrhalis, a Gram-negative diplococcus is clinically the most important species of this genus. These bacteria are commensals of mucosal surfaces but often cause otitis media, sinusitis and, occasionally, bronchitis and pneumonia concerning children. Moraxella catharralis is sometimes the causative agent of opportunistic infections descending into the lower respiratory tract in case of immunodamaged persons, so that tracheobronchitis and pneumonia can develop (see also Chapter 11.6.3.).
M. catarrhalis endocarditis is a very rare illness occuring among patients with previous history of valvular conditions, balloon angioplasty or prosthesis, but also among patients, who were previously healthy. Sporadic cases of other infections caused by M catarrhalis include meningitis, neonatal ophthalmia, septic arthritis, keratitis and conjunctivitis as well.
Moraxella lacunata, can cause chronic blepharoconjunctivitis, angularis among people.
Treatment: antibiotic
RFR-treatment: detects and eliminates the bacteria
Its most frequent resonances are: 296-299, 350-352, 392-400, 512-550 kHz

6.9. The Order of the Thiotrichales

There exists only the Francisella genus belonging to this order, which includes pathogens infecting people.

6.9.1. Francisella Tularensis (Tularaemia or Rabbit Fever)

This bacterium species is a small, non-motile gram-negative pleomorphic coccobacillus, a facultative intracellular parasite of macrophages. It is the causative agent of tularaemia. Tularaemia is a bacterial zoonosis, in case of which the infection happens when contacting infected animals, vectors or their carcases. *F. tularensis* is worldwide found in more than 100 kinds of wild animals, birds and insects. This occurs in both terrestrial (rabbits, hares, ticks, and flies) and aquatic animals (muskrats and beavers). There exist four major strains, which differ in virulence and in their geographic range. For human beings and rabbits it is less virulent. The primary vectors are ticks and deer flies, though the disease can also be spread by other arthropods, too. The bacterium can penetrate the unbroken skin. Hunters, butchers, farmers, fur handlers, and laboratory workers are commonly infected. It can be transmitted also by eating inadequately cooked game meats or by being bitten by infected ticks, deerflies and mosquitos.

This disease can also develop by direct contact with contaminated pelts or by ingesting contaminated soil and water, as well as by inhaling water aerosols or dust from soil and grains. Transmission from person-to-person is rare, though possible among laboratory workers.
Depending on the locus of infection, tularemia has six characteristic clinical forms, such as ulceroglandular, glandular, oropharyngeal, pneumonic, oculoglandular and typhoidal.
The **Symptoms:** are depending on the way the infection is likely going to take.
If the infection occurs **via inoculation** (i.e. skin contact), the characteristic manifestations of the disease will be a mucous or cutaneous lesion at the locus of the inoculation and a regional lymph node enlargement as well. Some single ulcers will appear, generally on the arms or legs, many of them in the mouth or on the eyes, usually only on one eye. The regional lymph nodes at the spot of the ulcer will become enlarged, produce pus, and will later on drain. This is the most common *ulceroglandular* form of the disease. A subcutaneous inoculum of but 10 bacteria can cause illness.
In case of an aerogen inhalation of the bacteria, the infection leads to a potentially lethal *pulmonary tularaemia,* in which case, fever and severe pneumonia is going to develop.
By eating or drinking contaminated meat or water an *oropharyngeal infection* may come to pass, the initial symptoms of which include headache, chills, nausea, vomiting, and fever.
In case of the oculoglandular form the organism enters via the conjunctiva either by a splash of infected blood or by rubbing the eyes after having contacted infectious objects (f.i. blood from a rabbit carcase).
Its typhoidal form is more severe than the others causing often pneumonia.
Symptoms include an abrupt onset of fever and chills, headache, myalgia, malaise and fatigue, cough, dyspnea, vomiting, pharyngitis, abdominal pain, diarrhea, gastrointestinal bleeding, pneumonitis, photofobia, tender ulcers, lid edema, lymphadenopathy, lymphadenitis, adenopathy and anorexia. If the therapy is promptly begun death will occur in less than 1% of the cases.
Prophylaxis: The best way to prevent tularemia infection is to wear rubber gloves when handling or skinning rodents or lagomorphs (f.i. rabbits), to avoid ingesting uncooked wild game and untreated water sources, to wear long-sleeved clothes and to use insect repellents to prevent tick bites. Tularemia species can be antibiotics polyresistant. An attenuated, live vaccine is available, but is restricted for high risk groups.
Diagnosis: can be confirmed by identifying the growing bacteria in the samples of ulcers, lymph nodes, blood, or sputum by PCR, bacterium culturing combined with antbiotics resistancy examinations.
Treatment: with antibiotics, f.i. Streptomycin, Gantamycin, Doxycycline, Fluoroquinolons, etc.
RFR method detects and can eliminate this pathogen.
The most frequent resonances are: 330-336, 419-421, 429-440 kHz
The list of resonance frequencies is not yet complete; there are other subspecies (f.i. *F. novicida, F. philomiragia*) associated with septicaemia and pneumonitis, which have different frequencies, not exactly known as yet.

6.10. The Order of Vibrionales

The **Vibrionaceae** are a family of Gammaproteobacteria, according to their own order. They usually inhabit fresh or salt water. The members of this family synthesize Tetrodotoxin, an ancient marine alkaloid and a powerful neurotoxin (Na+ pump inhibitor). Several of their species are pathogenic.

6.10.1. The Vibrio Genus

Several members of this genus are clinically important human pathogens. Living in saltwater they all can cause food poisoning, associated with eating undercooked seafood e.g. crabs, prawns etc). Some can infect open wounds and cause septicemia too.

6.10.1.1. Vibrio Cholerae (Cholera)

The Vibrio cholerae is present and naturally attached to the zooplankton of fresh, brackish and salt water. Its infection is transmitted by contaminated water or seafood. In case of infection, the bacteria colonize the gastrointestinal tract producing enterotoxin (choleratoxin), which apparently is responsible for all known pathophisiologic alterations of cholera. The enterotoxin stimulates the adenylcyclasic activity of the intestinal epithelial cells, and the resultant increases in intracellular cyclic adenosin 3,5-monophoshate leading to the secretion of isotonic fluid by every part of the small intestine. The enterotoxin-induced electrolyte secretion occurs without any demonstrable histologic damaging of the intestinal epithelial cells or the capillary endothelial cells of the lamina propria.
The **Symptoms** usually start with a sudden, painless, watery diarrhea and vomiting. As the saline depletion progresses, severe muscle cramps are going to be felt, particularly in the calves. A severely ill cholera patient is typically cyanotic, a pinched face, scaphoid abdomen, poor skin turgor, thready or absent peripherial pulse can be experienced. The voice is faint, high-pitched, and often inaudible, tachycardia, hypotension, and varying degrees of tachypnea are present. If not treated, the resulting severe imbalances as regards the blood volume and the increased concentration of salt lead to kidney failure, hypovolemic shock and coma. The predominant causes of the death of inadequately treated patients are hypovolemic shock, metabolic acidosis, and uremia, caused by acute tubular necrosis.
Prevention: by proper sanitary practices, hygiene and vaccination
Diagnosis: by stool and swab samples, collected in the acute stage of the disease in order to identify the V. cholerae using the immobilization-test of vibrios, or by serotyping, by way of agglutination with specific sera, or by specific immunoflurescent methods.
Treatment: Prompt oral rehydration therapy or in most severe cases intravenous fluid therapy with saline and alkali – as the replacement of water and electrolyte is essential. Antibiotics, such as Doxycyclin, Erythromycin etc.
RFR method: used in conjunction with antibiotic treatment, detects and eliminates the bacteria.
The most frequent resonances are: 302, 336, 350-353, 372, 410, 416, 430-434, 453, 460, 495, 529 kHz

6.10.1.2. Other Vibrio Species

The *Vibrio parahaemolyticus* can cause severe gastroenteritis among people. *Vibrio vulnificus* can lead to an acute and fatal septicaemia.

6.11. The Order of the Xanthomonadales

There is only one opportunistic human pathogen species belonging to this order of gammaproteobacteria is the so called *Stenotrophomonas maltophilia,* which causes uncommon, difficult to treat infections among human beings. It is ubiquitous in aqueous environments, soil and plants, including water, urine, or respiratory secretions. Stenotrophomonas maltophilia colonizes endotracheal or tracheostomy tubes and urinary catheters. In case of immunocompromised persons the bacteria can lead to nosocomial infections. The most effective treatment in these cases is the removal of all prosthetic (plastic or metal) devices.
Symptoms are those of pneumonia, urinary tract infection or bacteremia.
Treatment: removal of the infected device, followed by giving antibioticum if needed.

RFR method: its characteristic frequencies are not known yet.

The Class of the Delta Proteobacteria

There exists no species belonging to this class of proteobacteria pathogenic in regard to human beings.

The Class of the Epsilon Proteobacteria

The Epsilonproteobacteria consist of only some pathogenic genera infecting human beings. They inhabit the digestive tract, as Campylobacter species is pathogenic regarding the duodenum and the Helicobacter pylori respecting the stomach.

6.12. The Order of the Campylobacterales

There are only two human pathogenic genus in this order.

6.12.1. The Campylobacter Genus

The members of this genus are motile, microaerophilic, flagellar bacteria. Campylobacter is often the cause of diarrhea among travelers visiting developing countries.

6.12.1.1. Campylobacteriosis (C. jejuni and C. coli)

The most common Campylobacter species causing bacterial foodborne diseases are the *Campylobacter jejuni* and the *Campylobacter coli.* The less common causes of illness are the *C. upsaliensis.* and the *C. lari.* The route of transmission can be fecal-oral, from person-to-person in case of sexual contact as well as the ingestion of contaminated food or water. The species cause tissue injuries of the jejunum, ileum and the colon. C. jejuni may invade and destroy the epithelial cells of the gut.

Symptoms: are diarrhea (in severe cases bloody or dysentery-like) with fever, abdominal pain and cramps. The infection is usually self-limiting. Some strains of the bacteria can produce cholera-like enterotoxin, in which case watery diarrhea presents itself. Now and then an association with a hemolytic uremic syndrome and thrombocytopenic purpura may occur. Among persons suffering from diabetes mellitus, cancer or among people otherwise immunocompromised, the infection can cause bacteremia.

Diagnosis: by laboratory analysis and bacterium serotyping.

Therapy: the supplying of liquids and electrolytes is usually sufficient. Antibiotic is only in severe cases needed.

RFR method: detects and may eliminate the bacteria.

Their general ranges are: 340, 352-357, 374, 522 kHz

Their other frequency resonances are: 340, 355, 361, 374-375, 387, 391, 402, 450, 466, 522, 535, 565 kHz

6.12.1.2. Campylobacter Foetus

This bacterium is, an opportunistic human pathogen, rarily causing bacteremia and thrombophlebitis among human beings. In case of newborns or among immunocompromised persons the infection can lead to fatal septicemia, meningitis, pleuro-pericarditis, arthritis and peritonitis.

The most frequent resonances are: 293-294, 365-371, 465-467 kHz

6.12.2. The Helicobacter Genus

The most common human pathogenic strains of this genus are those of the *Helicobacter pylori.* The fast-moving, microaerophilic, Gram-negative bacteria can produce large

quantities of enzyme urease and are able to raise the locally highly acidic pH of the stomach to a roughly neutral value. In the last years, numerous studies confirmed the carcinogen association between the Helicobacter pylori infection and the gastric adenocarcinoma. The infection significantly increases also the risk of gastric lymphoma of MALT (mucosa associated lymphoid tissue).
Symptoms: are like those of chronic gastritis, duodenitis and, in severe cases, peptic ulcers.
Diagnosis: by using both invasive methods (such as endoscopy), followed by histological examinations and bacterial culture procedures), and noninvasive methods (such as urease testing, as well as antibody testing of stool samples and in primary cases serological assays).
Treatment: Combining antibiotica (such as amoxicillin, tetracycline, metronidazole, azithromycine) with bismuth preparates. The eradication of the bacteria can sometimes be problematic.
RFR method: detects and may eliminate the bacteria.
The most common frequencies are: 346, 355, 360, 377-379, 449, 452, 554-562 Khz
Its other frequencies are: 346, 352-358, 360, 365-373, 377, 450-451, 554-562 kHz

The Phylum of the Chlamydiae

The members of this bacterial phylum are obligate intracellular pathogens, infecting eukaryotic cells. They have an intracellular replicative form as well as a surviving extracellular infectious form, and can be succesfully isolated but only inside the host cell. They are as small as the viruses, but more closely related to bacteria, having, similar to bacteria, cell walls with chemical and metabolic properties.

6.13. The Order of the Chlamydiales

The family Chlamydiaceae is belonging to this order, including two genera: i.e. Chlamydia and Chlamydophila. They are Gram-negative bacteria and express genus-specific LPS (lipopolysaccharide) epitops.

6.13.1. The Chlamydia Genus

The Chlamydia trachomatis species are the only human pathogenic members of this genus, infecting solely human beings. Chlamydia trachomatis organisms grow primarily in the epithelium of the conjunctiva, urethra, and cervix. The species has two biovars causing trachoma and lymphogranuloma venereum. Serotypes A, B and C were mainly associated with ocular Chlamydia trachomatis infections. Serotypes from D to K are the predominant types, being associated with ocular infections, non-gonococcal urethritis, and probably with cervicitis too.

6.13.1.1. Trachoma (Chlamydia trachomatis biovar 1.)

Trachoma is an infectious eye disease caused by Chlamydia trachomatis. The infection can be spread by direct contact with the infected secretion of the eye, nose and throat, or by contact with fomites, having contact with these secretions. The disease persists in many parts of the developing world, particularly in communities with no adequate access to water and sanitation. Epidemically, there are two types of the trachomatous eye disease: the endemic and the sporadic one. In the endemic areas of the world, transmission happens from eye-to-eye, while in non-endemic areas the sporadic trachoma is transmitted to the eye from the genital tract. Newborns and children are those who are most susceptible to the

infection. In endemic areas, the reinfection is rather common. An untreated infection can cause a, with adequate therapy preventable blindness.
Symptoms: The inclusion conjunctivitis begins 5 to 12 days after being infected, just like in ase of an *acute conjunctivitis with swollen eyelids*. Sporadic trachoma is usually at first manifested as an acute or subacute follicular conjunctivitis with keratitis and eye discharge. If inadequately treated with antibiotics, or improperly treated with corticosteroids, the disease may become worse: a painful scarring of the conjunctiva together with the swelling of the regional lymph nodes will develop, the eyelids will turn inward causing the eyelashes to scratch the cornea, corneal vascularization, leucocytic infiltration, and pannus formation, leading to visual loss will occur. Ear, nose and throat complications may also show up.
Prevention: by using the WHO-recommended strategies
Diagnosis: the Giemsa-stained or fluorescent antibody-stained smears of the conjunctival epithelium is considered to be an adequate proof of the trachoma infection.
Differential diagnosis: from adenoviral, herpes simplex, herpes zoster, rubella, measles, vaccina, smallpox, and dengueviral infections.
Treatment: by giving oral Erythromycin, Doxycyclin, surgery of eyelid deformities.
RFR method: following the antibiotic treatment.
The most frequent resonances are: 378-384, 430, 440, 482-483, 566-569 Khz
Its other resonances are: 303, 317-319, 430, 440, 443, 481-483, 566-569 kHz

6.13.1.2. Lymphogranuloma Venereum (Chlamydia trachomatis biovar 2.)

This disease is transmitted by sexual intercourse. It is generally caused by invasive serovars (L1, L2 or L3) of Chlamydia trachomatis Gaining entrance through breaks in the skin, the bacteria cross the mucous membranes and infect the regional lymphatics and lymphnodes.
Symptoms of the primary stage is a painless wound at the locus of the infection, healing within a few days. Some weeks later, the acute lymphogranuloma venerum is almost always showing non-specific systemic symptoms, including fever and leucocytosis, decreased appetite, malaise, nethertheless, severe systemic complications, such as meningoencephalitis do but rarely occur. The diagnosis at this stage of the illness is most difficult to state in case of women and homosexual men who may not have inguinal symptoms.
Symptoms of the secondary stage: depend on the place of the entry of the bacteria:

- into the genital mucous area (penis, vagina) it leads to the *inguinal syndrome* (such as abscess formation and drainage of lymphnodes even in case of a deeper lymphnode, cervicitis, metritis, salpingitis),
- into the anal area it leads to the *rectal syndrome* (proctocolitis, anorectal pain, tenesmus).
- The *pharyngeal syndrome* is rarely present. The pharyngeal tissue being infected, buboes (i.e. enlarged lymph nodes) in the neck region are apt to occur. The buboes are painful and being inflamed, the overlying skin becomes thin. The progress of the disease may lead to necrosis, suppuration, abscesses, fistulas and stricturas

Symptoms of the late stages vary. Fibrosis, lymphatic obstruction, causing chronic oedema and strictures, so that genital elephantiasis, fistulas of the penis, urethra and rectum may develop. The latter symptoms remain mainly permanent. A systemic spread may also happen, causing pneumonitis, hepatitis or arthritis.
Diagnosis: by using serologic methods, as well as PCR techniques
Treatment: with antibiotics (Doxycyclin, Erythromycin), and surgically, if necessary
RFR method: together with antibiotic treatment, it detects and eliminates the Chlamydia.
The most frequent resonances are: 317-320, 370-386, 429, 440, 444, 482, 566 kHz

6.13.2. The Chlamydophila Genus

This genus differs from the Chlamydia genus but very little. Since 1999, the time of reclassification, many Chlamydia strains were reorganized and counted into this genus. The full-length 23S rRNA genes of *Chlamydophila* and *Chlamydia species* are in less than 95% of the cases identical. Distinctions, such as EM morphology, antibiotic resistance and extrachromosomal plasmid are its typically species-specific characteristics.

6.13.2.1. Psittacosis (Chlamydophila psittaci)

Chlamydophila psittaci was previously classified as *Chlamydia psittaci.* Psittacosis is an infectious disease found in birds, the transmission from infected birds to human beings results in a febrile illness characterized by pneumonitis and systemic manifestations. Chlamydophila psittaci is transmitted by inhalation, by way of contact, or in case of birds by ingestion. The bacteria can infect human beings by inhalation. The term ornithosis is sometimes applied to infections contracted from other birds than parrots, but nevertheless, psittacosis is the preferred generic term for all forms of this disease. The bacterial invasion of the lung probably takes place rather via the bloodstream than by direct extension from the upper air passages. A lymphatic inflammatory response occurs on both the interstitial as well as the and respiratory surfaces of the alveoli and so also in the perivascular regions. The alveolar walls and the interstitial tissues of the lung thickens and becomes oedematous, necrotic, and hemorrhagic. Histologically, the affected areas show alveolar spaces, filled with fluid, lymphocytes, macrophages and neutrophil granulocytes.

Symptoms: The disease may start with chills, high fever and malaise. In case of apparent infections, mild influenza-like symptoms may appear. Chest pain, dry, hacking, usually nonproductive cough are characteristic; though small amounts of mucoid or bloody sputum may be raised as the disease progresses. Marked dyspnoe with cyanosis does only occur in cases of severe psittacosis with extensive pulmonary involvement. Pericarditis and myocarditis have also been reported. Headache is almost always a prominent symptom. Generalized myalgia is also common. Spasm and stiffness of the muscles of the back and neck may lead to an erroneous diagnosis of meningitis. Lethargy, mental depression, insomnia and disorientation were also prominent features of the illness in some epidemics. Gastrointestinal complaints, such as abdominal pain, nausea, vomiting, or diarrhea are also present in some cases. Icterus, the result of a severe hepatic involvement, is a rare but ominous finding.

Diagnosis: x-ray, bacterial culture, serological tests, and antibiotics resistance tests.

Differential diagnosis: viral and bacterial pneumonia, Mycoplasmal pneumonia, Q-fever, coccidiomycosis, tuberculosis, influenza.

Treatment: Doxycycline, Minocycline, and other broad-spectrum antibiotics.

RFR method: detects and may eliminate the bacteria.

The most frequent resonances are: 298, 311, 317-319, 336-340, 375-386, 429, 435-440, 444, 476, 480-486 kHz

6.13.2.2. Chlamydophila Pneumoniae (Chlamydia pneumoniae)

Chlamydia pneumoniae is a common cause of pneumonia all over the world. The bacteria infect otherwise healthy people. It affects all age groups and is most common among the 60-79 year old age-group. Reinfection may occur after a short period of immunity. This bacterium species is probably also associated with several other illnesses, such as meningoencephalitis, arthritis, myocarditis, fibromyalgia, CFS (chronic fatigue syndrome), atherosclerosis, asthma, etc.

Symptoms: are like those of *pneumonia* but caused by other microorganisms, including cough, fever, breathing difficulties, without any characteristic difference. *Pharyngitis,*

laryngitis and sinusitis are also often caused by infection due to Chlamydia pneumoniae. A physical examination cannot offer a difinite diagnosis either. Complications are rare.
Diagnosis: by antibody testing with reanalyzis or by using the PCR method. Chest x-rays often show opacity.
Treatment: by giving Doxycycline for 10-14 days.
RFR method: detects and may eliminate the bacteria.
The most frequent resonances are: 301, 317, 481-483, 490 kHz

6.13.2.3. Chlamydophila Abortus

This species of the family Chlamydiaceae causes abortion and fatal weakness or death of the fetus in mammals, human beings included.

The Phylum of the Actinobacteria

The members of this phylum are Gram-positive bacteria. The phylum includes some pathogens infecting human beings, such as the species of Actinomyces, Mycobacterium, Corynebacterium, Nocardia, Rhodococcus and Streptomyces. The phylum has its own class and the order of actinomycetales.

6.14. The Order of the Actinomycetales

6.14.1. The Actinomyces Genus (Actinomycosis)

The members of this genus belong to the class of the order of the Actinomycetales. They can be either anaerobic or facultatively anaerobic. Some form fungus-like networks of hyphae.
Actinomyces naeslundii species occupy the oral cavity and colonize the surface of the tooth. They can cause *periodontal diseases and root caries* as well.
Many *Actinomyces* species are opportunistic human pathogens and can cause actinomycosis, a rare disease characterized by forming of abscesses *in the oral, cervicofacial region, the lungs and the gastrointestinal tract.* The illness can be caused by many species, mostly by *Actinomyces israelii, Actinomyces bovis, Actinomycosis gerencseriae* and *Propionibacterium propionicus* (previously Arachnia propionica), the illness can possibly be polymicrobial. These bacteria are normal colonizers of the mucosa of the mouth, tonsils, the vagina and the colon. The frequency of the illness in regard to the face and the neck may be due to the great amount of Actinomyces israelii in loco. The disease can develop if the mucosal barrier is breached f.i. through a maxillodental infection of a carious teeth, tonsillar crypts, or by a trauma resulted by eating, dental procedures etc. The inflammatory reaction in case of actinomycosis is generally characterized by chronic suppuration, extensive necrosis, and intensive fibrosis in the end. *Propionibacterium propionicus* may produce draining sinuses too. The infection may spread by contiguity or through the bloodstream. Actinomycosis can occur also concerning women, who use intrauterine devices (IUDs) for contraception, or in case of diverticulitis. Misdiagnosed cases are frequent, often confused with neoplasms.
Symptoms: In case of an oral-cervicofacial infection, the lesion caused by the bacteria may appear near the jaw, a week or more after suffering certain kindes of trauma (such as tooth extraction, compound fracture of the mandible). As the lesion increases in size, points of suppuration (which later on become the openings of fistulas) develop in the livid-red-coloured oedematous skin. In other cases a small, flat, hard and painless swelling appears in the mouth or in the skin or deeper, below the jaw. Soft areas will develop thereafter, discharging a fluid, containing small, round, yellowish granules. The infection

may then extend to the cheek, tongue, throat, salivary glands, bones, or brain and its lining (meninges), especially in case of elderly patients.
In case of pulmonary actinomycosis, the infection can occur via aspiration. The anaerobic conditions of the atelectatic areas of the lung prevail in the development of the illness. In case of mediastinal actinomycosis, the spreading probably happens from the esophagus into the superior or posterior mediastinum, quickly invading the pleura, producing early pleural effusion or empyema. The infection may tend to attack the adjacent ribs and vertebral bodies. Prior to this, the patient may only notice fever, cough and expectoration. Symptoms of the *thoracic type*, such as chest pain, fever, weightloss and productive cough appear only in case of severe infections.
The abdominal type is caused by swallowing mouth secretions contaminated with this bacteria. The infections may affect the intestines and the abdominal cavity. Abdominal pain, fever, vomiting, diarrhea or constipation and severe weight loss are common symptoms. Ileocecal actinomycosis is the most common intestinal form, occurring after appendiceal rupture. The escaping infective bacteria form an inflammatory mass in the right iliac fossa. **In cases of generalized forms,** the bacteria may be carried in the blood to the skin, the spinal vertebrae, the brain, the liver, the kidneys, and the genital organs.
Diagnosis: by isolation of Actinomyces israelii in the sputum or the tissues. Hence, actinomycosis is characterized as a mixed anaerobic infection.
Treatment: antibiotic treatment with penicillin or some other effective agent for a long time (for months or even for a year). In extensive cases surgically.
RFR method: together with an antibiotic therapy, detects and eliminates the bacteria.
The general most frequent resonances of Actinomyces israelii are: 290, 372, 382, 395-398, 402, 418-421, 454, 476, 499 kHz
Its other frequency resonances are: 321, 337, 347, 353, 369, 376, 393, 401, 407, 452, 466, 473, 476, 485, 503-504, 551 kHz
The frequency resonances of other Actinomyces species are: 309, 341-374, 392-393, 420, 453-454, 499, 504, 578 kHz
The frequency resonances of Propionibacterium propionicus are: 308, 383-388, 414, 491 kHz

6.14.2. The Corynebacterium Genus

The members of this genus are Gram-positive, facultatively anaerobic, non-motile actinobacteria. A part of them are normal inhabitants of the human skin flora. Certain species are human pathogens, attacking healthy persons, certain others again attack immunosuppressed people.

6.14.2.1. Non-diphtheria (diphtheroid) Species of Corynebacterium

Nondiphtherial corynebacteria, originally thought to be mainly contaminants, recently have been recognized as pathogenic, especially in immunocompromised hosts.
Three strains of Different Corynebacteria are recognized, in decreasing order of virulence: gravis, intermedius, and mitis. These strains all produce an identical toxin. DNA of the phage integrates into the host bacteria's genetic material, the bacteria develop the capacity to produce this polypeptide toxin. The tox gene is regulated by a corynebacterial iron-binding repressor (DtxR). In the presence of ferrous iron, the DtxR-iron complex attaches to the *tox* gene operon, inhibiting transcription. Humans are the only known reservoir for the disease. The primary modes of dissemination are by airborne respiratory droplets, direct contact with droplets, or infected skin lesions. Immunization reduces the likelihood of carrier status. The toxin-induced manifestations involve mainly the heart, kidneys, and peripheral nerves. Cardiac enlargement is common and due to myocarditis. The kidneys become edematous and develop interstitial changes. Both the motor and sensory fibers of the peripheral nerves demonstrate fatty degenerative changes and disintegration of the

medullary sheaths. The anterior horn cells and posterior columns of the spinal canal can be involved, and the central nervous system may develop signs of hemorrhage, meningitis, and encephalitis. Death is mainly due to respiratory obstruction by the membrane or toxin effects in the heart or nervous system. Pathogenic Corynebacterium groups or species include the following: *C. diphtheriae*, *C. ulcerans*, *C. pseudotuberculosis (also known as C. ovis)*, *C. pyogenes*, *C. haemolyticum*, *C. aquaticum*, *C. pseudodiphtheriticum (also known as C. hofmannii)*, Group D2 (also known as *C. urealyticum)*, and Group E, *C. jeikeium (group JK)*.

Nondiphtherial corynebacteria are ubiquitous in nature and commonly colonize human skin and mucous membranes. Only recently has the role of these organisms in human infections been appreciated. In fact, many of these organisms cannot be speciated or typed easily, even in research laboratories, although recent advances in polymerase chain reaction (PCR) technology are improving our ability to identify these bacteria.

Because these corynebacteria are also pathogenic for animals (C. ulcerans, C. pseudotuberculosis, C. ovis), a history of exposure to sick animals or to animal products (milk, offal, hides) is common. C. ulcerans generally causes respiratory symptoms, while C. ovis produces a suppurative lymphadenitis.

In hosts colonized with diphtheroids (groups D2, JK), bacteria can be recovered from both skin and mucosal surfaces. C. striatum and C. pseudodiphtheriticum (or C. Hofmannii) are normal inhabitants of the anterior nares and skin. Symptoms relate to the organ system affected. Immunocompromised patients appear to have higher colonization rates than healthy persons and may have a greater chance of developing an infection after being colonized. Antibiotics resistance is also more common isolates from immunosuppressed patients.

Transmission from patient to patient, from colonized hospital staff to patients, and from environmental contamination to patients all are suggested. In antibiotic-resistant corynebacteria, transmission of the plasmid responsible for the resistance may be important.

Signs of diphtheroid-associated infection relate to the affected organ systems. Species of corynebacteria recovered from skin ulcers include C. ulcerans, C. bovis, and C. haemolyticum (Arcanobacterium haemolyticum). Those associated with bacteremia and sepsis include C. pyogenes, C. bovis, C. xerosis, and C. group D2, group E and group JK. These organisms are associated with endocarditis, prosthetic device infection, pneumonia, septic arthritis, and osteomyelitis.

Corynebacteria type D2 originally was identified as a pathogen causing chronic or recurrent cystitis, bladder stones, and pyelonephritis.

C. haemolyticum, is classificated and renamed nowadays as Arcanobacterium haemolyticum and was first described as Corynebacterium pyogenes, as some investigators believed the bacterium to be a mutant of this species and appended a subspecies name, C. pyogenes subspecies hominis. (This kind of bacterium is to be distinguished from Streptococcus pyogenes.)

Since its original description, the spectrum of diseases caused by *Arcanobacter haemolyticum* has been expanded to include invasive infections, including sepsis and osteomyelitis. The importance of A. haemolyticum to dermatology lies in the characteristic rash associated with pharyngeal infection. The smooth biotype predominates in wound infections, and the rough biotype predominates in respiratory tract infections. A. haemolyticum has been occasionally isolated in patients with sepsis, osteomyelitis, septic arthritis, cellulitis, wound infections, venous ulcers, skin abscesses, peritonsillar abscesses, cavitary pneumonia, pyothorax, paronychia, omphalitis, otitis media, endocarditis, sinusitis, orbital cellulitis, canaliculitis, meningitis, brain abscesses, diabetic soft tissue infections, and spontaneous bacterial peritonitis. The pathophysiology of the rash is caused by a bacterial exotoxin is reasonable and micro-hemorrhage. Phospholipase D,

neuraminidase, and a hemolysin have been identified as extracellular toxins secreted by A. haemolyticum. It has been described as erythematous, pruritic, urticarial, scarlatiniform, and maculopapular. This infection can produce coral red or violet-bluis skin color, hemorrhage, blood effusion, that may later turn into fine brown scales.
Erythromycin, Azithromycin, Clindamycin.
C. ulcerans usually causes skin infections but occasionally is associated with pharyngitis and respiratory disease.
C. striatum is found on catheters in patients who are neutropenic and have malignancies and has been recovered from the blood of patients with pleuropulmonary infections, endocarditis, or peritonitis.
C. pseudodiphtheriticum also is found in immunocompromised hosts, associated with both native and prosthetic valve endocarditis, pneumonia, lung abscesses, tracheobronchitis, and suppurative lymphadenitis.
Group JK can be found on the skin of healthy people. Patients with prolonged hospitalization, neutropenia, or on a prolonged course of antibiotics have a high prevalence for highly resistant JK bacteria. The most common manifestation is endocarditis with bacteremia, often associated with indwelling catheters.
Antibiotics are the treatment of choice for nondiphtherial and diphterial corynebacteria infections. Many antibiotics are effective, including penicillin, erythromycin, clindamycin, rifampin, and Doxycycline, Erythromycin, Azithromycin, Clindamycin, but may develops antibiotics resistance.
Diagnosis: serology and bacterium culture.
Treatment: antibiotics.
RFR method: detect and eliminate this bacteria.
The most frequent resonances are: 302, 315-319, 348, 356, 372, 383-389, 399-403, 409-410, 450, 460, 476, 492, 503-505, 578-578 kHz

The non-diphtheroid species can be found in many an environment, including soil, trees and the human skin or the mucous membranes. Diseases caused by Corynebacterium species include *granulomatous lymphadenitis, pneumonitis, pharyngitis, skin infections and endocarditis*. The latter develops in patients with indwelling intravascular devices (f.i. heart valves, shunts or catheters), the others mostly in cases of elderly, neutropenic or immunocompromised persons.
Trichomycosis axillaris, trichomycosis palmellina and trichomycosis pubis are superficial colonizations in the hair shafts of the sweat gland-bearing areas which are beleaved to be caused by the species *Corynebacterium tenuis*. The **symptoms** are characterized by yellow-reddish nodules that stick to the hair shaft.
Treatment: with locally given preparations containing benzoil peroxyd or erythromycin.

6.14.2.2. Diphtheria (Corynebacterium diphtheriae, Klebs-Löffler bacillus)

These causing agents of diphtheria are immobile, facultatively anaerobic, Gram-positive rods, pathogenic only in regard to human beings. The bacteria produce diphtheria toxin, a proteic exotoxin. This toxin inhibits the protein synthesis.
Its known four subspecies are: *Corynebacterium diphtheriae mitis, Corynebacterium diphtheriae intermedius, Corynebacterium diphtheriae gravis* and *Corynebacterium diphtheriae belfanti* all of which may be either toxigenic, causing diphtheria, or non-toxigenic.
Diphtheria is an acute infectious disease, characterized by a local inflammatory lesion, usually in the upper part of the respiratory tract (pharyngo-laryngitis), and by theremote effects of the diphtheria toxin, which particularly affects the heart and the peripheral nerves.

The most common portal of entry for the diphtheria bacillus is the upper respiratory system. Skin, genitalia, eyes and middle ear may also be loci of the invasion. The bacteria grow in most cases but superficially, invading the lymphatics or the bloodstream only in the terminal stage. The exotoxin elaborated in the local lesion will be absorbed and carried by the bloodstream to all parts of the body.
Symptoms: In case of infection, the primary lesion of the mucous membrane is thick and leathery, characterized by necrotic epithelium, phagocytes and fibrin. Regional lymphadenitis is frequently occuring. The toxic manifestations involve primarily the heart, the kidneys, and the peripheral nerves. The brain is rarely affected. Cardiac enlargement is also frequent; which fact appears to be related to myocarditis rather than hypertrophy. Cranial nerve dysfunction, loss of accommodation and difficulty in swallowing are the most often experienced toxic manifestations. However, any of the peripheral nerves may be affected, and can cause the paralysis of the extremities, the diaphragm, or the intercostal muscles. A severe failure of the respiration may cause death. Encephalitis is a rare toxic complication of diphtheria.
Prevention: with active immunization.
Diagnosis: by bacterial culture methods in Loeffler's medium; by immunofluorescence tests.
Treatment: with antibiotics
RFR method: used together with antibiotic treatment, detects and eliminates the bacteria.
The most frequent resonances are: 302, 319, 397, 403, 409, 434, 442, 458, 473, 544, 575 kHz
Those of Corynebacterium xerosis are: 400, 503 kHz
Those of other Corynebacterium species are: 348, 356, 372, 383-389, 396-399, 402, 409-410, 450-460, 476, 492, 505, 576-578 kHz

6.14.3. The Micrococcus Genus

Micrococci isolated from human skin are to be found in many other places of the environment, including water, dust, and soil. *Micrococcus luteus* can transform the compounds of sweat into compounds having an *unpleasant odour* on the human skin. The members of this genus are generally thought to be saprophytic or commensal organisms, though they can be *opportunistic pathogens* too in case of immunocompromised persons, such as HIV patients. As bacteria are normally present in the skin microflora, it can be difficult to identify *Micrococcus* as the true cause of an infection. In rare cases Micrococci may be involved in bacteremia, septic shock, septic arthritis, endocarditis, meningitis, and cavitating pneumonia concerning immunosuppressed patients.

6.14.4. The Mycobacterium Genus

Mycobacteria are in most cases non-motile, aerobic, acid-fast Gram-positive bacteria, with a thick, hydrophobic, waxy cell wall. Some species can be very difficult to culture, and some besides have extremely long reproductive cycles. Mycobacteria are typically living in water and food sources, except some of their species, including *Mycobacterium tuberculosis* and *Mycobacterium leprae,* which are obligate parasites. Mycobacteria can colonize their hosts without causing any adverse signs in their host. Billions of people around the world are infected with Mycobacterium tuberculosis, but being without symptoms, they will never know it.
The members of this genus can be classified into major groups for the purpose of diagnosis and treatment: **Mycobacterium tuberculosis complex**, the members of which can cause tuberculosis are: *M. tuberculosis, M. bovis, M. africanum* and *M. microti:* **Mycobacterium leprae**, causes leprosy (Hansen's disease) while the members of the **Nontuberculous Mycobacteria Complex (NTM)** can cause pulmonary diseases resembling tuberculosis, lymphadenitis, skin diseases and disseminated diseases.

6.14.4.1. Tuberculosis (Mycobacterium tuberculosis complex)

Tuberculosis is a contagious, potentially fatal infection caused by the airborne bacteria of *Mycobacterium tuberculosis, M. bovis, M. africanum and M. microti. Mycobacterium bovis* may be transmitted via unpasteurized milk. Tuberculosis is a necrotizing bacterial infection with protean manifestations and wide distribution. Most commonly it is the lungs that are affected, though lesions may also occur in the kidneys, bones, lymph nodes, or meninges, or can be disseminated throughout the whole body. The infection may also cause a clinical disease either shortly after the inoculation (primary tuberculosis), or after a period of months or even decades of dormancy. Although the infection may remain lifelong dormant, it may develop into a clinical tuberculosis at any time.

The immunity to tuberculosis is mediated largely by T-lymphocytes. Responsing to the specific antigen stimulation, they can liberate several lymphokines which promptly help by the phagocytosis and lysis of the mycobacteria. The role of the immunoglobulins in the immune response is less clear, though the specific IgA levels are often elevated in case of patients suffering from active tuberculosis and get lower after an effective therapy. The most easily obtainable evidence of a past or present tuberculotic infection is the experienced hypersensitivity to tuberculin, a specific protein derivate.

If there are no complications, the initial tuberculous infections do not cause any significant clinical symptoms. A massive haematogenous dissemination occurs mostly in case of recently infected children, younger than three years old. In case of older children, the infection progresses but rarely to its fatal form and passes often completely unnoticed. An initial tuberculous infection produces occasionally pleurisy with effusion, cervical lymphadenitis, miliary tuberculosis and meningitis. The immune system of a person infected with tuberculosis usually destroys the bacteria or seals them off at the spot of the infection. However, sometimes the bacteria are not destroyed, but remain dormant inside the scavenger white blood cells for many years. The reactivation of the dormant bacteria can occur, if the person's immune system becomes impaired, e.g. owing to AIDS, by the applied corticosteroids, or with chemotherapy.

Pulmonary tuberculosis may follow the initial infection at once or after a short or longer period of dormancy. The solid caseation, developed at the initial stage contains only few bacilli; in contrast with the liquid caseum in case of a tuberculous cavity, which contains abundant numbers of bacilli, so that the infection may spread via the bronchi to other parts of the lung or into its surroundings.

The earliest **symptoms in case of pulmonary tuberculosis** are constitutional and result chiefly from the liberation of lymphokines, stimulated by proteins, liberated from numerous bacilli of the hypersensitive host. In the late afternoon or in the *evening fever* is often present. *Weight loss* may precedethe other symptoms. *Cough* is frequently present, but is often disregarded as being a „cigarette cough". *Sputum,* odourless, green or yellow in colour befalls the patient principally when rising in the morning, sometimes accompanied by a small amount of blood. *Hemoptysis* may also be concomitant with the cough. In case of bleeding from the lung, the blood arises from the ulceration of the bronchial mucosa, causing streaks of bright red blood in the sputum. The bleeding usually subsides spontaneously if the patient lies still.)

A superficial tuberculous lesion may involve the overlying pleura and give rise to *dry pleurisy,* accompanied by localized pleuritic pain when taking a deep inspiration; or else a small caseous pulmonary focus actually erodes the visceral pleura and extrude a small amount of liquid caseum. In instance of such pleural contaminations, the immune response will be a vigorous inflammatory reaction with considerable *pleural exudates*. The onset of tuberculosis is occasionally rather acute, resembling the onset of a bacterial pneumonia. This situation occurs most often in case of persons with diabetes, children with an overwhelming infection and elderly patients. As to them, the lungs may be flooded with

bacilli discharged from an area of liquid necrosis in the lung or in the hilar nodes. Chills, fever, productive cough, pleuritic chest pain, and leukocytosis can be experienced. A minimal pleural contamination with a small superficial caseous focus will produce only clear exudates, however a massive contamination accompanied by the rupture of a large caseous lesion will produce *pneumothorax* and *tuberculous empyema.*

Bronchial ulceration may result in hemoptysis and a localized wheeze during respiration. The bronchial lumen may also be attacked by the pressure of the enlarged hilar lymph nodes even in the early course of infection.

The **normal gastrointestinal tract is resistant** to the penetration of tubercle bacilli. But in case of a cavitary pulmonary tuberculosis, associated with the excretion of large amounts of bacilli, the mucosa of the ileocecal region may be penetrated by them. In these cases, symptoms are as follows: *abdominal pain, cramps and diarrhea.* Occasionally, the infection spreads through the wall of the intestines, producing *tuberculous peritonitis.*

Localized tuberculous infections may occur in a number of other organs, notably in the lymph nodes, kidneys, long bones, genital tract, brain, and meninges.

The most common **involvement of lymph nodes** occurs in the hilus. The enlargement of the lymph nodes is usually modest but may sometimes be massive and able to give rise to obstruction and even to the ulceration of a major bronchus. Cervical lymphadenitis may appear as a late manifestation of the illness. Swellings begin insidiously without causing any systemic symptoms.

The kidney can also be a common locus of the late appearance of a localized tuberculous infection. The symptoms of renal tuberculosis are usually insidious and may easily be overlooked until *cystitis or epididymitis do not appear.*

The infection of the genital tract of males is secondary to renal tuberculosis. Bacilli, discharged from a caseous lesion in the kidney may reach the seminal vesicles, the prostate gland, and the epididymis because of their being connected. The infection usually causes *scrotal pain* caused by the inflammation of the epididymis and the vas deferens. *Tenderness and swelling of the vas deferens, seminal vesicles, and prostate gland* may also be experienced.

The infection of the genital tract of women may spread into the uterus and give rise to endometritis. The symptoms are usually mild, and begin insidiously with *abdominal pain, white vaginal discharge, metromenorrhagia, and dyspareuria.* Its most common manifestation is *sterility*, but in case of tubal scarring an *ectopic pregnancy* may also happen. The tuberculosis of the fallopian tubes may spread to the peritoneum too and thereby produce either a tuberculous *pelvic abscess* or a *generalized peritonitis.*

The tuberculous infection of the long bones usually begins at their endparts and may become obvious when involving the adjacent joints: the hip, knee, elbow, or wrist. Tenosynovitis is most common present at the wrist. **Spondylitis** may develop in childhood or be delayed until later in life. A paravertebral abscess looks like a fusiform density extending over the length of several vertebrae, occasionally dissecting downwards to the inguinal area.

Tuberculous infection may spread from the mediastinal lymph nodes or from the contiguous segments of the lung to the **pericardium** and also to the pleura.

Occasionally, the hematogenous tuberculosis localizes in the **adrenal glands**, which fact may result in their total destruction, causing adrenal cortical insufficiency.

Tuberculosis may involve the meninges, either as a sort of miliary tuberculosis or as an extension of the infection from a focus within the brain. Its symptoms are *headache, restlessness* and *irritability*, usually accompanied by *fever, malaise, night sweats and loss of weight. Nausea and vomiting* may be prominent. Stiffness of the neck and Brudzinski's sign are usually present.

A potentially life-threatening type of tuberculosis can develop if a large amount of bacteria spreads throughout the body via the bloodstream.

This infection is called **miliary or disseminated tuberculosis**, because of the millions of tiny lesions disseminated in the body of the infected person. The symptoms of miliary tuberculosis can be very vague and difficult to identify; they include *weight loss, fever, chills, weakness, general discomfort, and difficulty in breathing.* The involvement of the bone marrow may cause *severe anaemia* and *other blood abnormalities suggesting leukemia.* The intermittent release of bacteria into the bloodstream from a hidden lesion may cause fever that comes and goes, gradually wasting the body.

Diagnosis: by way of tuberculin skin tests, x-ray, CT, PCR, and MRI. The only absolute proof of an active tuberculosis is the cultural identification of Mycobacteria from tissues or body fluids such as sputum, gastric lavage, urine, or cerebrospinal fluid.

Differential diagnosis: carcinoma of the lung, mycotic infections, actinomycosis and nocardiosis, sarcoidosis, aspiration and other forms of pneumonia, pneumoconiosis.

Treatment: with a combinated chemotherapy administering e.g. Isoniazid, Rifampin, Pyrazinamide, Streptomycin, Ethambutol, Para-aminosalicylic acid (PAS), Cycloserin, Ethionamide, Kanamycin, Viomycin, Thiocetazone, Isoxil, etc. and surgery.

Prophilaxis: BCG vaccine.

RFR method: detects and may eliminate the bacteria. The role of RFR method is very important in drug-resistant cases. Using the RFR method after treatment with antibiotics or even simultaneously too.

The general range of the human tuberculosis is: 430-435 kHz

The resonant frequencies of Mycobacterium tuberculosis are: 328, 340, 344, 353-368, 372-378, 383-387, 396-397, 401, 409, 418-422, 429-436, 440, 471, 476, 512, 544, 548 kHz

The resonant frequencies of Mycobacterium bovis are: 325, 382, 387, 428, 448, 468, 534-538 kHz

The resonant frequencies of Mycobacterium avium are: 294, 301, 310-316, 320-328, 338-352, 341-348, 358, 393-395, 398, 403, 411-415, 421-458, 479, 487, 512, 530-531, 540-544 kHz

The resonant frequencies of Mycobacterium phlei are: 325-326, 409, 412 kHz

The resonant frequencies of non differenciated mycobacterium species are: 298, 328, 377, 387, 397, 410-411, 513, 544, 553 kHz

The resonant frequencies of TB vaccine are: 338-339, 374-377, 382-389, 435, 534, 555, 562 kHz

This list is not yet complete; there are other species with different frequencies not proved yet.

6.14.4.2. Leprosy or Hansen's Disease (Mycobacterium leprae)

The causative agent of leprosy is the Mycobacterium leprae. This is an obligate intracellular, pleomorphic, acid-fast, Gram-positive, aerobic mycobacterium.

Leprosy is a chronic granulomatous disease of human beings, which attacks the superficial tissues, especially the skin, the peripheral nerves, the mucous membrane of the nose, the testes and the eyes. The entry route of *M. leprae* into the human body is not definitely known. The two seriously considered way are the skin and the upper respiratory tract via droplets. About half of the people with leprosy probably contracted it through close contact with an infected person. The two exit routes of *M. leprae* from the human body are the skin and the nasal mucosa.

The two major clinical types are lepromatous and tuberculoid; when the disease has the features of both types, it is called borderline leprosy. In addition, an **early indeterminate form** can also be seen, which may later develop into one of the three types mentioned. In **lepromatous leprosy** bacillemia is frequent (**multibacillary**)and often so profuse that the organisms can be seen in stained smears of peripherial blood. Even in the most advanced lepromatous cases, the destructive lesions are limited to the skin, peripheral nerves, to the

anterior portion of the eye, to the upper respiratory passages above the larynx, testes, and to structures of the hands and feet. The patients suffering from lepromatous leprosy are deficient in their ability to develop delayed type of hypersensitivity; their lymphocyte transformation response is weak, and the paracortical areas of their lymph nodes are deficient in lymphocytes. For these reasons, lepromatous leprosy is thought to be the result of a poor immune response, and tuberculoid leprosy the result of a stronger immune response. But whether these differences in immune state precede the infection, or are caused by it, remains to be clarified. Mycobacterium leprae multiplies very slowly, and thus symptoms usually do not begin until at least one year after a person has been infected.

Lepromatous leprosy is one of the polar forms of the disease. The involvement is extensive, diffuse, and bilaterally symmetrical. Histologically, there is a diffuse granulomatous reaction with macrophages, large foam cells, and many intracellular bacilli, frequently in spheroidal masses. The leptomin reaction is negative. The skin lesions are macules, nodules, or papules. The macules are often hypopigmented. The borders of the lesions are not sharp, and the centers of raised lesions are convex. There is a diffuse infiltration between the lesions. The sites of predilection are the face, ears, wrists, elbows, buttocks, and knees. Nasal symptoms are common, early complaints. Complete nasal obstruction, followed by laryngitis and hoarseness, are also frequent. Septal perforation and nasal collapse lead to saddlenose. Painless inguinal and axillary lymphadenopathy occur.

Diffuse hypesthesia involving the peripheral portion of the extremites is common in advanced lepromatous disease.

Tuberculoid leprosy is the other polar form of the disease. Skin lesions appear singly or are few in number, and are sharply demarcated. The neurologic involvement is relatively pronounced and may be severe. The histologic picture consists of lympocytes, epitheloid cells, some giant cells and bacilli are such a few, **(paucibacillary)** that sometimes difficult to demonstrate. The leptomin reaction is usually positive.

In cases of **borderline**, or **dimorphus leprosy**, the clinical features and the histological changes represent a combination of the two polar types.

Early tuberculoid leprosy is frequently manifested by a hypopigmented macule that is sharply demarcated and hypestetic. Eventually the lesions increase in size, and the margins become elevated and circinate or gyrate. The process is marked by peripheral spread and central healing. The lesions are not symmetrical. Nerve involvement occurs early, and the nerves leading from the lesions may become enlarged. The larger peripheral nerves may be palpably and visibly enlarged, especially the ulnar, the peroneal, and the greater auricular nerves. There may be severe neuritic pain. The neural involvement leads to muscle atrophy, especially of the small muscles of the hand. When the facial nerves are involved, there may be lagophthalmus, exposure keratitis, and corneal ulceration leading to blindness.

Diagnosis: lepromin reaction, bacteria culture, and biopsy histology.

Differential diagnosis: lupus erthematosus, lupus vulgaris, sarcoidosis, yaws, dermal leishmaniasis, tuberculoid disease, and syringomyelia.

Treatment: a multidrog treatment is recommended Rimfampin, Clofazimine, Dapsone, etc. Antibiotics can arrest the progression of leprosy or even cure the disease, but some of the mycobacteria may be resistant to certain antibiotics.

RFR method: used simultaneously with antibiotics; detects and eliminates the bacteria. Nota bene: the antibiotic-resistant Mycobacteria are not resistant to RFR method.

Its most frequent resonances are: 307, 319, 332-340, 353-358, 372, 380, 383-384, 389, 396-410, 438, 450-460, 476, 510, 516 kHz

6.14.4.3. The Nontuberculous Mycobacteria Complex (NTM)

To this group of Mycobacteria belong some *Mycobacterium avium subspecies,* forming the **Mycobacterium avium complex (MAC).** They can cause infection most commonly in cases of immunocompromised people, when inhaled or swallowed. Their symptoms are reminiscent of tubeculosis, such as fever, fatigue and weight loss. The pulmonary involvement is similar to TB, diarrhea and abdominal pain are the symptoms of the gastrointestinal involvement.
Treatment involves a combination of anti-tuberculosis antibiotics, but these bacteria are highly resistent to most antibiotics. (See Chapter 6.14.4.1.)

6.14.5. The Nocardia Genus

The members of this genus are Gram-positive, rod shaped bacteria. These microorganisms are relatively acid-fast, and their bacillary form resembles the tubercle bacillus. Nocardia species, in contrast to the tubercle bacillus, grow rapidly also on Sabouraud's medium and appear in exudates as long-branched, gram-positive mycelial forms. Some species are pathogenic with low virulency and can cause illness, named **nocardiosis**. Infections occur mostly among immunocompromised persons, however, a lot of mostly elderly persons, suffering nocardiosis, have no preexisting disease. Conditions or therapies, suppressing a person's immune system are f.i.: increased age, AIDS, cancer, chemotherapy for cancer, radiation therapy, immunosuppressive drug therapy, corticosteroids, autoimmune diseases, mononucleosis infectiosa, etc. The *Nocardia asteroides* is the species of the Nocardia genus, which most frequently causes nocardiosis. Nocardia asteroides usually live on decaying matter in the soil. The bacteria are carried by air, contaminated with soil dust and can be breathed into the lungs. In rare cases, the bacteria enter the body by being swallowed or by traumatic introduction through the skin. The illness caused by these fungi-like Nocardia asteroides bacteria, usually starts in the lungs and then can spread to the skin and the brain.
Symptoms: Nocardiosis often starts as a lung infection, such as a slowly progressing pneumonia. The symptoms are similar to those of tuberculosis. The first complaint is a *cough,* which usually produces a thick, and sometimes bloody *sputum. Chest pain, dyspnea, fever, sweats, chills, leukocytosis, general weakness, anorexia, and weight loss* are common. Fluid may collect in the pleural space. Bacteria can spread throughout the bloodstream, in some cases causing pockets of abscesses in many areas of the body, including the brain, the skin and less frequently the kidneys. Abscesses develop in or beneath the skin in about a third of the cases. Brain damage usually occurs in and around the thalamus, and in cases of elderly persons often develops the clinical picture of the parkinsonism. By nearly one-third of the patients, the pulmonal symptoms are suddenly followed by *acute neurological changes* of a metastatic brain abscess. The patients experience *severe headache and focal sensory, or motor disturbances.* Nocardia toxins can produce brain damages too. Nowadays, nocardiosis is considered to be the cause of some chronic brain infection. Parkinson's disease may be perhaps resulted by this chronic progressive disease.
Nocardia species (especially *Nocardia brasiliensis)* may cause cutaneous infections, such as *actinomycetoma, cellulitis, subcutaneous abscesses.*
Diagnosis by identifying Nocardia asteroides in samples of body fluid or tissues taken from the infected person. With or without treatment, acute nocardiosis can be fatal in cases of immunosuppressed individuals.
Treatment: antibiotic, f.i. sulphonamide, amikacin etc. for a very long time (min. six month), but there are many antibiotic-resistant groups viable, and many antibiotics do not penetrate across the blood-brain dam.
RFR method: useful for detection; antibiotic treatment is necessary, followed by RFR method, which can eliminate the nocardia.
Their general ranges are: 352-356, 361-371 kHz

Their other frequency resonances are: 355, 368-369, 382, 450, 454, 466, 473, 485, 565 kHz

6.14.6. The Propionibacterium Genus

The members of this genus are commensal inhabitants of the human skin and usually non-pathogenic. These species are slow-growing, non-sporulating, Gram-positive anaerobic bacilli.

The *Propionibacterium acnes* is mostly present on the skin and linked to the skin disease *acne*. Living on fatty acids in the sebaceous glands and on the sebum secreted by pores, it may also be found throughout the gastrointestinal tract. This bacterium can but seldom cause *chronic blepharitis* and endophthalmitis, which latter can occur following an intraocular surgical intervention.

The *Propionibacterium propionicus* is held to be a less common causative agent of a disease process similar to that of actinomycosis.

Treatment: antibiotic

RFR method: detects and may eliminate the bacteria.

The resonant frequencies of Propionibacterium propionicus are: 308, 383-388, 414, 491 kHz

6.14.7. The Rhodococcus Genus

The species of this genus are facultative, intracellular, nonmotile, non–spore-forming, gram-positive coccobacilli. One species of this genus, i.e. the *Rhodococcus equi* (RE) causes primarily zoonotic infections, nevertheless it has become an important opportunistic pathogen among immunocompromised patients, especially among those with an acquired immunodeficiency syndrome (AIDS). The human RE infection is associated with significant mortality. The exposure happens usually by inhalation, the infections come off by via oral route.

Symptoms: The primary locus of the infection is the lung, from where dissemination as well as and bacteremia is likely to begin. Necrotizing pneumonia is the most common form of the infection caused by *Rhodococcus equi*. The extrapulmonary infections caused by this pathogen include diseases such as wound infections, subcutaneous abscesses, brain abscesses, thyroid abscesses, retroperitoneal abscesses, peritonitis, meningitis, pericarditis, osteomyelitis, endophthalmitis, lymphadenitis, lymphangitis, septic arthritis, osteitis, bloody diarrhea and fever of unknown origin.

The infection among children caused by this bacterium differs from that observed in adults. Immunocompromised conditions account for only about one third of the cases reported. The immunocompromised conditions in children with these infections include hematopoietic malignancies, immunosuppression associated with chemotherapy, and HIV infection. Among immunocompetent children the infection accounts for approximately one third of the cases which is probably due to the increased occurrence of trauma among children, as it is predisposing them to localized wound infections with RE. The prognosis concerning immunocompetent children is extremely favorable.

Pulmonary infections cause fever and cough, malaise, chest pain, dyspnea, hemoptysis and loss of weight. Pneumonia of a chronic or relapsing form does possibly not respond to an empirical treatment.

Other presentations of *Rhodococcus equi* infection include lymphadenopathy, eye drainage and pain, joint pain, an altered level of consciousness, bloody diarrhea, anemia or fever, while the other form includes tachypnea, crackles, and other common physical findings of pneumonia, lymphadenopathy, septic arthritis, meningitis, corneal laceration, hyperemia, a decreased visual acuity, which is an evidence of anterior chamber involvement and it has soft tissue masses, induration, fluctuance in localized infections.

Rhodococcus equi has been found in bovine, porcine, and equine fecal flora growing best at summer temperatures. In case of patients in an immunocompromised state (such as malignancy, recent chemotherapy, solid organ transplantation or bone marrow transplantation, diabetes mellitus, alcoholism, and immunosuppressive medications such as corticosteroids) it should be made sure, that the ambient air of the horse farms on dry windy days does not contain RE organisms. Anamnestic data pertaining to sexual practices and abusus with iv. drug injections are also important.
Diagnosis: by taking RE blood cultures and culture from other infected sources, such as abscesses, eye drainage, and cerebrospinal fluid examinations, detect coinfections f.i. HIV, mycoplasma and others. A plain radiograph in osteomyelitis may demonstrate an osteolytic lesion. CT scan, x-ray and MRI study may demonstrate a mass with a necrotic center. Appropriate imaging is also necessary in cases of meningitis, brain abscess, and abdominal infections.
Treatment: by administering Vancomycin (Vancocin), Rifampin (Rimactane, Rifadin), Erythromycin (EES, E-Mycin, Eryc), Ciprofloxacin (Cipro), Gentamicin (Garamycin), Imipenem and cilastatin (Primaxin), and Meropenem (Merrem IV).
RFR method: favorable done in case of brain processes. Detects and eliminates RE.
The most frequent resonances are: 345-349, 368-369, 427-428, 442, 507-508 kHz
Detects and eliminates immunosuppressant coinfections. Use RFR with antibiotics!

6.14.8. The Streptomyces Genus

The species of the Streptomyces genus are producing a lot of antibacterial, antifungal, immunosuppressive, antitumorous and other bioactive compounds. Streptomyces rarely cause infections. *Streptomyces sudanensis* and *Streptomyces somaliensis* are sometimes the isolated pathogens of mycetoma cases to be found in African countries.

The Phylum of the Firmicutes

The human-pathogenic bacteria grouped into this phylum have mostly aGram-positive cell-wall structure. They are generally susceptible to penicillin and usually slow to develop resistance to this antibiotic. Their cells are round, (called cocci) or rod-shaped. Many of them produce endospores getting thus resistant to desiccation. Some Gram-positive bacteria can penetrate deep into the tissue, while others inflict harm by producing extremely poisonous substances. The phylum is greatly diverse in its phenotypic characteristics due to the promiscuous plasmid exchange between its species and genera. Some species of the Mollicutes genus (mycoplasma) have no cell walls, and are thus not Gram-positive, moreover they even lack the second membrane found in Gram-negative bacteria. Some of its members are part of the gut flora and thus involved in the energy resorption and obesity.
There are three classes classified into this phylum: the class of Bacilli, Clostridia and Mollicutes.

The Class of Bacilli

The members of this class are either obligate or facultative aerobes. Both order of the Bacillales and the order of the Lactobacillales belong to this class.

6.15. The Order of the Bacillales

There are three human pathogenic familiae in this order: the Bacillaceae, the Listeriaceae and the Staphylococcaceae familia, including three representative genera, causing infections among people.

6.15.1. The Bacillus Genus

There are two Bacillus species, considered as medically significant in this genus: Id est: The species *Bacillus anthracis* which, infecting human beings can cause anthrax and the species *Bacillus cereus*, causing foodborne illnesses. They appear as rods and usually possess an oval endospore at one of its ends.

6.15.1.1. Anthrax (Bacillus anthracis)

Anthrax is a disease caused by the bacteria named *Bacillus anthracis*, which can infect the skin, the lungs and the gastrointestinal tract. The bacterium is a Gram-positive, facultatively anaerobic, soil-dwelling bacillus. The bacteria produce three exotoxins: the protective antigen, the oedema factor and the lethal factor. The bacteria form endospores, which can live in the soil or in animal products for decades, and are very hard to eradicate. After ingesting or getting spores into a cut in the skin, a new host allows the spores to reactivate themselves and to multiply in their new host very rapidly. Some strains of this species are highly virulent, so that the disease can be highly contagious and potentially fatal. The infection can spread over to people from animals, especially from cows, goats and sheep. People are usually contaminated with the bacteria via their skin or by inhaling the spores of the bacteria. Anthrax cannot be spread directly from person-to person; but the spores can be transported by one's clothing, shoes, etc. and if persons die of anthrax their body can be a very dangerous source of anthrax spores.

Symptoms:

The skin infections begin with red bumps that get enlarged, swelling considerably at their edges. The characteristic cutaneous lesion is a painless necrotic ulcer with a black center (eschar), which develops on the spot where the spore had penetrated, within 2-5 days after being infected. The lymph nodes in the affected area may swell, the infected person may feel ill, sometimes experiencing muscle aches, headache, fever, nausea and vomiting. The disease may also be associated with a disseminated infection, which, if not treated, can lead to toxaemia and death.

The pulmonary process begins by way of the inhalation of spores, which then are transported through the air passages into the alveoli of the lungs. The spores picked up by macrophages of the lungs and transported by lymphatics to the lymph nodes in the mediastinum, cause chest pain and breathing difficulties. The first symptoms are *mimicking those of an influenza* for several days, and are then followed by a severe (and often fatal) *respiratory collapse*. After being multiplied in the lymph nodes, the bacteria get into the blood stream (bacillemia) and release their exotoxins causing tissue destruction, *bleeding, septic shock and even death.* Anthrax attacks the endothelial cells too, causing vascular leakage similar to hemorrhagic bleeding, and hypovolemic shock aswell. The respiratory anthrax (which means inhalation of cc. 10 000-20 000 spores) is highly fatal, having a mortality rate of nearby 100%.

A gastrointestinal infection is mostly caused by eating anthrax-infected meat, and is characterized by vomiting of blood, severe diarrhea and the acute inflammation of the intestinal tract. After invading the bowel system, bacteria spread along the bloodstream causing toxaemia. Gastrointestinal infections may cause fatality rates of 25-60%, depending on how soon treatment is commenced.

Treatment: It is essential to begin the antibiotic treatment as early as possible.

Prevention: by vaccination.

RFR method: must be commenced as early as possible! Detects and eliminates the bacteria!

The most frequent resonances are: 318-329, 393-401, 512 kHz
Its other frequency resonances are: 293, 309, 314, 349-350, 359-370, 409, 422-423, 460-461, 466, 476, 492, 501, 510, 515, 532 kHz
The resonant frequencies of its spores are: 380-396 kHz

6.15.1.2. Bacillus Cereus (Fried rice syndrome)

These beta-haemolytic, facultatively aerobic, Gram-positive bacteria cause foodborne illnesses, such as *heavy nausea, vomiting and abdominal pain.* Bacterial spores cause the illness in case of eating infected food improperly cooked. The infection causes two types of illnesses: the *diarrheal type* which is characterized by a long incubation time from 8-16 hours, and the *emetic type* with a shorter incubation time (1-5 hours). The symptoms develop depending on the diverse enterotoxins of the *Bacillus cereus* species.
The most frequent resonances are: 298, 372-376, 475 kHz

6.15.2. The Listeria Genus (Listeriosis)

The *Listeria monocytogenes* species is the human pathogenic member of this genus. The disease, caused by this bacillus is named **listeriosis**, a rare, but sometimes fatal *foodborne* illness. Listeriosis is an opportunistic pathogen: it mostly prevails among elderly people, pregnant mothers, and AIDS patients.
The severity of the disease is given by two facts: by the grade of the ability of the bacilli to spread into the nervous system to cause there meningitis, and by the degree of its potency to infect the fetus by penetrating the endothelial layer of the placenta of its infected mother.
At the beginning of the infection the bacteria will be phagocytosed by the host's macrophages, remaining then there as intracytoplasmatic parasites, replicating themselves inside of the host cells.
Symptoms: In case of adults, the most common form of listeriosis is the meningitis. The infection affects the membranes covering the brain and the spinal cord, producing *fever* and a *stiff neck*, *confusion*, *coma*, and, possibly, even *death*.
This disease sometimes infects the *eyes*, making them red and painful. The infection can then spread to the lymph nodes, into the blood and the meninges.
Treatment: by antibiotics.
RFR method: detects and may eliminate the bacteria.
Its general ranges are: 320-326, 396, 482, 553 kHz
Its other frequency resonances are: 365-372, 386, 503 kHz

6.15.3. The Staphylococcus Genus

The members of this Gram-positive genus have a spherical shape, appearing under the microscope as round (cocci) forming grape-like clusters. The genus includes several significant human pathogens, others again, are harmless and reside normally on the skin and the mucous memranes of human beings and animals. They represent a small component of the microbial flora of the soil. *Staphylococci* can cause a wide variety of diseases attacking human beings and animals owing to their capability of toxin production and invasion. Highly prone to staphylococcal infections are breastfeeding women, newborns, people with chronic diseases, adverse skin conditions and those suffering surgical incisions, and people whose immune system is suppressed by corticosteriods, radiation therapy, immunosuppressive drugs, or cytostatic treatment. The best known human pathogenic species are the *Staphylococcus aureus*, the *Staphylococcus epidermidis* and the *Staphylococcus saprophyticus.*

6.15.3.1. Staphylococcus Aureus

This species, the most infective one in this genus, is a facultatively anacrobic and opportunistic pathogen. It can survive on dry surfaces with an increased chance of being transmittable. These cocci frequently live on the skin or in the nose and throat of a person (a socalled „staph. carrier") without causing any remarkable harm. *S. aureus* infections can be spread by contact with the pus of an infected wound or other infected objects, by skin-to-skin contact with an infected person, by producing hyaluronidase destroying tissues. *S. aureus* can infect other tissues as well, if the normal barriers have been breached (e.g. skin or mucosa). The species can infect any part of the body, the symptoms depend on the location of the infection. In case of surgical operations nosocomial infections are often caused by this pathogen. Staphylococcus aureus is extremely prevalent on the skin of patients suffering from atopic dermatitis so that their skin disease may get worse.

Skin infections: are often caused by the Staphylococcus aureus species. A break or other injury of the skin may help the bacteria to attack the defense-mechanisms of the body and thus cause infections. The most common pyogenic minor illnesses of the skin are *pimples, folliculitis, furuncles, carbuncules*. This pathogen is also one of the most common causes of closed-space infections of the fingertips, called *paronychia*. Staphylococci can also cause *cellulitis* and are generally the cause of nosocomial postsurgical wound infections.

Getting into deeper tissues via the bloodstream, the infection can cause **abscesses** in the internal organs, that is the *lungs*, *osteomyelitis* in the bones and *endocarditis* involving the inner lining of the heart and its valves. The staphylococcal **osteomyelitis** predominantly affects children, though elderly people are likewise susceptible. A staphylococcal **endocarditis** typically runs an acute course with high fever, progressive anaemia and metastatic abscess in the skin and in deeper structures.

Staphylococcal bacteremia often causes death of severely burned people. Being generally accompanied by a polymorphonuclear leukocytosis it can lead to *meningitis*, of the brain and the spinal cord, *pneumonia, and/or carditis*.

Depending on its strain, the bacteria can secrete several toxins.

The *pyrogenic toxin* of certain strains causes the **Toxic Shock Syndrome** (TSS). It is an exotoxin named toxic shock syndrome toxin-1 (TSST-1) and is the main toxin produced by *S aureus* strains. TSS is characterized by a sudden onset of fever, chills, vomiting, diarrhea, muscle aches and rash. Anaemia can develop within the first week, as well as kidney, liver, and muscle damage leading to severe hypotension and multisystem dysfunction. The process may rapidly turn into severe, untreatable shock. Desquamation, particularly on the palms and soles can occur 1-2 weeks after the onset of the illness.

The *exfoliative toxins* of some strains are present in a serious skin disease called **Staphylococcal Scalded Skin Syndrome (SSSS)**, also known as Ritter-Lyell syndrome occuring among *neonates (aged 1-3 months)* or young children, suffering with an impaired immunity and most often even a renal insufficiency. The causative strain of the disease is the Staphylococcus aureus group 2 (mostly the *phage type 71*). The exotoxins with their protease activity detach the epidermis. The clinical picture is marked by erythema, exfoliation, followed by skin necrosis, making the skin look as if it were scalded. The complications (e.g. fluid and electrolyte loss, sepsis, involvement of other organs of the body) may cause death with a mortality rate of 2-3%.

The Staphylococcal food poisoning (such as f.i. from cheese or salami) is caused by certain strains producing *pyrogenic enterotoxins*. The pathogenic bacteria can multiply f.i. in improperly-stored food. The illness is characterized by fever, diarrhea, abdominal bloating and distention.

Other staphylococcal toxins, attacking the cell membranes are as follows: *alpha-toxin, beta-toxin, delta-toxin, and several bicomponent toxins*. The latter, called Panton Valentine leukocidin is associated with the developing of **severe necrotizing pneumonia** among children.

Diagnosis can be confirmed by way of laboratory examinations.

Treatment: infections must be treated for a month or so (depending on their severity) using antibiotics chosen according to the bacterial sensitivity. Severe Methicillin resistant staphylococcus aureus (MRSA) requires treatment with Vancomycin and methoprim-sulfamethoxazole. Vancomycin usually kills the bacteria, whereas trimethoprim-sulfamethoxazole acts by inhibiting their ability to multiply.
RFR method: detects and eliminates the staphylococci.
The most frequent resonances are: 371-382, 402, 434 kHz
Its other frequency resonances are: 301, 303, 324-332, 336 kHz
The resonances of other pathogen species of the staphylococcus group are: 294, 308, 323-329, 345, 347, 367, 376-381, 388, 401-402, 421, 434, 448-453, 458, 463, 465, 482, 484, 486, 490-491, 504, 511, 517, 542, 552, 556-557, 563-568, 576 kHz

6.15.3.2. Staphylococcus Epidermidis

It is a coagulase-negative species, a commensal of the skin, causing disease only in case of immunosuppressed patients.
Its most frequent resonances are: 327, 329, 332, 372, 482 kHz

6.15.3.3. Staphylococcus Saprophyticus

This coagulase-negative species is part of the normal vaginal flora. The bacteria can cause genitourinary infections among sexually active young women.

6.16. The Order of the Lactobacillales

There are only two important human pathogenic genera belonging to this order of Gram-positive, lactic-acid bacteria group, such as the Enterococcus genus and the Streptococcus genus. The members of this lactic-acid bacteria group can convert lactose and other sugars into lactic acid. The species of the non-pathogenic Lactobacillus genus, also belonging to this order, are symbiotic members of the mucosal flora of the vagina and the gastrointestinal tract. The produced lactic acid makes its environment acidic inhibiting thus the growth of some harmful microbas.

6.16.1. The Enterococcus Genus

These Gram-positive cocci which often occur in pairs (diplococci) were formerly classified into the Streptococcus Group D. Two species of this genus are common commensal bacteria of the intestines of human beings, the *Enterococcus faecalis (formerly Streptococcus faecalis)* and the *Enterococcus faecium.* They are facultative anaerobic bacteria which can cause *urinary tract infections, bacteremia, endocarditis, diverticulitis and meningitis.* Enterococcal meningitis is a complication seldom met with in neurosurgery. These infections can lead to life-threatening states, especially in nosocomial enviroments, because of the high-level antibiotic resistance (beta-lactam-based antibiotics, aminoglycosids and vancomycin as well) of most Enterococcus strains.
The most frequent resonances are: 351, 374, 418 kHz

6.16.2. The Streptococcus Genus

The members of this Gram-positive, lactic acid group bacteria are responsible for many diseases among people, though many streptococcal species are non-pathogenic. Streptococci are also part of the normal mucosal flora of the mouth, the skin, the intestines and the upper respiratory tract of human beings. The individual species of *Streptococci* are according to their haemolytic properties classified into the Alpha haemolytic group (characterized by the reduction of iron in the haemoglobin), the Beta haemolytic group (characterized by a complete rupture of the red blood cells) and the Gamma haemolytic group (by which no haemolysis takes place).

The Beta-Haemolytic Streptococci are further characterized by Lancefield-serotyiping into Group A, B, C, D etc. of Streptococci. The most important species of pathogens concerning human beings are the Alfa-Haemolytic Streptococcus pneumoniae and the Alfa-Haemolytic Streptococcus viridans groups, the Beta-Haemolytic Streptococcus Group A and Group B, where as Streptococci Group C and D and non-haemolytic are pathogenic in case of animals.

6.16.2.1. The Alfa-Haemolytic Streptococcus Group

6.16.2.1.1. Streptococcus Pneumoniae (Pneumococcus)

This species is a significant human pathogen. It is the leading cause of *bacterial pneumonia* and many other types of infection as well, like *acute sinusitis, otitis media, meningitis, osteomyelitis, septic arthritis, endocarditis, pericarditis, peritonitis and cellulitis.* Streptococcus pneumoniae is one of the most common cause of otitis media infections and also the most common cause of certain bacterial meningitis cases among adults and children. Pneumococcal pneumonia may also follow secondary to a chronic bronchitis, or develop after a common respiratory viral (notably the influenza virus) infection, causing damage in the lining of the respiratory tract. Among people who live with a high risk of getting pneumococcal pneumonia are mostly those with chronic illnesses and compromised immune systems.

The general resonant frequencies of Streptococcus pneumoniae are: 320, 360-372, 397 kHz

Its other frequency resonances are: 290-294, 317, 320, 330, 340, 349-352, 363, 366, 368, 370, 372, 397, 410, 433, 466, 472-480, 515, 550, 567 kHz

6.16.2.1.2. The Streptococcus Viridans Group

The members of this group are Alfa-Haemolytic Streptococci and generally non-pathogenic, while some are commensals of the oral cavity of human beings. The *Streptococcus mutans* species can contribute to *tooth decay.* The *Streptococcus viridans* species can cause local infections such as *gingivitis* or *dental abscesses. Endocarditis* infection may only occur in cases the bacteria get into the bloodstream and if the infected patient has damaged heart valves.

The general resonant frequency range of the Streptococcus viridans group is: 542 kHz

Its other frequency resonances are: 310-311, 397-398, 435, 443, 455, 467, 476-478, 517 kHz

6.16.2.2. The Beta-Haemolytic Streptococcus Group

6.16.2.2.1. Streptococcus Pyogenes (Group A Streptococcus)

Streptococci pyogenes are the most virulent human pathogenic species. These bacteria usually cause *strep throat (streptococcal pharyngitis) and tonsillitis*, or, attaching wounds of the skin they can cause *impetigo*, a superficially spreading, localized bacterial skin infection. If the bacteria get into the deeper layers of the skin, they spread laterally, causing *erysipelas* and getting more deeper *cellulitis.* In case of its invasion by producing various virulence factors and in case of its multiplication in the fascia, developing there into *necrotizing fasciitis.* This infection can cause a life-threatening state. Many strains of Streptococcus pyogenes can secrete pyrogenic exotoxins (SpeA and C). These cause the rash of *scarlat* fever and some symptoms of the *streptococcal toxic shock syndrome* as well. Streptolysin O and S are the toxins of some strains, which are apt to poison the cells, Streptolysin O is likewise cardiotoxic.

The post infectious, autoimmune mediated complications of the streptococcus pyogenes infections are *rheumatic fever*, with *polyarthritis, carditis, chorea minor* and the acute poststreptococcal *glomerulonephritis*.

The general frequency resonances of Streptococcus pyogenes are: 320, 358-362, 363-375, 540 kHz

Its other frequency resonances are: 310, 315, 337, 340, 368, 372, 376, 402-403, 432, 450, 473 kHz

6.16.2.2.2. Streptococcus Agalactiae (Group B Streptococcus)

These streptococci are members of the normal mucosal flora of the gut and the female urogenital tract, which increase the risk of a premature rupture or a transmission to the fetus. The bacteria can cause dangerous infections in case of newborns, such as perinatal pneumonia, meningitis and sepsis entailing a high mortality rate. The symptoms are non-specific, like fever, vomiting, poor feeding and irritability.

6.16.2.2.3. Streptococcus Bovis (Group D Streptococcus)

These bacteria are usually found in the alimentary tract of ruminants, and can occasionally be pathogenic, causing endocarditis, neonatal sepsis and meningitis among human beings.

The Diagnosis of the streptococcal infections: is made by taking a throat swab and a culture revealing thereby the characteristic bacterial colonies.

Treatment: with penicillin or other antibiotics in case of resistance to antibiotic, administer analgetics, antipyretics and antiinflaamatory drogs.

RFR method: detects and may eliminate the streptococci.

The general frequency resonances of Streptococcus Beta-Haemolytic are: 325, 347, 376, 380-389 kHz

Its other frequency resonances are: 290, 307, 325, 342, 345, 353, 362-364, 376, 389, 450, 453, 482, 486, 524, 530, 547-548, 555 kHz

The general frequency resonances of Streptococcus mitis are: 313-322, 403, 506 kHz

The general frequency resonances of Streptococcus pneumoniae are: 320, 366-370, 397 kHz

Its other frequency resonances are: 290-291, 293-294, 317, 320, 330, 340, 349-352, 363, 366, 368, 370, 372, 397, 410, 433, 466, 473, 475, 515, 550, 567 kHz

The general frequency resonances of Streptococcus pyogenes are: 320, 358-362, 363-375, 540 kHz

Its other frequency resonances are: 297, 310, 315, 337, 340, 368, 372, 376, 402-403, 432, 450, 473 kHz

The general frequency resonance of Streptococcus viridans is: 542 kHz

Its other frequency resonances are: 310-311, 397-398, 435, 443, 455, 467, 476-478, 517 kHz

The frequency resonances of Streptococcus group G are: 293, 367-369, 466 kHz

The frequency resonances of Lactococcus lactis (Streptococcus lactis) are: 306, 488 kHz

6.17. The Class of Clostridia

There are only some few species of the genus being human pathogenic of this Class of Clostridia. The bacteria of the genus of Clostridium are obligate anaerobes capable of producing endospores. This genus includes common free-living bacteria as well as pathogens. The bacteria are able to produce a variety of toxins. Their infections can easily lead to death. A cultivated rich soil presents the highest density of Clostridia microorganisms. More over, clostridia may be present as commensals of the normal human colonic, skin and vaginal flora. The most common diseases caused by Clostridium bacteria are gas gangrene, food poisoning, the pseudomembranous colitis, botulism and tetanus.

Some clostridia species can very rarely infect human beings causing bacteremia and only in case of immunodeficiency, e.g. associated with malignancy.

6.17.1. Clostridium Perfringens

The Clostridium perfringens species are Gram-positive, anaerobic, spore-forming, rod-shaped bacilli, which are ubiquitous in nature. They are commensals of the soil, the decaying vegetation and can be found in the intestinal tract of insects, vertebrates and human beings as well.

Due to the various toxin productions of Clostridium perfringens, its infections usually show evidence of tissue necrosis. The most common diseases caused by these bacteria are clostridial myonecrosis (gas gangrene), clostridial food poisoning and clostridial necrotizing enteritis.

6.17.1.1. Clostridial Myonecrosis (Gas gangrene)

Clostridium perfringens is the most common cause of clostridial gas gangrene, being present in 80-90% of the cases. Some other clostridia species may include *Clostridium novyi*, *Clostridium septicum, Clostridium histolyticum, Clostridium bifermentans* and *Clostridium fallax*. Usually, more than one of their species is isolated from clinical specimens. The most important prerequisite for the conversion of clostridial contamination of a wound into a progressive infection is an environment with a low oxidation-reduction potential, which permits spore germinations and anaerobic growth. Infection does only occur if the organisms are inoculated into the tissues and the oxygen tension is low enough to allow the proliferation of the microbas. In these circumstances bacteria multiply and produce more tissue-destructive, soluble exotoxins leading to a rapid myonecrosis, systemic toxicity, shock and death. The development of this highly lethal, necrotizing, soft tissue infection of the skeletal muscle is caused by toxin- and gas-producing *Clostridium* species depending also on the host and the local wound factors.

A massive intravascular hemolysis caused by clostridial gas gangrene is rather rare. The risk of death is high, especially in case of infants, elderly people and those who underwent an immunosuppressive treatment.

Diagnosis: anaerobe bacterial culture. The sample must be carefully handled as its exposure to air might kill the anaerobic bacteria.

Differential diagnosis: gas gangrene must be differentiated from non-clostridial infections of gangrenous limbs caused by anaerobic streptococci, aerobic gas-forming coliform bacilli, Bacteroides species, and Streptococci group A.

Treatment: chiefly with antibiotics like penicillin, patients resistant to penicillin must be treated with other antibiotics.

The resonant frequencies of Clostridium perfringens are: 315, 502 kHz

The frequency resonances of its spore form are: 394-398 kHz

The resonant frequencies of Clostridium septicum are: 362-366, 461, 579 kHz

6.17.1.2. Clostridial Food Poisoning

Some strains of *C. perfringens* produce toxins causing food poisoning if ingested. This food-borne illness can mostly occur after eating poorly prepared and undercooked meat or poultry, the clostridial enterotoxin being heat-resistant. *C. perfringens type A* is a common cause of enterocolitis.

Symptoms: Following an incubation time of 8-16 hours after having ingested contaminated food, abdominal cramps and diarrhea will set in. The symptoms usually subside within 24 hours. Many cases of food-poisoning remain subclinical.

6.17.1.3. Clostridial Necrotizing Enteritis

This fatal and very rare disease is caused by a Beta-toxin producing strain Type C of the *C. perfringens*. The toxin has an ulcerative potency, causes inflammation and destroys the walls of the intestines.

The resonant frequencies of Clostridium perfringens are: 315, 502 kHz

The resonant frequencies of its spore form are: 394-398 kHz

6.17.2. Clostridium Difficile (Pseudomembranous colitis)

This species can be a normal commensal of the human intestines, its transmission can happen from person-to-person in the fecal-oral way. The bacteria can form heat-resistant spores, which will remain in hospital environment for a long time. If ingested, the spores survive the acidic substance of the stomach. Getting into the colon, the spores become active and multiply. These bacteria can cause severe colon infections called *pseudomembranous colitis*. The illness often *begins after an antibiotic treatment* with wide spectrum activity, having eradicated the normal gut flora. Clostridium difficile is resistant to most antibiotics, survives and overgrows.

Symptoms: The pathogenic strains produce enterotoxins and cytotoxins, causing *diarrhea, abdominal pain and inflammation* of the gut. *Sepsis* and *bowel thickening* with perforation may occur. The symptoms can vary from mild to life-threatening. An immunocompromised state a mistaken diagnosis or a delayed therapy can heighten the risk of causing death.

Treatment: by early intervention and an aggressive therapy (with Vancomycin, Metronidazole, linezolid)

The resonant frequencies of Clostridium difficile are: 325, 344, 396 kHz

6.17.3. Clostridium Botulinum (Botulism)

This rod-shaped, anaerobic, endospore-forming Gram-positive bacterium is commonly found in the soil *(Types A and B)* in fish *(Type E)* in honey, in corn syrup, the spores, however, cannot grow in a highly concentrated sugar solution. Its serotypes *(from A-G)* produce different botulinum toxins, though Type C and D are not human pathogens. Some C. botulinum strains do not produce botulin toxins, referred to as *Clostridium sporogenes*. The pathogenicity of these clostridia is owing to their capacity to form botulin. The botulin toxin is a powerful neurotoxin, which blocks the nerve functions causing thus respiratory and musculosceletal paralysis.

Infant botulism can occur by swallowing the spores of the bacteria which then increase in the intestines of the infant and then releasing toxins.

Food-borne botulism happens by eating food contaminated with the rather rare botulinum toxin.

Wound botulism caused by the toxins produced by *Type F* botulism among adults is caused by the neurotoxigenic *Clostridium baratii* coming from an unknown source. All forms of botulism can be dangerous being a public health risk due to a contaminated food source.

The Symptoms: of the food-borne botulism occur 12-36 hours after eating botulin-contaminated food. Dry mouth, doubble vision, difficulties in swallowing and breathing, paralytic ileus, and body paralysis can develop. In case of a respiratory muscles paralysis the failure can even lead to death. Wound butulism has an incubation period of 4-14 days. Infant botulism occurs generally among infants under 6 months through the colonization by clostridial spores in the gut. Its symptoms are constipation followed by general weakness and symmetric flaccid paralysis. The illness can be often fatal due to respiratory failure.

The resonant frequencies of Clostridium botulinum are: 360-365, 458, 576 kHz

6.17.4. Clostridium Sordellii

This bacterium is a rare anaerobic, Gram-positive, spore-forming rod which can cause pneumonia, endocarditis, peritonitis and myonecrosis. Among patients in immunosuppressed condition the infection can lead to bacteremia, causing sepsis. This bacterium, associated with some gynecological infections can cause a severe toxic shock syndrome, so also in case of newborns if associated with some umbilical infection.

The symptoms appear with a marked leukocytosis hypotension, tachycardia, haemoconcentration and capillary leak syndromes A toxic shock syndrome caused by this infection proves to be usually fatal.

RFR method: detects and eliminates these anaerobic bacteria.

The resonant frequencies of Clostridium sordellii are: 317-325, 364-368, 461 kHz

The list is not complete yet, as some subspecies have different frequencies not yet identified.

6.17.5. Clostridium Tetani (Tetanus, Lockjaw)

Tetanus is a disease caused by toxins produced by the Gram-positive, spore-forming, anaerobic bacterium named Clostridium tetani. It is found as spores in the soil or as bacteria in the gastrointestinal tract of animals. Its toxins are the largest known poisons, named tetanospasmin and tetanolysin. Being a neurotoxin, it is the tetanotoxin and not the actual bacteriae, which cause the symptoms of the infection. The spores of Clostridium tetani can live for years in the soil and in the animal faeces. Once the tetanus bacterium or spore gains entry through a wound into an anaerobic locus of the cutan or subcutan tissue, the illness is able to develop. The tetanus toxin is generated by living bacteria, is released during the spore germination or during its vegetative growth.

The symptoms usually appear five days after infection. The most common symptom is appearing the jaw stiffness. Other symptoms include restlessness, difficulty in swallowing, irritability, headache, fever, sore throat, chills, muscle spasms and stiffness in the neck, arms and legs. The spasms of the facial muscles cause a fixed smile and raised eyebrows. The rigidity or the spasm of the abdominal muscles, of the muscles of the neck and the back can cause a distinctive posture in which the head and heels are pulled backward while the body is arched forward. Spasms of the muscular sphincters of the lower abdomen can lead to constipation and urine retention. In case of a spasm of the whole body, a person cannot cry out nor speak because of the rigidity of his chest muscle or owing to throat spasms, which might even cause oxygen deprivation or fatal suffocation. The mortality rate of the disease is about 50%.

Prevention: by vaccination. In most countries the tetanus vaccine (toxoid) is ordered to be given to children in series combined with diphtheria and pertussis vaccine (DPT vaccine).

Treatment: in case of a suspected infection, the person who had never been vaccinated or had never received the complete series of vaccinations, needs the administration of tetanus immune globulin (antitoxin) and, having been stabilized, active vaccination has to be given him too. Tetanus immune globulin is given for the neutralizing the toxin, while large doses of antibiotics, (such as im. Penicillin G, Metronidazole or Doxycyclin) should be given him in order to prevent a further toxin production.

RFR method: is only employed as a secondary measure, following the neutralizing of toxins.

The frequency resonances of Clostridium acetobutylicum are: 305, 382-392, 487 kHz
The frequency resonances of Clostridium botulinum are: 360-365, 458, 576 kHz
The frequency resonances of Clostridium difficile are: 325, 344, 396 kHz
The frequency resonances of Clostridium perfringens are: 315, 502 kHz
The frequency resonances of its spore form are: 394-398 kHz
The frequency resonances of Clostridium septicum are: 362-366, 461, 579 kHz

The most frequent resonances of Clostridium tetani are: 292, 307, 321, 358, 360-372, 402, 409, 450, 468-475, 499, 504, 566-567 kHz
This list is not yet complete.

6.17.6. The Peptococcus and the Peptostreptococcus Genus

The species of this Gram-positive, anaerobic, coccoid Peptococcus genus are *commensals of the normal flora of the mouth, the upper respiratory tract and the large intestine* of human beings causing infections of soft tissues or bacteremia solely in case of immunocompromised patients. The species of the Peptostreptococcus genus are also living in the mouth, the gastrointestinal tract moreover even in the skin, the urinary tract as normal inhabitants. In case of immunosuppressed or traumatic people these bacteria can cause septicaemia, abscesses in the brain, the liver and the lung as well as necrotizing infection of the soft tissue in every part of the body in immunosuppressed or traumatic condition.
The frequency resonances of Peptostreptococcus are: 322, 411 kHz

The Class of Mollicutes

This is an unusual group of bacteria characterized by the absence of a cell wall. The members of this class are commonly named mycoplasma. Those certain species which cause diseases among humans by entering cells of the respiratory or urogenital tracts or the skin include the species of Mycoplasma, Ureaplasma and Erysipelothrix. The order of the *Mycoplasmatales* contains one single family, the *Mycoplasmataceae*, which again contains two genera: the *Mycoplasma* and the *Ureaplasma.*

6.18. The Order of the Mycoplasmatales

Mycoplasmatales are one of the most mysterious pathogen microorganisms, being the smallest bacteria-like organisms lacking cell walls and capable of self-replication and able to cause various diseases among humans, animals and plants. There are about 200 species of mycoplasmatales, though only few of them are known to be human pathogens as yet. They can invade tissue cells, incorporating the nutrients of the host's cells and using the cell for their own replication (like the retroviruses). Their cell-membrane incorporates sterol compounds, requiring cholesterol for their growth.

6.18.1. The Mycoplasma Genus

All members of the Mycoplasma genus have vertebrate hosts. When Mycoplasmas break out of the host's cells, they take a piece of the host-cell membranes with them. In that way, the attack of the host's immune-system against the Mycoplasmas, will „turn on" the host cells too. Severe mycoplasmal infections may even destroy cell-lines. Mycoplasmas may induce also cellular changes, including chromosoma abortations, changes in the metabolism and in the cell-growth.

6.18.1.1. Mycoplasma Pneumoniae Group

These species have one of the smallest genomes known and are obligate parasites. These strains are usually the cause of primary atypical pneumoniae (or socalled walking pneumoniae) and other airway disorders, such as tracheobronchitis and pharyngitis spreading through by respiratory droplet transmission. Mycoplasma species attach themselves to the mucosa of the respiratory tract causing usually a mild ot moderate respiratory tract infection.
The symptoms are atypical and slowly progressing *lacking sputum production* but show a *wealth of extra pulmonary symptoms* such as rheumatological disorders, liver inflammation, pancreas and cardiovascular syndromes. The infection can be complicated

by haemolytic anaemia, nervous system inflammations (such as encephalitis, Guillain-Barrè syndrome) and allergic reactions, f.i. Stevens-Johnson syndrome.
The currently unculturable haemotrophic mollicutes (haemoplasmas) *Eperythrozoon* and *Haemobartonella* species are sorted nowadays also into this group and cause anaemia etc. (See Chapter 19.2.2.)
The resonant frequencies of Mycoplasma pneumoniae are: 321-324, 337-344, 346-350, 352, 363-364, 397, 499 kHz
The resonant frequencies of Mycoplasma pulmonis are: 307-308 kHz (zoonozis from castle).
The most frequent resonances found in case of Eperythrozoon anemia are: 364-367, 378-386, 397-390, 478, 480-486, 548, 558-566 kHz
The most frequent resonances of Haemobartonella felis are: 308, 487 kHz

6.18.1.2. Mycoplasma Genitalium

This species is the smallest known free-living bacterium. The infection caused by *M. genitalium* can be transmitted from partner-to-partner by unprotected sexual intercourse. The infection often remains asymptomatic or causes non-gonococcal urethritis or other urogenital inflammatory symptoms among men. In case of women, the mycoplasmal infection is generally found associated with bacterial vaginosis, cervicitis, pelvic inflammatory disease, endometriosis and premature birth.
The resonant frequencies of Mycoplasma genitalium are: 307-308, 342-350 kHz

6.18.1.3. Mycoplasma Salivarium

This species may play an etiological role of periodontal diseases facilitating the accumulation and retention of inflammatory cells in gingival connective tissue. It can be the causative agent of oral infections, periodontal diseases, eye and ear disorders and even of septic arthritis.
The resonant frequencies of Mycoplasma salivarium are: 387-389, 425-430, 461-463, 518, 570-572 kHz

6.18.1.4. Mycoplasma Fermentans, M. Pirum, M. Hominis and M. Penetrans

All these Mycoplasma species are supposed to be human pathogens and possible cofactors in case of HIV infections. These bacteria may contribute to the clinical variations occuring within the time from being infected with HIV to the development of the AIDS symptoms. Mycoplasma fermentans can be found in the infected human mucosal tissues and in the saliva as well. Although mycoplasmas are primarily known to be extracellular parasites or pathogens of mucosal surfaces, recently found cases show that certain species are able to invade host cells. They can overcome the blood/brain barrier, causing brain and spinal infections and can cross the placental barrier infecting the fetus. Mycoplasmal organisms can be found in the blood, the body-fluids, the spinal fluid, the bone marrow, the urine, the lungs, in the nose and mouth, which fluids can infect other persons.
The molecular and cellular base for the invasion of M. fermentans into the blood-stream are the mucosal cells. The invasion of the host's blood-cells by M. fermentans is due to the inhibition of phagocytosis by antiphagocytic proteins (such as proteases, phospholipases) as well as owing to oxygen radicals produced by the mycoplasma. Mycoplasma fermentans bacteria are able to fuse with CD4+T lymphocytes and thus change their manner of the cytokine production.
Mycoplasma fermentans can be found as the causing co-factor of almost every chronic syndrom, f.i. the diverse tumours, autoimmune (SLE, scleroderma) and other collagen-vascular diseases, chronic/rheumatoid arthritis, psoriatic arthritis, spondyloarthropathies and fibromyalgia syndromes, Crohn's disease, Diabetes type 1, sarcoidosis, Multiple

Sclerosis, Parkinson's disease, Wegener's granulomatosis, AIDS and systemic neurodegenerative syndromes, such as Alzheimer's disease. In case of those illnesses, in which a Mycoplasma fermentans infection is combined with other infections (such as HPV, HTLV, HBLV, HIV, and/or several other microorganisms) the pathological effect of these microorganisms will be increase. Mycoplasma fermentans can co-infect also with Borrelia Burgdorferi sensu lato, Chlamydia, Babesia, Ehrlichia, Coxiella and Bartonella species. The infections caused by Mycoplasma and Borrelia species by changing their behavior, may cause various severe neurosystemic syndromes. The inhibition of phagocytosis and a pure immune response to these microorganism is due to the invasion of M. fermentans of such infections. The absence of the DNA of M. fermentans in the blood-cells with the simultaneous presence of antibodies produced against of the mycoplasma in the serum of the same patient, suggests thus a chronic Mycoplasma fermentans infection of other tissues or cells. Another explanation could be the cross reactivity. This means that the antibodies, produced against collagen, cartilages, and thyreoid cells of some patients suffering from an autoimmune disease, may cross-react with mycoplasmal antigens giving thus a false-positive result. Mycoplasma bacteria are able to inhibit the adequate, effective immune response of the host. This damage of the immune system happens owing to the invasion of the Natural Killer cells (NK cells) by weakening them, reducing their number, and by rendering them susceptible to viral infections, (such as f.i. Human Herpes Virus-6 (HHV6), HHV7 or HHV8. The mycoplasmal infection can trigger the overproduction of inflammatory cytokines commonly experienced in case of CFS/FMS (chronic fatigue syndrome, fibromyalgia syndrome). Mycoplasma bacteria can induce CD4+(helper) T cells resulting in an overproduction of cytokines (Interleukin-1, Interleukin-6 and Tumor Necrosis Factor-alpha). These elevated cytokines play a role in the development of many CFS/FMS symptoms, including a neurological involvement as well. These cytokines have specific and nonspecific, stimulative and suppressive effects on the activation of the B and T cells. In addition, the mycoplasmal infection has immunomodulating effects too, by activating the hypothalmic-pituitary-adrenal axis. This can cause a cascade of symptoms of the limbic system, characteristic of the CFS/FMS. The Lou Gehrig's disease (ALS) and the autoimmune diseases, including lupus erythematodes, Hashimoto thyreoiditis, sclerosis multiplex, etc. are also associated with mycoplasmal infections, which can penetrate into both the peripheral and the central nervous system.

Diagnosis: by using the polymerase chain reaction (PCR) method of blood or tissue samples. Mycoplasma fermentans can act as co-factors in case of most chronic syndromes mentioned above, in that way the clinical PCR testing can be positive of other co-factors too, such as of the human herpes viruses, particularly of the Epstein-Barr Virus (EBV) and the Human Herpes Viruses 6, 7 and 8 (HHV6, HHV7, HHV8). The Human T-cell Lymphotropic Virus (HTLV) type 1 and 2, the foamy or Spuma virus and the Chlamydia pneumoniae were also most recently described as co-factors of the Mycoplasma.

Treatment: the total elimination of the mycoplasma concerning severe cases is very difficult and seldom feasible. Especially the Minocyclin, Doxycyclin, the second generation quinolones, such as Lefloxacin, and sometimes the second generation macrolide antibiotics proved to be effective antibiotics.

If diagnosed and treated in time, diseases associated with invasive mycoplasmal infections are curable by administering effective high-dose antibiotics for a long time, to be repeted if needed. Since the microorganism is a slow-growing, intracellular type of bacterium with a long life-cycle, a treatment with several, long-term courses of antibiotics might be required. The infection may need a treatment for several months or even years. The disease is treated in the same way as the Lyme disease. As long as a person is taking antibiotics, the PCR testing will not detect the presence of Mycoplasma in the blood. In order to render the PCR test accurate, (after stopping administration of antibiotics) one must wait for at least two months.

Prevention: an adequate vaccine is not developed yet.
RFR method: detects and may eliminate the mycoplasma.
The resonant frequencies of Mycoplasma fermentans are: 442-451, 493-495 kHz
The resonant frequencies of Mycoplasma pirum, M. hominis and M. penetrans are: 311-313, 329, 352-354, 361, 384-394, 404-405, 440-442, 464, 491-504, 520-521 kHz
The other frequencies of non-identifiable Mycoplasma species are: 339-341, 377-378, 359-362, 399-400, 424, 432-433, 439, 470-472, 492, 508, 515, 520-521, 524-526, 535, 543-546, 551, 569, 587 kHz
In case of co-infections, characterized by the above mentioned chronic syndromes, the Mycoplasma species is the first among the various microorganisms to be eliminated. It can be concluded, that the mycoplasmal infections are important factors or co-factors of a variety of chronic illnesses and syndromes. The infection of a certain Mycoplasma species used to be the cause of many a sign and symptom of CFS/FMS, of the dysregulation of the immune system and the abnormal autoimmune findings as well. This organism is worth testing, and if found positive, it has to be treated with the antibiotics recommended.

6.18.2. The Ureaplasma Genus

The most known species of this genus is the Ureaplasma urealyticum. It belongs to the normal genital flora of both men and women, but can also cause non-specific urethritis, infertility, stillbirth, premature birth and even pneumonia or meningitis among newbornss.
The most frequent resonances are: 386-388 kHz

6.18.3. The Erysipelothrix Genus (Erysipelothricosis)

The *Erysipelothrix rhusiopathiae* is a species of the class of Mollicutes, which is primerily an animal pathogen distributed worldwide, causing eysipeloid, infecting human beings. The disease, named Erysipelothricosis, presents a mild cutaneous-subcutaneous infection among people handling raw meat or fish. The infection happens by abrasion of the hand and develops but slowly. A raised, purplish-red, infiltrated area appears on the skin at the spot of the injury. Other symptoms may include itching, burning, and swelling around the affected area. Although the infection heals generally even without treatment, pain and disability may remain for about three weeks. In rare cases, the infection can spread into the bloodstream and affect the joints or the heart valves.
Treatment by administering Penicillin or Erythromycin
The most frequent resonances are: 315, 340, 372, 397, 402, 432, 450 kHz
The list is not complete yet.

The Phylum of the Bacteroidetes

There are only three genera of this phylum causing rare *opportunistic infections* among human beings: i.e. the Bacteroides genus, the Porphyromonas genus and the Prevotella genus. They are all Gram-negative, non-endospore forming, anaerobic bacteria and commensals of the gastrointestinal flora playing a role in the processing of complex molecules into simpler ones as well as hindering potencial pathogens from colonizing the mucosa.
The *Bacteroides fragilis* species are opportunistic pathogens of the gut, which can mostly cause *peritonitis and abscessive appendicitis.*
The *Porphyromonas gingivalis* species are inhabitants of the oral cavity, causing *gingivitis*, tooth decay and other *periodontal infections* in case of periodontal lesions.
The *Prevotella* species are commensals of the gastrointestinal tract of mammalians as well of human beings colonizing the mouth and causing severe *periodontal infections and tooth diseases* in areas previously infected by some other bacteria.

The most frequent resonances of Bacteroides fragilis are: 323-327, 412-413, 517-519 kHz
The most frequent resonances of Porphyromonas gingivalis are: 371-372, 396-398, 410, 450, 476 kHz

The Phylum of the Fusobacteria

The species belonging to this phylum are anaerobic Gram negative bacteria. They are commonly present in the oropharyngx of animals and human beings. They should always be eliminated as being pathogens.
Fusobacterium necrophorum species can infect human beings and cause recurrent *sore throat,* and in complicated cases *meningitis, thrombosis of the cerebral veins* and *urogenital and gastrointestinal infections* as well.
Certain other *Fusobacterium species* can play a role *in periodontal disease.*
The Streptobacillus moniliformis species are facultatively anaerobe bacteria which can cause a form of **rat-bite fever** occuring usually in the USA. The disease is caused by rodent bite (mostly rats) or by ingestion of contaminated food or water (Haverhill fever). The symptoms start with *fever, headache, vomiting, muscle pain,* followed by *rash and arthritis*. Having no specific character, the illness is diagnosed mostly as FUO (fever of unknown origin). Complications may develop as well as *endocarditis and bacteremia* leading even to death.
Diagnosis: by examination of the blood using dark-field microscop, Wright-stained smears; and by making serologic tests.
Differential diagnosis: Borrelia recurrentis infection, malaria, meningo-coccemia etc.
Treatment: with antibiotics (f.i. penicillin, macrolides, metronidazole)
RFR method: detects and may eliminate the bacteria.
The most frequent resonances of Fusobacterium necrophorum are: 320, 339, 360, 381, 393, 399, 407 kHz
This list is not complete yet.

The Phylum of the Spirochaetes

The species of this phylum are corkscrew-shaped Gram-negative bacteria moving by way of undulating, propeller-like motions of their flagella. When reproducing, a spirochete will undergo an asexual transverse binary fission. The species, pathogenic among human beings belong to two families: the Spirochetaceae and the Leptospiraceae. The human pathogenic members of the Spirochetaceae family can be found among the species of the Treponema genus and the Borrelia genus.

6.19. The Treponema Genus

The human pathogenic species of this genus is the Treponema pallidum having at least four subspecies: i.e. the *Treponema pallidum pallidum* which causes syphilis, the *Treponema pallidum pertenue* which causes yaws, the *Treponema pallidum carateum* which causes pinta and the *Treponema pallidum endemicum* which causes bejel. These subspecies are indistinguishable from each other differing only in their transmission and in the course of each illness.

6.19.1. Treponematosis

Treponematosis is the name of the group of nonvenereal diseases caused by Treponema pallidum species. Bacteria are transmitted by direct contact, invading the traumatized cutaneous or mucosal surfaces of children living in tropical or subtropical countries. The infection spreads in the body either topically or a haematogenous way. The disease may

cease, recure or persist and can be characterized by multiple cutaneous lesions as well as by the destruction of cartilage and bones.

6.19.1.1 Bejel - Endemic Syphilis (Treponema p. endemicum)

This non-venereal illness is similar to syphilis. The transmission of the bacteria occurs by contact with an infected lesion. The primary lesions are painless ulcers within the oral cavity. The secondary lesions are usually disseminated macules and papules. The tercier stage leads to bone deformities and to the destruction of the nasopharyngeal cartilages. There often occur ocular inflammations causing uveitis, optic atrophy or chorioretinitis.

6.19.1.2. Pinta (Treponema p. carateum)

This rare spirochetal infection caused by Treponema pallidum carateum is common among young adults living in the Caribbean and in Central or South America. The primary lesions of this illness are hyperkeratotic blue papules, associated with swollen lymphnodes nearby. These papules are persisting or healing hypopigmentatedly. Other parts of the body are not involved in this disease.

6.19.1.3. Yaws (Treponema p. pertenue)

In this disease the incubation time lasts longer than two weeks. The primary symptom is a large papilloma persisting for several months and healing with scarring. The secondary stage is characterized by maculo-papular rashes and swollen lymphnodes. If not treated, a long lasting periosteal infection will develop, wich can lead to bone destructions causing deformities and gummas of the sternum, the tibia or other bones.

6.19.2. Syphilis (Lues) (Treponema pallidum pallidum)

Syphilis is a sexually-transmitted disease caused by Treponema pallidum pallidum. This bacterium enters the body via sexual intercourse through the mucous membrane of the vagina, the mouth, or the skin of the penis. From there bacteria infect the regional lymph nodes, and then spread throughout the whole body via the blood. In case of a later stage of pregnancy the infection can also be transmitted into the fetus in a transplacental way causing connatal syphilis. The symptoms depend on the stadium (primary, secondary, and tertiery) of the disease. Without any treatment the infection may persist for years and progress, causing damages of the heart, aorta, liver, brain, eyes and even death.

The symptoms of the primary stage usually begin one week after being infected via close sexual contact with a person suffering with syphilis. A painless sore or ulcer appears at the spot of the infection: on the penis, vulva, or vagina. This primer lesion, the socalled chancre, may also appear on the anus, rectum, lips, tongue, throat, cervix, or fingers. The nearby lymph nodes usually become painless enlarged. The lesion may persist for 4-6 weeks and then disappear spontaneously.

The secondary stage begins 1-6 months after the primery infection and can be without any symptoms, but it can also start with maculopapular, disseminated, non-itching rashes of the whole body, affecting the palmar and plantar skin, the mucosa of the mouth and the genitalia. In the genital, moist region the papules are flat, broad and whitish named condyloma latum. These lesions are infectious, harbouring living spirochetes. The rash may be short-lived or lasting for months. Even if not treated, the rash usually clears up after a while. Other symptoms of this stadium include headache, fever malaise, generalized, enlarged lymphnodes, meningismus. If there occurs an inflammation of the eyes, optic neuritis, interstitial keratitis, iritis, uveitis may develop and sometimes even a mild inflammation of the kidneys or the liver, seldom resulting in proteinuria or jaundice. A syphilitic meningitis causes headache, neck stiffness, and sometimes deafness. Cranial nerve abnormalities mostly with facial, optic or vestibulocochlear nerve involvement may also result. The hair may fall out in patches, leaving a moth-eaten appearance. These

extremely infectious areas may flatten and become dull pink or gray. Other symptoms as nausea, fatigue, fever, anemia, a gastic crisis and proctitis may also come about.
Latent syphilis is characterized by having a serologic proof of the infection without showing any signs or symptoms of the disease. The latent syphilis can be either early or late. The early latent one is defined as having syphilis for two years or less from the time of the initial infection without any signs or symptoms of the disease. People in this condition are very infectious. In case of the late latent syphilis the infection is lasting longer than two years without any clinical evidence of the illness.
The tertiary stage of an untreated syphilis is characterized by any one of the following three main types of symptoms: benign tertiary, cardiovascular and neurosyphilis. The benign tertiary syphilis is characterized by formations of gummas (i.e. soft tumour-like chronic granulomas) of the skin and of various other organs. The bones may be affected too, resulting in a deep, penetrating pain that usually worsens at night. In case of a cardiovascular syphilis aortitis, an aneurysm of the aorta or an aortic valve insufficiency may be present. There are four late forms of neurosyphilis: i.e. the asymptomatic, the meningovascular, the paretic and the tabetic neurosyphilis. The meningovascular, the tabetic and the paretic symptoms include paresthesias, unilateral numbness, vertigo, insomnia, weakness of the upper and lower extremities, headache, personality changes, general paresis, chronic dementia, psychosis, depression and mania as well.
The **Definitive diagnosis** is based on the darkfield microscopy, the result of laboratory tests and physical examinations. Screening tests: VDRL and RPR. The more specific antibody test, the FTA-ABS test is used to confirm a positive screening test.
Treatment: with penicillin, given by im or iv.injection depending on stage in question. People who are allergic to penicillin may be administered with Doxycyclin, cephalosporine etc.
RFR method: is advised but only after the antibiotic treatment.
The resonant frequencies are: 307-320, 332-338, 345-355, 362, 404, 422, 440, 460-461, 552-555 kHz

6.20. The Borrelia Genus

The species of this genus can cause zoonotic, vector-borne spirochetal diseases among humans. There are about 40 known species of the Borrelia genus. The species of *Borrelia recurrentis* cause relapsing fever transmitted by lice. The species of *Borrelia hermsii, Borrelia turicatae, Borrelia Parkeri* and *Borrelia duttoni* cause tick-borne relapsing fever diseases. The species of *Borrelia Burgdorferi sensu lato* spirochetes are the causative agents of borreliosis (otherwise named Lyme disease, Lyme borreliosis).
Relapsing fever diseases and the Lyme borreliosis refers to a group of acute and chronic infectious diseases which are clinically characterized by cyclic periods of the symptoms.
Regardless of the way of transmission, the *relapse phenomenon* is caused by the genetically programmed shifting of outer surface proteins (Osp-s) of the Borrelia which will be thus followed by a new Borrelia clone avoiding its destruction by antibodies directed against the Osp-s of the originally infecting microorganisms. The symptoms cease completely until the new clone multiplies sufficiently to cause another relapse. The tick-borne disease tends to have more (3-10) while the more severe louse-born variety has less (1-2) relapses. The season of the onset and the region suggesting possible infected tick exposure can be important clues of the diagnosis.

6.20.1. Relapsing Fever (Borrelia recurrentis)

Borrelia recurrentis, the causative agent of the rare relapsing fever, is transmitted from person-to-person by feeding infected human body lice *(Pediculus humanus)*, having no animal reservoir. The infection happens via an abraded or intact skin or mucous membranes, following which the bacteria will invade the bloodstream. Relapsing fever is

an acute febril illness usually occuring following breakdowns in the public health (f.i. war, poverty and overcrowding, large refugee camps) most common in Asia, Africa, Central and South America.

The Symptoms begin after an incubation period of 3-18 days. The developed spirochetemia abruptly causes tachypnoe, tachycardia, fever and hypotension. A dry cough, nausea, vomiting, abdominal tenderness with hepatosplenomegaly, petechial rash, nuchal rigidity, lymphadenopathy and iritis-iridocyclitis are common. Neurologic findings including coma, cranial neuropathy (f.i. Bell palsy), hemiplegia, meningitis and seizures can also come about.

The high fever lasts for 3-6 days and then abates spontaneously causing a crisis that can culminate in fatal shock. The fever tends to recur abruptly about 7-10 days later. Relapses are less severe.

The mortality rate of untreated patients during epidemics of the louse-borne relapsing fever is 30-70% which can fall to about 5% by treatment.

The most frequent resonances of Relapsing fever are as follows: 308-310, 314-315, 344-346, 352-353, 372-375, 388-389, 408-410, 439-442, 452-453, 471-472, 516-517, 520, 524-525, 537-538, 545-546 kHz

6.20.2. Tick-borne Relapsing Fever (Borrelia parkeri, B. hermsii, B. turicatae, B. duttoni)

Soft bodied ticks *(Ornithodoros)* spread the also rare tick-borne relapsing fever which can occur in Spain, Africa, Saudi Arabia, Asia, certain areas in the Western United States and Canada. Soft ticks, for which rodents and other animals are the principal reservoirs, feed for but short periods of time (an hour or so), so that the *Borrelia* bacteria are inoculated into the skin within minutes. The clinical picture of the illness is similar to that of the louse borne variant, but usually milder, causing abrupt fever, headache, tachypnoe, tachycardia, myalgia and chills. Arthalgia, weakness, weight loss and cough is common. In case of patients suffering from tick-borne fever disease with repeated relapses may develop iritis or iridocyclitis with permanent visual impairment. Pregnant women suffering from relapsing fever often abort. The mortality rate of treated patients suffering from tick-borne relapsing fever is less than 1%. Relapses can recur even more often than 10 times.

The Diagnosis: may be established by visualizing spirochetes in multiple smears (both thick and thin, using Wright and Giemsa stains) of peripheral blood during a febrile episode and as well as by antibody tests and PCR examinations.

Treatment: with antibiotics such as Doxycycline and Chloramphenicol which are most effective, but may nevertheless induce a Jarisch-Herxheimer reaction in more than 65% of the patients. (JH reaction produces apprehension, diaphoresis, fever, tachycardia, and tachypnea with an initial hypertension-response followed rapidly by hypotension. Tumor necrosis factor-alpha (TNF-alpha) is partly responsible for this reaction.

RFR method: detects and may eliminate these microorganisms.

The resonant frequencies of B. hermsii are: 378-383 kHz

The resonant frequencies of B. parkeri are: 377-382 kHz

This list is not complete, there are other subspecies and plasmids having some other resonances. Use RFR method and administer antibiotics together.

6.20.3. Borreliosis (Borrelia Burgdorferi sensu lato)

Borreliosis is the common name of emerging infectious diseases caused by certain species of the Borrelia Burgdorferi sensu lato (B.B.s.l.) group. The Borrelia species, known to cause Lyme disease, have greater strain diversity than previously estimated. There are three genospecies known to cause Lyme disease in most of the cases: i.e. *Borrelia Burgdorferi sensu stricto* (occuring predominantly in North America, but also in West-Europe), while *Borrelia afzelii* and *Borrelia garinii* (are both prevailing in Eurasia). The

newly discovered genospecies however (i.e. *Borrelia valeisiana, Borrelia lusitaniae, etc.*) have also been found to cause diseases infecting human beings. It is not yet known how many of the worldwide found Borrelia strains can cause lyme disease.
Borrelia species causing borreliosis are transmitted to humans mostly by hard-bodied ticks of the genus *Ixodes* (i.e. *I. ricinus* in Europe and *I. dammini et scapularis* in North America). While Lyme spirochetes have also been found in insects other than ticks (fleas, mosquitoes and mite), their actual infective role cencerning the transmission appears to be rare. Insects get borrelia into their midgut by feeding on infected small rodents, cats, dogs, deer, raccoons, squirrels, chipmunks, birds etc. The transmission by sexual intercourse of the disease is a compelling evidence. Connatal transmission of the Lyme bacteria occurs from an infected mother to the fetus via the placenta during pregnancy. Some data indicate that B.B.s.l. are able to survive and be transmitted by blood transfusion procedures. The borreliosis is one of the fastest growing epidemic in the world. The early detection of the disease is difficult as by about 20-40% of infected people the tick went unnoticed, and about 30-50% of infected people have no characteristic rash (about or more than 2 inches i.e. 5 cm in diameter). B.B.s.l. bacteria enter the skin by tick bite. The dissemination of the pathogen occurs via the bloodstream. B.B.s.l. species were recovered in every part of the body, the brain, joint, heart, liver, spleen, skin etc. of infected people.
The Lyme disease symptoms, can be sorted into three stages. *Stage I.* (i.e. the early localized stage) is the short time after being bitten by an infected tick, which is defined by the local presence of borrelia in the skin with or without the characteristic rash named erythema migrans and is found sometimes with flu-like symptoms. The *second stage (i.e. the early disseminated stage)* is the time of the early dissemination of the bacteria via the lymphatics and the bloodstream. At that stage there appear, besides tiredness, certain symptoms of the skin, the joints, the muscles or/and the central or/and the peripheral nervous system, moreover there will generally develop even psychiatric symptoms as panick attacks, depression etc. Infected people feel ill and get more tired, suffering characteristically from headache, stiff neck, muscle and joint pains. Backache, nausea and vomiting, sore throat, swollen lymph nodes, lymphadenosis benigna cutis and an enlarged spleen can occur. In some cases multiple erythema migrans is going to appear. These early symptoms will appear after being bitten by tick within some days or within a few months. Although most symptoms come and go, the ill feeling and fatigue will persist. Some people, found positive concerning the pathogen, remain asymptomatic for a long time. If not treated effectively as early as possible, the *third stage*, (i.e. the late and chronic form of the disease) will develop, which is difficult to treat and cure. The symptoms can vary, depending on the species of the B.B.s.l. and on other co-infections of the person affected. Swelling and pain in the large joints, especially the knees, are the leading symptoms among III. stage patients in the USA. In Europe neurological symptoms and acrodermatitis chronica atrophicans are more typical. Chronic borreliosis infection is affecting simultaneously many organs of the body: f.i. the peripheral nervous system and/or the central nervous system (neuritis retrobulbaris n.optici, n. glossopharyngei, n. acustici, n. vestibularis, n. olfactorii, facial paresis, radiculomyelitis, peripheral neuropathy, encephalitis, encephalomyelitis, muscle twitching, polyneuropathy, paraesthesia), as well as the joints (arthritis, arthalgia, getting Baker cysts) and the connective tissues (panniculitis, entesitis, tendinitis, etc.) and the urogenital tract (prostatitis, interstitial cystitis, infertilitas, abortus spontaneus, etc.) so also the heart (irregular heartbeats, arrhythmia, pericarditis, endocarditis) and the gastrointestinal tract too (mild hepatitis, obstipation), these are all the most often affected organs.
Psychiatric disorders (such as panic attacks, depression, changes in mood or affect etc.) are very common. Patients feel fatigue, their attention slackens, they suffer memory loss, which symptoms all indicate chronic low-level encephalitis-encephalopathia. The symptoms may wax and wane over hours or days, varying in intensity and persistence

recurring and worsening even throughout many years. Lyme borreliosis being a multisystemic disease will be often poorly managed owing to the fragmentation of health care system. Misdiagnosed and/or mistreated Lyme disease patients may, instead of having been treated in time with adequate antibiotics, get pacemaker or/and a hip joint prothesis and/or a knee joint prothesis.

Special characteristics of the borreliosis: Lyme bacteria are causing particular immunological processes.

The Borrelia bacterium has a ring-like cell-wall, holding its specific outer surface protein (Osp) antigens. By the time the borrelia attacks the host's tissue, the host's immune system will produce a specific antibody against this Osp-antigen of the bacterium. The Osp-antigen attached to the cell-wall binds the antibody of the host. Following this, the Borrelia emerges from its ring-like cell wall, as if it were a coat, so that the antigen-antibody complex will thus remain in the host's tissue recognized and attacked newly by the immune system, even though the Borrelia is not present any more. B.B.s.l. is able to survive and spread even without having a cell wall, which form is the cell-wall-deficient (CWD) form. In this way the attack against the pathogen turns against the host's own tissue. The Borrelia survives as the attack and the immune response damages the host's own tissue.

Some Os-proteins can bind factorH, which leads to an aspecific activation of the complement system.

In case of borreliosis these pathogenic processes may provoke the *development of autoimmune diseases.*

Similar to syphilis, a drug-induced flare reaction (the so-called *Jarisch-Herxheimer reaction* or *Herx)* may occur during the antibiotic therapy of Lyme patients. Leading to the exacerbation of symptoms caused by died-off bacteria, delivering toxins that circulate in the blood causing f.i. fever, night sweats, chills, fatigue, sleepiness, myalgias and even the worsening of other symptoms of the disease. The „herxing" patient may also become acutely suicidal, violent, psychotic, and/or confused. A trial course of the use of antibiotics which causes the worsening of the psychiatric symptoms followed by improvement suggests a Jarisch-Herxheimer reaction and can help to support the impression that a chronic infectious process is contributing to the psychiatric symptoms. The Herxheimer reaction evidently indicates that the antibiotic has reached its target.

Vaccination: not available yet

The prognosis can only be improved with a prompt diagnosis and an early, appropriate, effective treatment, extended and repeated if necessary. The prognosis is rendered more difficult by co-infections, got either by tick-bite (f.i. FSME or other tick-borne viral infections, babesiosis, ehrlichiosis, Mycoplasma fermentans infection, bartonella henselae infection) or by being present in any other way.

The diagnosis: is mainly based on the clinical exam findings and the history told by the patient about of his exposure to endemic Lyme areas. The EM rash, which does not always occur, is considered to be sufficient for the diagnosis even if the serologic data were negative. The serological testing is useful but not diagnostic. In stage I there is no seroreaction yet. Depending on the stage of the illness and on the state of the patient's immune response, the tests are not sensitive and specific enough. The PCR testing is specific and has a sensitivity of about 30-70%. The antibody tests, like the EIA and the ELISA have about 70% sensitivity, while the Westernblot test has about 70-96% sensitivity. A two-tiered protocol is recommended by the CDC: the more sensitive ELISA is to be performed first, and if it is positive or equivocal, the more specific Western blot is to be run. The reliability of the tests as concerns the diagnosis remains controversial. False negative results are common and false positive results are possible. In the late stage a seronegative disease can occur for the same reason as in the case of neurosyphilis: incomplete and/or intercurrent antibiotic treatments abrogate the antibody response, but are

not able to eliminate the infection. Though the possibility of the infection can not be ruled out by tests, the results nevertheless make a clinical diagnosis of a Lyme disease more (or less) likely. The clinical diagnosis is based on the anamnesis, the clinical signs and symptoms, and the serologic tests, which latter must often be repeated. Borrelia culturing, phase-contrast or DIC microscopy examinations are used mostly for scientific identifications and studies. The tests concerning co-infections are of great value.

Differential diagnosis: STARI (i.e. the southern tick associated rash illness), acute or chronic febrile illnesses, including malaria, salmonellosis, dengue, rat-bite fever, Weil's disease, autoimmune diseases etc. must by all means be considered.

Treatment: with antibiotics (such as doxycycline, amoxicillin azithromycine, clarythromycine, penicillin G, ceftriaxone, cefotaxime, cefuroxime minocycline must be administered for a long time (min. 6 weeks in the later stages) either in mono-treatment or in combination. Antibiotic treatment is the central pillar in the management of Lyme disease. In the late stage of borreliosis, symptoms may persist despite extensive and repeated antibiotic treatment. A lot of subspecies may be antibiotic-resistent. There is no consensus among the clinicians all over the world concerning the diagnosis, the testing and the exact and definite therapy of borreliosis in stage II-III, moreover, the controversy has led to two different opinions as regards care (see the ILADS-Guidelines and the Clinical Practice Guidelines of IDSA)

The Brain SPECT imaging method, the cognitive and memory tests, the measuring of the count of CD57+ Natural Killer cells with flow cytometry are useful for tracking the clinical progress or improvement.

RFR method: detects and eliminates the bacteria! Borrelia have a very great number of subspecies and more than 58 plasmids, thus it remains a very-very difficult problem to eliminate them.

The resonant frequencies of Borrelia Burgdorferi sensu stricto are: 378-382, 481-482 kHz

The resonant frequencies of Borrelia afzelii are: 378-382, 387-388 kHz

The resonant frequencies of Borrelia garinii are: 378-383 kHz

The most frequent resonant frequencies of its vegetative forms are: 301, 341, 420-422, 555-556 kHz

Other resonant frequencies of the CWD forms of Borrelia are: 300-302, 327-328, 341-342, 412-420, 421-424, 429, 510-511, 547-548, 556, 562-565 kHz

The resonant frequencies of Borrelia plasmids are: 305, 309, 312-322, 329, 344, 352-353, 357, 375, 389, 404, 409, 442, 452-453, 494-496, 500-520, 524, 536, 444-547 kHz

This list is not yet complete. You also have to measure it!

6.21. The Leptospira Genus

There are some opportunistic and eight humanpathogenic species of this Leptospira genus, such as the *L.interrogans*, the *L.kirschneri*, the *L.noguchii*, the *L.alexanderi*, the *L.weilii*, the *L.genomospecies*, the *L.borgpetersenii* and the *L.santarosai*. The Leptospira bacteria are also grouped into serovars according to their antigenic relatedness. The correct designation of a serovar is characterized by its genus, species and serovar name. f.i. Leptospira interrogans serovar Pomona. (The L.interrogans species contains more than 200 recognized serovars; sorted into 23 serogroups. Some serovars of the L.interrogans species are f.i. the Icterohemorrhagiae, Canicola, Australis, Tarassovi, Pyrogenes, Pomona, Grippotyphosa, etc.) All members of these Leptospira species have the same morphology. They are motil, aerobic, spiral-shaped spirochetes, having Gram-negative-like cell envelop and two flagellae. The outer membrane of the cell envelop contains a variety of lipoproteins and outer membrane proteins (Osp). Several leptospiral outer membrane proteins can be attached to the host's extracellular matrix and the factor H, which latter potency is important regarding its complement resistancy. The outer membrane of

Leptospira contains, as like as those of most other Gram-negative bacteria, lipopolysaccharides (LPS), accounting for the numerous serovars of Leptospira and for their low endotoxin activity.

6.21.1. Leptospirosis (Weil's disease, canicola fever, canefield fever, nanukayami fever, 7-day fever, etc.)

The leptospirosis is a bacterial zoonotic disease found all over the world, being caused by any one of the pathogenic leptospira species, regardless of its specific serovar. The syndromes of the infections caused by different serovars lead to the conclusion that a certain serovar of leptospira may be responsible for a variety of clinical features; conversely, a certain syndrome, e.g. aseptic meningitis, may also be caused by many of the serovars. Leptospirosis occurs in many of wild and domestic animals. Some animals act as carriers, sheding the bacteria via their urine, while others become ill and die. Though the infection is very common among animals, human beings acquire these infections but rarely, i.e. through moist contact with infected animal, or with their urine or owing to contaminated water getting via unhealed breaks of the skin, the eyes or the mucous membranes. Leptospirosis usually causes vague, flu-like symptoms, so that probably many cases go unreported. Infected people may seldom be without any symptoms, but.

The symptoms usually start after a 4-14 days latency abruptly with flu-like signs as high fever, strong headache, severe muscle pains, fatigue and chills. These symptoms cease and after a short latent period the second phase will begin. Meningitis can develop causing stiffness of the neck, headache and, in some complicated cases, even stupor and coma. Abdominal pain, vomiting, jaundice caused by liver damage may be present. The more severe form of the disease is known as Weil's disease or Weil's syndrome, characterized by continuous fever, stupor, disturbed haemostasis, causing bleedings within the tissues (f.i haemorrhage into the adrenal glands, hemorrhagic pneumonitis, subarachnoidal hemorrhage) and thus to anaemia. Complications such as liver damage (f.i. in case of L.interrogans serovar Icterohaemorrhagiae infection) and renal failure (f.i. in case of L.interrogans serovar Pomona infection) with painful and bloody urination, proteinuria, pyuria, azotemia and tubular necrosis are common. Neurological symptoms may include meningoencephalitis, optic neuritis, myelitis, peripheral neuropathy etc. Pulmonary haemorrhage can be an often lethal manifestation of leptospirosis. Pregnant, leptospirosis-infected women may miscarry.

Diagnosis: confirmed by tests as ELISA, PCR or microscopic agglutination test

Treatment: by administering antibiotics (Doxycyclin, penicillin, Amoxicillin etc.) and giving supportive therapy (detoxication, electrolytes, etc.)

RFR method: detects and may eliminate the bacteria.

The resonant frequencies are: 307-319, 332, 337-340, 353-358, 372, 383-389, 395-411, 425, 438, 450-454, 460-476, 506-510 kHz

This list is not yet complete; there are other subspecies with different frequencies not identified yet.

6.22. The Mysterious Nanobacterium

Nanobacterium sanguineum is thought to be the smallest, sized 20-200 nanometer, slow-growing organism which can only be seen with an electron microscope and is of a unique structure containing 16S ribosomal RNA. The term of calcifying nanoparticles (CNPs) is also used for it, side-stepping the question of their status as a life form.

Nanobacteria can be found in the milk of infected cows, anthropoids and human beings. These kinds of milk are long since held to be atherogen, the reason of which atherogeneity is nowadays thought to be the presence of nanobacteria in the kinds of milk mentioned.

Nanobacteria are rather heat-resistant, they can be eliminated only on 120 centigrade temperature lasting for 20 minutes.
Nanobacteria were cultured from human blood, calcific vascular wall plaques and kidney stones. It divides very slowly, splitting into daughter cells once every three days. The initial size of this nanobacterium is 20 nanometer and is dividing by binary fission within a self-elaborated biofilm, which begins to thicken and calcify developing into a calcium-coated slimy shell. This shell is an immobile calcium carbonate apatite deposit, an extraskeletal calcium compound found in the human body. This calcified shelter surrounding the nanobacterium sanguineum initiates calcification and plaque- formation in the coronary and other arterial walls, as well as the formation of kidney stones, choleliths, brain plaques, dental plaques and calcification in the prostate and the pineal glands. The nanobacterium population is not a unified group and various subspecies may live in the human body. A certain one of its subspecies causes the calcification of the arteries and the heart, while an other one causes that of kidney stones and a third one, again, that of dental plaques. Nanobacteria were observed by electron microscopy in the coronary artery plaques and in kidney stones coming into being in case of renal diseases. This calcification, triggered by this pathogen, may have a significant role in the pathogenesis of the diseases mentioned above.
Diagnosis: by special bacterium culturing, electron microscopy, applied calcium-specific and fluorescent monoclonal antibodies, x-ray, and CT Scanner can be used to localize and quantify this calcium-plaques.
Treatment: by administering chelating agents as EDTA with Doxycyclin.
RFR method: detects and may eliminate the nanobacterium.
See also Chapter 12.16.
The most frequent resonances are: 294-298, 305-310, 317-318, 324-325, 332-336, 345, 372-387, 395-399, 424-436, 430-435, 440-442, 466-476, 485-486, 528, 556-568 kHz

7. HUMAN PATHOGENIC FUNGAL INFECTIONS

The human pathogenic fungi have a particular tendency to cause infections among people with a compromised immune system. The manifestation of fungal infections often follows an antibiotic treatment. Local or systemic fungal infections occur frequently among elderly people, having a diminished immune defense. People with impaired immunity can get infections caused by those types of fungi that seldom, if ever inflict harm on people having a normally functioning immune system. Such infections are f.i. the *mucormycosis,* the *aspergillosis, etc.* A few species of fungi, f.i. some *Candida species*, can live also on healthy human body surfaces or on the mucous membranes of the intestines. These normal inhabitants of the body only occasionally cause local infections of the mucous membranes (f.i. of the vagina or the mouth), moreover, they can also produce infections in the lungs and the liver. These fungal infections develop slowly and usually their treatment also takes a long time.

Mycotic diseases are but seldom transmitted from person-to-person. Many fungal infections may be *acquired by inhalation of spores* growing freely in nature. The chlamydospore is the thick-walled resting spore of several kinds of fungi. In this stage of its life the fungus survives in unfavourable conditions f.i. in dry, hot seasons. They can be rectangular and unicellular mycelial fragments known as chlamydospores, or spherical, multicellular, asexually produced on thin mycelial stalks, and are called chlamydoconidia.

Some fungal infections, such as the *sporotrichosis*, are results of the *inoculation* of spores directly into the skin, while others are mildly contagious and can be transmitted from domestic animal-to-man or person-to-person, such as the ringworm, which may be spread by skin-to-skin contact, as well as via contact with contaminated items such as hairbrushes.

The *hypersensitivity of the host* can also be a possible pathogenetic factor of fungal infections. In most cases of the fungal diseases, the intradermal injection of the causative microorganism provokes a marked local or even a systemic reactivity of the host's immune system. In case of *coccidiomycosis* this type of reaction is closely associated with the development of erythema nodosum and pleural effusion. In case of *allergic pulmonary aspergillosis*, the disease results from both the reacting and the precipitating antibodies against the aspergillus antigens. The occurrence of necrosis at the spot of the intradermal injection containing fungal antigens suggests that the hypersensitive reaction of the immune system can also be responsible for the necrosis of the infected tissues.

The human pathogenic fungi belong mostly to the thermally dimorphic fungi, the molds, the budding yeasts and the dermatophyton species.

7.1. Human Pathogenic Thermally Dimorphic Fungi

Dimorphic fungi are fungi that can reproduce themselves as either a mycelial or a yeast-like state. The mycelial, saprophytic form generally grows at 25° C and the yeast-like, pathogenic form grows at 37° C. This dimorphism is important in the identification of mycoses, as it makes possible a rapid identification of many pathogenic organisms. The most important diseases caused by dimorphic fungi are the *sporotrichosis*, the *blastomycosis,* the *cryptococcosis*, the *histoplasmosis,* the *coccidioidomycosis*, the *paracoccidioidomycosis* and the *penicillosis.*

7.1.1. Sporotrichosis

Sporotrichosis is worldwide a rare chronic infection caused by *Sporothrix schenckii*. This fungus lives in the soil or as a saprophite on plants, and usually affects gardeners and agricultural workers. The illness is characterized by the formation of suppurating nodules along the lymphatics of the spot of the infected skin and the subcutaneous tissues. A haematogenous dissemination is very rare, occuring only in case of immunosuppressed patients.

Symptoms: There is a marked disproportion between symptoms and findings. In case of the *cutaneous form* of the disease, a chain of hard, reddened and discrete lumps is extending over the arm to the axilla or on the leg to the groin, while the intervening lymphatics being though red and thickened, do not cause any pain, fever, or any other constitutional symptoms. The older nodules may rupture producing thus fistulas or ulcers. In case of the *rare pulmonary sporotrichosis* the infection occurs via inhalation of spores, causing swollen hilar lymphnodes, productive coughing, nodules and cavitations of the lungs leading to fibrosis. In the *disseminated form* of the disease the infection can spread to joints and bones, as well as to the CNS and the brain causing monoarthritis which can result in severe functional impairments and even meningitis, weightloss and anorexy. The disease can prove to be rapidly fatal.

Diagnosis: by recovering the fungus microscopically (typical clusters of pear-shaped spores are found at the tips of the conidiophores arising from the tangled mass of delicate, branched mycelia) and by isolating the fungus in a specimen culture or by using serological techniques.

Differential diagnosis: Mycobacterial infections, yeast infections or other fungal infections.

Treatment: by administering antifungal drugs or potassium iodide for a long time. The disseminated sporotrichosis is resistant to iodides, but may respond to intravenous antifungal drugs.

RFR method: detects and eliminates the fungi. The treatment must often be repeated.

The resonant frequencies are: 306, 357-359, 365, 385-386 kHz

This list is not complete; there are other subspecies having different frequencies.

7.1.2. Blastomycosis

Blastomycosis is a fungal infection of the skin and the viscera caused by *Blastomyces dermatitidis*. The disease is endemic in some regions of the United States, but there are reported cases in Canada (Ontario, Manitoba) and the African continent as well. Though *Blastomyces dermatitidis* can seldom be cultured from the soil, it still remains the most likely source of the fungus. The infections occur mostly among people living in close contact with soil. The lung appears to be the main portal of entry for both types of blastomycosis. As regards both infections primary lesions may give rise to progressive diseases, with or without a variable latent interval. The mucosal lesions, in turn, invariably spread to the regional lymphatic to produce massive lymph node enlargements with necrosis and sinuses draining through the skin. Hematogenous spreads can cause massive enlargements of the abdominal or the mediastinal lymph nodes. The spleen, the liver, the urogenital tract and the brain can also be infected. An intestinal ulceration can develop after the rupture of infected submucosal lymphoid tissue. In the skin and in the mucous membranes this combination of abscesses and epitheloid cell granulomas occurs in the midst of pseudoepitheliomatous hyperplasia.

Symptoms: the disease has more clinical types. Similar to flu, the first type causes fever, a productive cough, joint and muscle pains showing thus multiple nodular pulmonary densities in the roentgenograms as well as budding yeast in the sputum. Concerning the other type, a pleural chest pain is also present, similar as in case of pneumonia. Hemoptysis, purulent sputum and dyspnea appear as the disease progresses. Although the

pulmonary infection may subside spontaneously, extrapulmonary lesions of skin, bones, joints, urinary tract and viscera eventually call attention to disseminations.
Diagnosis: by microscopic examinations of the biopsied material, of sputum, or pus. By fungi culture, CT, and MRI.
Differential diagnosis: tuberculosis, carcinoma, coccidioidomycosis, actinomycosis, nocardiosis, and histoplasmosis.
Treatment: by administering Amphotericin B, Itraconazole, Fluconazole, Sulfadiazine, Sulfisoxazole, Sulfonamide and by the surgical excision of destroyed tissues.
RFR method: detects and eliminates the fungi.
The resonant frequencies are: 304-307, 316-319, 371-373, 428-434 kHz
This list is not complete. The destruction of fungi requires a long-term treatment, with repeated check-ups for effectiveness.

7.1.3. Cryptococcosis

The dimorphic basidiomycota are smuts being either in a yeast state or in an infectious hyphal state. The *Filobasidiella genus* forms basidia on hyphae, its main infectious stage is commonly known by the anamorphic yeast name *Cryptococcus*.
The Cryptococcosis is an infection caused by the *Cryptococcus neoformans* species, which is an encapsulated yeast having a special affinity for the central nervous system. The illness occurs with increasing frequency in case of patients suffering from leukemia or lymphoma. This fungus can be isolated from the soil, from the surface of fruits, from the farmers' skin and even from the normal human intestinal tract. The most frequent sources of the virulent strains, however, are the pigeon droppings, providing an alkaline medium rich in nitrogen and salts, which promotes the survival of the Cryptococcus neoformans in the dust composed of these materials. Though the neurologic disturbances overshadow that of the other ones, there is a good evidence, that the infection usually begins in the lungs and in other viscera before the disseminating into the brain and the meninges. The Cryptococcus does not evoke any active inflammatory response observed in case of other fungi or bacteria. The cellular reaction is very slowly developing and is seldom intense. The cryptococcus seems to meet little resistance and frequently proliferates so freely that the macroscopic masses of gelatinous yeasts fill the lesions. However, many cryptococci are present even within the mononuclear and the giant cells. Granula, gelatinous cryptococcal amorphous nonfibrillar masses, fibrillary tangles or amyloid fibers may appear in the nervous system. In the nervous system, lesions usually develop in the meninges at the base of the brain, involving the brainstem, the cranial nerves and the cerebellum. Large masses of yeast in the subarachnoid space may extend diffusively along the perivascular spaces and, getting into the brain, produce cystic nodules.
Symptoms: The clinical picture is often the same as that to be experienced in case of Alzheimer's disease. Cryptococcal infections may play an important role in the development of Alzheimer's disease. Most patients suffering from cryptococcal infections consult a physician only after experiencing the onset of neurologic manifestations. Complaints, such as severe headache, diplopia, dizziness, ataxia, vomiting, tinnitus, memory disturbances, memory loss and Jacksonian convulsions are common. Fever is usually absent. Many patients die within a few months, while others live for many years as the disease undergoes remission and relapses.
If only a pulmonary infection is present, the patient is generally free from all constitutional symptoms. The possibility of an underlying Hodgkin's disease, lymphosarcoma, leukemia, or diabetes is to be considered in case of every patient infected with cryptococcus.
Diagnosis: by the isolation of Cryptococci, by examinations with CT, MRI and x-ray. If the fungi cannot either be seen microscopically, or be cultured, the discovery of cryptococcal antigens in the cerebrospinal fluid can be particularly important when diagnosing meningitis.

Differential diagnosis: by distinguishing it from other fungal infections. The criptococcal meningeal process must be distinguished from other diseases resembling or mimicing aseptic meningitis, including brain abscess, tuberculous meningitis, coccoidal meningitis and carcinomatous meningitis.
Treatment: by administering Amphotericin B infusion, Itraconazole, etc.
RFR method: detects and eliminates the Cryptococci.
The resonant frequencies are: 295-298, 303-305, 372-375, 401-405, 438-446, 454, 486-488, 534, 540-544 kHz

7.1.4. Histoplasmosis (Histoplasma capsulatum)

The histoplasmosis is a fungal infection caused by the dimorphic Histoplasma capsulatum. The infection occurs mainly in the lungs but can spread sometimes to all parts of the body. The spores of this Histoplasma are present in the soil and in materials contaminated with bat or bird droppings. People infected with HIV are more likely to develop histoplasmosis.
Symptoms: may appear within 3-17 days after inhaling a big amount of histoplasmal spores represented by microconidia. In the alveolar spaces the spores transform into budding yeast cells. In some cases of infection the patients have no symptoms at all. In some *acute cases* the person may feel sick, have fever and nonspecific respiratory symptoms such as chest pain, cough and asymptomatic pleural effusions may also be present as well. Arthritis, erythema nodosum or erythema multiforme may indicate the presence of an immunological process. In case of a patient with a compromised immune system this acute form of histoplasmosis could even be fatal.
The *acute, progressive, disseminated form* usually occurs among infants and people having an impaired immune system, f.i. AIDS. The infection can rapidly spread, so that the brain, the liver, the spleen and the lungs will be affected and the lymph nodes may become enlarged. The occurrence of ulcers in the mouth and in the intestines is less common. Sometimes, but not too often, the adrenal glands may be damaged, causing Addison's disease.
In case of the *subacute, progressive, disseminated form* gastrointestinal ulcers and lesions, meningismus, muscle weekness and other neurological abnormalities and even cardiac involvements including endocarditis may develop.
In the *chronic form* of histoplasmosis, the lung infections develop gradually over several weeks, producing cough and an increased difficulty in breathing. The symptoms include weight loss, a feeling of illness, and mild fever.
In case of chronic, progressive, disseminated histoplasmosis there are oropharyngeal ulcers also present. The difficalty in breathing can get gradually worse, and some people may cough up blood, even in large amounts. The lung damage or a bacterial co-infection of the lungs may eventually cause death mostly as regards immunocompromized individuals.
If an ocular histoplasmosis syndrome is present, the involvement of the eyes may be bilateral, retinal hemorrhages and retinal detachment, or edema may come about, moreover, if the infection is located on the macula, this process can lead to blindness.
Diagnosis: considering the identification of the histoplasma capsulatum cultured from sputum, lavage or blood, as well as by tests using complement-fixing antibodies or immunoprecipitating antibodies or by antigen detection from the serum and the urine.
The treatment: is not indicated in case of immunocompetent persons as the illness is self-limiting. Concerning a prolonged infection, or in regard to a systemic one, as well as in case of immunocompromised patients there usually is an intravenous antifungal treatment recommended. In the chronic cavitary form of the histoplasmal lung disease, drugs may eliminate the fungus, nevertheless the destruction caused by the infection heals by leaving a scar. Drug-resistent and polyresistent subspecies often occur. Therefore, treatment should begin as soon as possible to limit lung damage.

RFR method: has an important role in case of antifungal drug-resistance, as the RFR detects and eliminates the histoplasma if used together with an antibiotic treatment. The histoplasma is often resistant to antimicrobic agents. Repeated treatment is necessary for an extended period of time.
The resonant frequencies are: 298-308, 315-319, 374, 380-385, 424, 432-435 kHz

7.1.5. Coccidioidomycosis (Coccidioides immitis and Coccidioides posadasii)

Coccidioidomycosis (named also *San Joaquin Valley fever*) is a dimorphic fungal infection caused mostly by inhaling spores of *Coccidioides immitis* affecting usually the lungs. This fungus grows in dry and semidry areas of the Western Hemisphere. In the United States, this disease is endemic from California to southern Texas and also in some regions of Utah. The infection occurs either as a mild lung infection healing without any treatment, or as a severe, progressive infection spreading throughout the body, proving mostly to be fatal among people undergoing a chemotherapy or a corticosteroid therapy or any other immunosuppressive therapy. AIDS, kidney failure, diabetes mellitus and cancerous diseases are all risk factors of the disease.
Though most infections are mild or unapparent, if disseminated, they nevertheless may be fatal, causing destructive lesions and abscesses in the lungs, the subcutaneous tissues, the spleen, the liver, the bones, the kidneys and the brain. Farmers and other people working with soil might inhale the spores and thus become most likely infected.
Symptoms: Most people suffering from the acute primary form of coccidioidomycosis have no symptoms, others, however, may experience fever, chest pain, chills, cough, conjunctivitis, arthritis, and, occasionally, erythema nodosum. Symptoms may include loss of appetite and weight aswell. The *lung infection* can worsen, causing shortness of breath. The infection may spread from the lungs to the bones, the joints, the kidneys, the spleen, the liver, the lymph nodes, and the brain. The most serious type of the *progressive disseminated coccidioidomycosis* is often fatal, causing meningitis too. This progressive form may be a sign that the person has a compromised immune system. The course of disseminated infections is marked by fungal, ulcerating skin lesions, multiple pulmonary nodules or cavities and a widespread destructive lymphadenopathy, osteomyelitis and even meningitis. Weight loss, fever, and weakness are common systemic manifestations of the illness. The course of the illness is often rapid, leading to death within less than a year. If, however, vital organs are spared, however, the patients with disseminated coccidioidomycosis may feel surprisingly well, can continue to work, and might even gain weight despite the presence of large numbers of *Coccidioides immitis* in the sputum or in the subcutaneous abscesses. If suffering meningitis with progressive hydrocephalus, the patients will experience severe headaches, cranial nerves palsies, memory disturbances, and disorientation.
Diagnosis: by the detection of antibodies and fungi.
Differential diagnosis: by distinguishing it from other fungal infections.
Treatment: by giving antifungal drogs. The treatment must be continued for years, often for the rest of the patient's life. Untreated meningitis is always fatal. If a person develops meningitis, Fluconazole or Amphotericin B have to be injected into the spinal fluid. Surgery.
RFR method: may enhance the effect of the antifungal therapy and reduce the time needed for the treatment, but the first step has to be the giving of antifungal drugs. The RFR method is very useful in case of cerebral infections.
The resonant frequencies are: 337-338, 347, 369, 434, 442-443 kHz
This list is not complete; there are other subspecies having different frequencies.

7.1.6. Paracoccidioidomycosis (Brazilian blastomycosis)

Paracoccidioidomycosis (known also as Lutz-Splendore-Almeida disease, South American blastomycosis or Brazilian blastomycosis) is caused by the thermally-dimorphic fungus *Paracoccidioides brasiliensis*. This fungus causes a systemic mycosis involving the mucous membranes, the lymphnodes, the bones and the lungs concerning patients in an immunosuppressed state.

Symptoms: The primary infection can be autolimited and asymptomatic. *The juvenile form of the disease* is progressive, causes high fever, generalized lymphadenomegaly and milliary lesions in the lungs, having a bad prognosis even with treatment. *The adult form* of the illness is certainly the reactivation of the disease, involving the lips, the oral mucosa causing painful lesions and cervical lymphadenitis. Lobal pneumonia or pleuritis with fever, coughing and weight loss are the usual symptoms. The bones, the arteries, the spleen and the meninges are seldom involved.

Diagnosis: by biopsy, culturing and by serodiagnostics.

Differential diagnosis: by distinguishing it from mucocutaneous leishmaniasis, yaws, tuberculosis, other fungal infections.

Treatment: by administering sulfa drugs, antifungal drugs for a long time even up to three years. Corrective surgery may be needed.

RFR method: detects and may eliminate the fungus.

The resonant frequencies are: 304, 348, 431-434 kHz

This list is not complet.

7.1.7. Penicillosis

The only known thermally dimorphic member of the Penicillium genus is the *Penicillium marneffei* species, which can cause penicillosis, a lethal systemic fungal infection among AIDS patients who had at one time visited Southeast Asia or were living there permanently. The most common

Symptoms are fever, anemia, hepatomegaly, lymphadenopathy, and productive coughing.

Diagnosis: by biopsy, culturing, xray examinations etc.

Treatment: by administering antifungal drugs, such as Amphotericin B, Itraconazole, Fluconazole.

The resonant frequencies are: 285-290, 315-316, 321, 342, 352-353, 366, 372, 391, 444, 472, 495, 509 kHz

This list is not complet.

7.2. Human Pathogenic Common Molds

The molds are asexually-reproducing, microscopic fungi growing in form of multicellular filaments, called hyphae. Molds can be found in the divisions of Zygomycota, Deuteromycota and Ascomycota fungi as well as f.i. the species of the *acremonium genus*, the *Fusarium genus*, the *Cladosporium genus*, the *Mucor genus*, the *Stachybotrys genus* and the *Aspergillus genus*. Some species belonging to the common molds may cause diseases of their own (f.i. fusarium species can cause fusariosis, aspergillus can cause aspergillosis etc.) There are many species of common molds, which are able to cause similar clinical manifestations f.i. the aetiological agents of the mycotic mycetoma can be either species of Acremonium or of Fusarium, or of Aspergillus etc. The brown or black-pigmented dematiaceous fungi can cause different diseases depending on their morphology in the tissue.

7.2.1. Mycotic Mycetoma

This clinical manifestation of infection caused by different fungi occurs among people and animals living in tropical or subtropical regions. Certain species of the genus such as *Acremonium, Fusarium, Aspergillus nidulans, Madurella mycetomatis, Madurella grisea* etc. can cause mycotic mycetoma. The disease is *characterized by granules and draining sinuses* resulted from the traumatic implantation of the fungus into the cutaneous or subcutaneous tissue, moreover, even into the fascia and the bone of the extremities of barefooted individuals.
The serosanguinous fluid discharged from the sinuses contains granules which vary in size, colour and degree of hardness, depending on the etiologic species of the fungi.
Symptoms: start with a small, painless, hard nodule at the spot of the inoculation. The nodule will soften and ulcerate discharging a purulent fluid containing granules. The infection may involve the deeper tissues such as the bone causing osteomyelitis.

7.2.2. Chromoblastomycosis (Cladosporiosis)

The Chromoblastomycosis is a rare, long-lasting fungal infection of the skin and the subcutaneous tissue occuring mostly in tropical or subtropical climates. The causative agents of the disease are soil fungi producing brown or black melanin pigments. These fungi, belonging mostly to the Dematiaceae family, are species of the *Cladosporium genus*, (such as the *Cladosporium carrionii*) the *Fonsecaea genus* (such as the *Fonsecaea compacta* and the *Fonsecaea pedrosoi*) and the *Phialophora genus* (such as the *Phialophora verrucosa*). The characteristic hallmark of the disease is the socalled ***sclerotic body in the infected tissue.*** It is a vegetative, rounded, muriform cell of these dematiaceous fungi. These fungi live in the soil or in the vegetation and can accidentally enter the skin of agricultural workers. The infection occurs mostly on the lower extremities of barefooted workmen, but sometimes on the upper extremities or buttocks, too.
Symptoms: The initial trauma caused by the infectionis often not noticed or forgotten. The infection can be latent at the locus of the inoculation for a long time, later on, however, a *small red papule* may appear and become *ulcerated.* If the fungi spread through the blood vessels or the lymph vessels metastatic lesions may develop. In the *verrucous form* of the disease the pathological process begins with microabscesses in the skin containing numerous fungi. The process ends in an extensive fibrosis, an epidermal hyperplasia and hyperkeratosis. The lesion may progress along the lymphatics. Over a period of years there appear adjacent new lesions and as the epithelial hyperplasia and hyperkeratosis increases, the entire area assumes a cauliflower-like appearance. A bacterial secondary infection and the scarring of the lymphatics may lead to the obstruction of the lymph vessels causing *lymphedema* and even *elephantiasis,* in which case pain and constitutional symptoms can also be present. In the *cystic form,* subcutaneous and intramuscular cysts and abscesses full of pigmented fungi can develop at the locus of punctured wounds. The species *Phialophora gougerotii* is the most common cause of such cystic skin lesions.
The resonant frequencies of Cladosporium are: 397, 448, 476, 520-523 kHz

7.2.3. Phaeohyphomycosis

This mycotic infection of human beings (and animals of lower order as well) is also caused by a number of dematiaceous (brown-pigmented) fungi *where the tissue morphology of the causative organism is mycelial.* (This separates it from other clinical types of the diseases (mycotic mycetoma and chromoblastomycosis) involving brown-pigmented fungi. The etiological agents include various dematiaceous hyphomycetes, f.i. species of *Exophiala, Phialophora, Wangiella, Bipolaris spp., Exserohilum, Cladophialophora, Alternaria, Phaeoannellomyces, Aureobasidium, Cladosporium, Curvularia,* etc.
The species *Cladophialophora bantiana (Xylohypha bantiana)* owns a distinct neurotropism. The CNS phaeohyphomycosis due to *C. bantiana* is an uncommon infectious condition, in ehich brain abscesses may be present, and is associated with high

mortality. These brain abscesses are hematogenous and tend to occur as opportunistic infections in case of debilitated patients. The phaeohyphomycosis of the brain is indistinguishable from other forms of brain abscesses.

Diagnosis: by microscopic identification of the typical granules or of the dark-brown septate hyphae or of the sclerotic bodies or of the myceliae. For a specific identification, it is necessary to culture the slow-growing fungus on Sabouraud's agar.

Treatment: by administering Amphotericin B, Flucanazol, Itraconazol and other systemic and local antifungal drugs. Surgical procedures may be necessary. The phaeohyphomycosis is invariably fatal, but the RFR technical treatment gives hope for survival.

RFR method: The frequency list is not complete; some certain frequencies of these fungi are not yet detected. These fungi are of low sensitivity to RFR method. Their eliminating requires a long-term, extensive treatment. The RFR method of the mycotic brain abscess may cause brain edema!

The resonance frequencies are: 396-400, 447, 449, 476, 478, 518-524 kHz

7.2.4. Hyalohyphomycosis

This illness occurs solely in case of a prolonged leukopenia, f.i. concerning leukaemia patients, bone marrow transplant recipients, AIDS patients etc. The fungi causing this illness are opportunistic non-pigmented fungal pathogens including species of *Acremonium*, *Fusarium* and *Scopulariopsis*. Hyalohyphomycosis can be characterized by its harmless saprophytic colonization which may delevop into an invasive fungal infection. In that case

Symptoms: may include arthritis, osteomyelitis, peritonitis, endocarditis, pneumonia, cerebritis and subcutaneous infection as well.

7.2.5. Fusariosis

Fusariosis is a fungal infection affecting plants, animals, and human beings. This Fusarium fungus is heat-resistant, its strong toxin causes Fusarium toxicosis. Fusarium is a filamentous fungus widely distributed on plants and in the soil. It is found in normal mycoflora of commodities, such as in rice, wheat, beans, soybeans and other crops. Although most species are more common in tropical and subtropical areas, some inhabit the soil even in areas of cold climate. In addition to being a common contaminant and a well-known plant pathogen, the Fusarium species may cause various superficial or systemic infections among humans or animals. Fusarium is one of the emerging causes of *opportunistic mycoses*. The genus Fusarium currently contains more than 20 species. The most common of these are *Fusarium solani*, *Fusarium oxysporum* and *Fusarium chlamydosporum*, the most virulent of them being the *Fusarium solani*. Trauma is the major predisposing factor for the development of *cutaneous infections* due to Fusarium strains. On the other hand, *disseminated opportunistic infections*, develop in immunosuppressed hosts, particularly as concerns neutropenic and transplant patients (see Chapter 7.2.4.). Fusarium infections which follow the solid organ transplantations tend to remain local and have a better outcome compared to those which develop in patients with hematological malignancies or who undergone bone marrow transplantation.

Symptoms: *In case of people with a normal immune system*, fusarial infections may occur in the nails causing there *onychomycosis*, while in the cornea they cause *mycotic keratitis*. *In an immunosuppressive state* f.i. *endophthalmitis*, *otitis media*, *cutaneous infections* particularly of burn wounds, *mycetoma*, *sinusitis*, *pulmonary infections*, *endocarditis*, *peritonitis*, *central venous catheter infections*, *septic arthritis*, disseminated infections and *fungemia* may be present. The Fusarium species produce *mycotoxins*. The main toxins are fumonisins and trichotecenes. These toxins can cause *parenchyma degeneration* of the

liver while the ingestion of grains contaminated with these toxins may give rise to *allergic symptoms* or become *carcinogenic* after a long-term consumption. They may be the indirect causal factors of oesophageal cancer. The zearalenones, produced by some Fusarium species growing in grains, are another group of mycotoxins.

Diagnosis: hyaline septate hyphae, conidiophores, phialides, macroconidia and microconidia by microscopically observing. In addition to these basic elements, even chlamydospores are produced by *Fusarium chlamydosporum, Fusarium napiforme, Fusarium oxysporum, Fusarium semitectum, Fusarium solani and Fusarium sporotrichoides*. Macroscopic and microscopic features, such as the color of the colony; the length and the shape of the macroconidia; the number, the shape, and the arrangement of the microconidia; and the presence or absence of chlamydospores are the key features of diagnosis.

Treatment: Fusarium is one of the most drug-resistant fungi. The species *Fusarium solani* tends to be the most resistant of all. The Fusarium strains have high MIC for flucytosine, ketoconazole, miconazole, fluconazole, itraconazole and posaconazole. Despite that it does not act if its own activity alone, the combination of caspofungin with amphotericin B appears to be synergistic against some Fusarium isolates. The only antifungal drugs which yield to a relatively low MICs for Fusarium are amphotericin B, voriconazole and natamycin. Compared to the itraconazole, the voriconazole has notably lower MICs.

The infections caused by Fusarium spp. are difficult to treat, and their invasive forms prove often to be fatal. Amphotericin B alone or in combination with flucytosine or rifampin is the most commonly used antifungal drug for the treatment of systemic fusariosis. Lipid formulations of amphotericin B, such as liposomal amphotericin. B and amphotericin B lipid complex, are also used. However, most cases remain resistant and fail to respond to amphotericin B treatment. Granulocyte and GM-CSF transfusion concommitant to amphotericin B therapy may be life-saving in case of some immunosuppressed patients with disseminated fusariosis. Topical natamycin is used for the treatment of keratitis caused by Fusarium. In addition to the antifungal therapy, keratoplasty is also required for certain patients. Patients with mycetoma due to Fusarium may respond to itraconazole, too. On the other hand, onychomycosis due to Fusarium, may be treated with itraconazole and ciclopirox nail lacquer.

RFR method: The genus Fusarium currently contains more than 20 species, and all frequencies are unknown.

The resonant frequencies of Fusarium oxysporum are: 339-342, 360-362, 398-401, 407-411, 416-417 kHz

The resonant frequencies of other Fusarium species are: 320, 381-383, 393 kHz

The destruction of these fungi requires a very long-term, extensive treatment.

7.2.6. Phycomycosis

The Phycomycoses are diseases mostly caused by various molds. Their individual forms are the *zygomycosis*, the *pythiosis* and the *lagenidiosis*. The zygomycosis (the synonym of mucormycosis) is caused by Mucorales (such as *Mucor, Rhizopus, Rhizomucor, Mortierella, Absidia and Saksenaea),* but can also be caused by some human pathogenic species of entomophthorales (such as *Basidiobolus* and *Conidiobolus*). The most common type of phycomycosis is the pythiosis caused by *Pythium* species, a type of water mold. The causative agent of Lagenidiosis is a water mold too, called *Lagenidium* species. The common name of pythiosis and lagenidiosis is oomycosis, the causative agents being members of the class of Oomycetes. Infections of the Lagenidium species have been not identified in mammals other than dogs. In a more specific sense, the Phycomycosis is an uncommon illness of the gastrointestinal tract and the skin, which is mostly found in dogs and horses and rarely affects human beings.

7.2.6.1. Zygomycosis

7.2.6.1.1. Zygomycosis Caused by Mucorales (Mucormycosis)

The causative agents of these rare, yet sometimes life threatening fungal infections are mostly the species of the Mucor genus and the Rhizopus genus. Mucormycosis is caused by inhaling the spores of these fungi. The infection usually affects the face and the oropharyngeal cavity, spreads rapidly involving the sinuses, the brain or the lungs and results in thrombosis and tissue necrosis. Sometimes the gastrointestinal tract and the skin may also be attacked.

The Symptoms of the very rare cutaneous form of mucormycosis include a painful, hardened area with grotesque swellings of the skin (most commonly of the neck and the chest) which may have a blackened central area. The symptoms *of the rhinocerebral form* of the disease are usually unilateral, starting in the paranasal sinuses after inhaling the spores. Retro-orbital headache, fever, nasal stuffiness with black discharge are characteristic. At first, the affected area of the skin does not alter, but when progressing, the skin becomes reddish, edematous and at the end stage, necrotic. Predisposing factors include uncontrolled diabetes mellitus or acidosis, steroid-induced hyperglycemia, especially regarding patients with leukemia and lymphoma, renal transplanted people and persons getting concomitant treatment with corticosteroids and immunosuppressive drugs. The infection usually involve the orbit, the palate, the face, the nose and the brain. In case of *pulmonary mucormycosis or disseminated mucormycosis* dyspnoe, a persistent cough and even hemoptysis can be present. The *intestinal mucormycosis* takes the form of a hemorrhagic segmental infarction of the ileum or the colon. The invasion into the coronary arteries may produce myocardial infarctions.

The more serious kind of mucormycosis is called *zygomycosis*. The causes of this form are certain opportunistic Rhizopus species (such as f.i. *Rhizopus oryzae*), which have a rapid growth-rate and are thermoresistent to high temperatures. The infection attacks the skin, the sinuses or the gastrointestinal tract of immunocompromised patients f.i. HIV/AIDS patients, elderly ones and people with uncontrolled diabetic, transplantant recipients, etc. Besides *Rhizopus oryzae (synonym Rhizopus arrhizus), also Absidia, Rhizomucor, Mucor, Cunninghamella, Saksenaea, Apophysomyces, Cokeromyces* and *Mortierella* species can cause subcutaneous and systemic zygomycosis. These infections are usually severe, even fatal. The symptoms include pain, fever, swelling and proptosis (protrusion of the infected orbit) of the affected eye. *The cerebral mucormycosis* may be associated with a hematogenous spreading to the pulmonary and intestinal vessels, moreover, it can show the signs of an acute, diffuse, cerebrovascular disease. The thrombosis of the arteries and the veins may lead to multiple infarcts all over the brain, though only a minimal inflammatory response is present. In case of a brain infection convulsions, inability to speak properly, ophthalmoplegia and other partial paralysis may occur.

Prognosis: the cerebral mucormycosis is almost invariably fatal.

Diagnosis: by the culturing and identification of the fungi taken from the skin or the sputum of immunocompromised patient.

Treatment: by iv. administering Amphotericin B, or drugs advised by guidelines and so often also by surgical action.

RFR method: detects and may eliminate the fungi.

The resonant frequencies of Zygomycosis (also called mucormycosis; usually associated with uncontrolled diabetes mellitus or immunosuppressive drugs) **are: 299, 318-320 kHz**

The resonant frequencies of Basidiomycetes are: 480-483 kHz

This list might not be complete; there are some other subspecies too, which have different resonance frequencies.

7.2.6.1.2. Zygomycosis Caused by Entomophthorales

Two species of this order, such as the *Basidiobolus* species and the *Conidiobolus* species can cause subcutaneous zygomycosis (synonym: entomophthoromycosis). This *subcutaneous form* of the zygomycosis is usually chronic, slowly progressing and generally restricted to the subcutaneous tissue of otherwise healthy individuals. The zygomycosis caused by *Basidiobolus ranarum* is a chronic inflammatory or *granulomatous disease* affecting the subcutaneous tissue *of the limbs, the chest, the back or the buttocks*, and is primarily occurring among children.

The zygomycosis caused by the *Conidiobolus* species is a chronic inflammatory or *granulomatous disease* which is typically restricted to the *nasal* submucosa, is characterised by polyps or palpable restricted subcutaneous masses. Its symptoms are nasal obstruction, draining and pain.

Diagnosis: by culturing and identification of the fungi from nasal fluid.

Treatment: by administering antifungal drugs, advised by guidelines and often by surgical action.

RFR method: detects and may eliminate the fungi.

The resonant frequencies are: 336-340, 376-389 kHz

This list is not complete yet.

7.2.6.2. Pythiosis

This rare oomycetic disease, caused by *Pythium insidiosum* occurs mostly in the Gulf Coast Region of the United States and also in South America, southeast Asia, eastern Australia and New Zealand. This fungus-like aquatic microorganism can survive in standing water which does not freeze in mild winters and can infect, through their open wounds, people working in contaminated wet and swampy areas.

Symptoms: This disease attacking human individuals may be manifested either in a *subcutaneous form*, or a *systemic vascular form* or an *ophthalmic* one. The systemic type occurs usually among thalassemic people or leukemic patients and involves the vascular system causing progressive ischemia and even the arterial occlusion of the lower extremities, and ascending arteritis and aneurysm. Most of these systemic forms are resistent to therapy. In case of a cutaneous-subcutaneous form lesions will appear on the limbs, the periorbital and facial areas causing corneal ulcers.

Diagnosis: by using immunoblot techniques and PCR techniques

Treatment: by administering supersaturated potassium iodide for the chronic cutaneous form of the illness, combinated with antifungal drugs, as well as by giving therapeutic vaccinations, and also by surgical action.

RFR method: detects and may eliminate the fungus.

7.2.7. Stachybotrys

The so-called toxic black mold *Stachybotrys chartarum* produces trichothecene mycotoxins (satratoxins) and can cause diseases by inhaling or ingesting its spores in great amounts. This occurs in case of poor indoor air quality that arises due to fungal growth on water-damaged building materials. The cellulose (especially the water damaged one) promote the growth of *Stachybotrys*.

Symptoms: Flue like symptoms, headaches, fatigue, fever and rashes. The inflammation of the conjunctiva and the mucous membranes of the mouth and the airways can cause itching, sneezing and catarrhal angina, bloody rhinitis, cough, throat pain, chest tightness and occasionally fever. Erosions can occur on the oral mucosa and the gingival mucosa, too.

Therapy: there is no special therapy. By altering the living conditions the symptoms can be avoided.

RFR method: detects the fungus
The resonance frequencies are: 395, 434, 578 kHz
This list is not complete.

7.2.8. Aspergillosis

Aspergillosis, caused by the fungus *Aspergillus fumigatus, Aspergillus flavus* and other species of the Aspergillus group, causes many a disease, mostly affecting the lungs and the sinuses. The Aspergillus fungus is commonly found in compost heaps, around the house, as well as on food and on the body, too. These molds grow chiefly on many common building materials soiled or damaged by water causing a significant health care problem, everywhere, where the maintenance of buildings is neglected and insufficient. The infections occur mostly by inhaling a great amount of spores. Many conditions or therapies can suppress a person's immune system furthering thus the chance of being infected by this mold, f.i. elderly people are more susceptible. The colonization of the mold on the respiratory tract (i.e. the sinuses and the lung) is common. Some persons experience allergic reactions to the Aspergillus found on their body surfaces even if their tissues are neither invaded nor infected yet. Some species of *Aspergillus* produce well-known toxins such as aflatoxins, ochratoxins and sterigmatocystin. Aflatoxins, produced by *Aspergillus flavus* and *Aspergillus parasiticus,* can be present in stored peanut and grains and are not only toxic but carcinogen, too. *Ochratoxins* are produced by many species of *Aspergillus* (*Penicillium* as well). Ochratoxin A is known to be present in cereals, coffee, dried fruit and red wine. It is also known to be carcinogen to human beings. Meat and meat products can be contaminated with this toxin, too. The exposure to ochratoxins can cause an acute toxicity to the kidneys of mammals. The sterigmatocystin is produced by *Aspergillus versicolor,* which toxin solely appears on mouldy cheese crusts. It is possibly carcinogen for human beings.

The Aspergillosis in the brain may play a role in the development of the Alzheimer's disease. It is not yet known whether and which Aspergillus species cause the accumulation of the amyloidal fibers, produced in the brain lesiona characteristically found in patients suffering from Alzheimer's disease. It is also a question whether the pathogen produces the amyloid plaque, or whether the fungus is in reality the amyloid? These plaques accumulate to a neurotoxic level, compressing the nerve fibers that lie in their path, effectively destroying these regions of the brain. In progressed cases, these amyloidal fibers aggregate around the blood vessels and provoke a structural weakening in the vessels and a subsequent leakage of the blood serum into the cerebral space.
There are three clinical forms of aspergillosis:

7.2.8.1. Invasive Aspergillosis

This form can only occur in case of patients with a compromised immune system, those suffering from leukaemia, cystic fibrosis, HIV/AIDS, Chronic Obstructive Pulmonary Diseases (COPD), Chronic Granulomatous Diseases (CGD), Severe Asthma With Fungal Sensitivity (SAFS) etc. It also occurs in case of people with a suppressed immune defence mechanism, such as those having had chemotherapy, steroid therapy, heart transplants, liver transplants, or in case of elderly persons, whose immune responsiveness is lessened.
The infection spreads by breathing in a lot of *Aspergillus spores*, or if *Aspergillus species* being present on the skin or the mucosa, invades the deeper-lying tissues, f.i. the ear canals or the *lungs*. The primary infections affect mostly the *ear, the orbit and the nasal sinuses* and extend usually locally into the middle ear and the brain. The *Aspergillus esophagitis* is the most common form of the invasive aspergillosis and is more invasive than the Candida esophagitis. It causes ulcers and can extend into the muscularis tissue or can even perforate the mediastinum. Aspergillosis can spread also via the bloodstream to the *brain* and *the*

kidneys causing CNS aspergillosis or renal abscesses. It can be present as an opportunistic invasive infection concerning people with AIDS or Hodgkin's disease.
The Symptoms of these invasive forms include fever, chest pain, cough, shortness of breath, chills and, in severe cases, even shock, delirium and can cause blood clots, too. The aspergillosis of the ear canal causes itching and can be painful. The fluid discharge from the ear overnight may leave a stain on the pillow. In case of a localized bronchitis caused by Aspergillus, the symptoms are dyspnea, wheezing, cough and seldom even a mild hemoptysis. The infection of the deeper tissues can make a person very ill. Kidney failure, liver failure and severe breathing difficulties may develop. Death can set in quickly in case of persons with a more suppressed immune system.

7.2.8.2. Aspergilloma

The aspergilloma, looking like a ball, is composed of a tangled mass of fungal fibers, blood-clotting fibers and white blood cells. It gradually grows bigger and, in its processing, destroys the surrounding tissue. Aspergilloma can grow in the lungs and in other internal organs, too. Lung aspergillomas usually attack people suffering from other kinds of lung diseases, such as emphysema or tuberculosis.
Symptoms: aspergilloma in the lung may not cause any symptoms at all and can be discovered only by chest x-ray. This form of the infection may cause a chronic coughing up of blood and, in rare cases, severe, or even fatal bleedings.

7.2.8.3. Allergic Bronchopulmonary Aspergillosis (ABPA)

The most common causative species of this allergic illness are the *Aspergillus fumigatus* and the *Aspergillus clavatus*. The disease attacks mostly asthmatics owing to their hypersensitivity to Aspergillus antigens as well as children suffering from cystic fibrosis. The affected people are occasionally otherwise healthy. Though the fungus causes a respiratory illness, it does not invade and destroy tissues.
Symptoms: These allergic patients are subjects to recurrent episodes of migratory pulmonary infiltrations with blood eosinophilia, having symptoms of increased wheezing, cough, fever and pleuratic chest pain. The allergic process may lead to bronchiectasis and seldom to aspergilloma. Both, the reaginic antibodies and the precipitating antibodies are considered to be responsible for pulmonary alterations and systemic responses. Allergic aspergillosis can be the most frequent cause of lung infiltrates with eosinophilia. Allergic bronchopulmonary aspergillosis sometimes owns symptoms characteristic of a severe allergic asthma bronchiale. The chronic form of this disease persists over a long period of time.
Diagnosis: by the culturing of Aspergillus, by x-raying the lungs, by CT scan, by MRI.
Differential diagnosis: by distinguishing it from other fungal infections.
Treatment: by administering iv. Amphotericin B, Ketoconazole or Itraconazole. Some strains of the Aspergillus are resistant to these drugs; an alternative treatment has an important role.
RFR method: is needed if the Aspergillus is resistant to the conventional treatment, or if patients have cerebral infections.
RFR method: detects and may eliminate the Aspergillus.
The resonant frequencies of Aspergillus flavus are: 434-438, 464-468, 504 kHz
The resonant frequencies of Aspergillus glaucus are: 387-389, 534-539 kHz
The resonant frequencies of Aspergillus niger are: 350-359, 393-397 kHz
The resonant frequencies of Aspergillus terreus are: 344-348, 380-387 kHz
The resonant frequencies of other Aspergillus species are: 346-362, 364-376, 380-387, 390-395, 465-469, 502-506, 531-537 kHz
This list might not be complete yet. There are other subspecies with different resonance frequencies.

7.3. Human Pathogenic Budding Yeasts

The budding (true) yeasts are eukaryotic fungi, sorted mostly to the order of Saccharomycetales. Some species of the budding yeasts are opportunistic pathogens causing infections among people. The most common human pathogenic yeasts are the species of the *Candida genus*. The species of the *Geotrichum genus* and the *Malassezia genus* are less significant opportunistic human pathogens. A non-pathogenic yeast i.e. *Saccharomyces cerevisiae*, used when baking and brewing, may be worth mentioning, as anti saccharomyces cerevisiae antibodies (ASCA) are found often in case of familial Crohn's diseases and in some other types of colitis. The significance of this fact in the pathogenesis of these illnesses is not yet determined.

7.3.1. Candidiasis

The members of the Candida genus can often be present on objects such as on food, counter tops, air-conditioning vents, floors, respirators and even, as normal commensals of diseased skins and mucosal membranes of the gastrointestinal tract, the genitourinary tract and the respiratory tract. Such species of the Candida genus are *Candida albicans*, *Candida glabrata*, *Candida krusei*, *Candida parapsilosis*, *Candida guilliermondii* etc. In an immunocompromized state or in case of some defects of the host, these strains can become pathogenic, penetrating the mucosal membranes, causing irritation and shedding in it. Such defects of the host can be f.i. the injury of the mucocutaneous barrier, granulocytopenia, chronic granulomatous diseases, myeloperoxydase deficiency, hypocomplementaemia, hypogammaglobulinaemia, endokrinopathies, etc. In case of people with an impaired immune system candidiasis develops spreading throughout the body causing candidemia, especially in case of patients with low white blood cell count. The infection of the heart valves (fungal endocarditis) and of the lungs (pneumonitis) may result from this invasive spreading. The Candida species often cause skin diseases in case of immune-suppressed people.

The Symptoms of candidiasis vary depending on the tissue infected, and on the person's immune state.

If the *genitourinary tract* is affected the most common illness caused by the Candida species is the *vulvovaginitis* causing a moderate to severe itching and burning of the vulva and the vagina. The surrounding skin area appears red and can be raw. A thick, white cheesy discharge from the vagina is characteristic. These symptoms may worsen in the week before the menstrual period. The candidal vulvovaginitis is recurring in case of women suffering from an uncontrolled diabetes, or if they a receiving long-lasting antibiotic or corticosteroid treatment. In case of patients suffering from a severe diabetes mellitus the candidiasis may often be even fatal. Men, infected by candidal *balanitis*, feel itchiness of the penis, where lesions and whitish patches are present. In regard to patients with indwelling urethral catheters asymptomatic candiduria, *candidal cystitis* causing dysuria, haematuria, urgency and suprapubic pain can occur. In case of those suffering from diabetes using stents and indwelling devices, an *ascending pyelonephritis* can develop. Abdominal pain and cramps, nausea, vomiting, fever, chills and hematuria are the clinical signs of this infection. The candidal *kidney* infection can cause very low blood pressure and the decrease in the urine production, too. Fungal masses rarely act as fungus balls in the renal pelvis, they obstruct the ureter.

The most common *cutaneous candidiasis syndromes* are the *angular cheilitis*, which are erythematous painful fissures at the corners of the mouth; and the *intertrigo* appearing mostly on the skin of the genitofemoral region with pruritic red rashes, rupturing vesiculo-pustules followed by maceration and fissures. The *Candida folliculitis* occurs in the hair follicules of the head and in the seborrhoic regions of the skin. *Paronychia* and

onychomycosis caused by Candida species is usually associated with diabetes mellitus and the immersion of the hands in water. In case of the rarely occuring, *generalized, cutaneous candidiasis* a heavily pruritic, erythemato-vesiculosus rash disseminates at first from the genito-femoral and the anal region, then from the axillae, the hands, the feet and then all over the body.
If *the gastrointestinal tract* is infected, the Candida species causes either creamy-whitish, or erythematous painful, burning patches *in the mouth* on the tongue, the buccal mucosa, the throat and on the hard and soft palates. Such patches *in the esophagus* make it difficult for the immunesupprimated person to swallow or eat. Dysphagia, retrosternal pain, nausea and vomiting may also come about. Candida *peritonitis* and *gastric candidiasis* can also occur. The candidiasis of the digestive tract, as well as that of *the small and the large bowels*, can cause a pronounced digestive problem. Concerning patients with an underlying hematologic malignancy a *hepatosplenic candidiasis* may develop.
Candidiasis of the respiratory tract occurs only in case of immunocompromised patients. Laryngeal candidiasis, candidal tracheobronchitis and pneumonia are very rare types of the disseminated candidiasis.
The *disseminated candidiasis syndromes* occur mostly due to nosocomially acquired infections spreading by the bloodstream and resulting in candidemia. Such budding yeast infections usually develop regarding people whose immune system is impaired by some anticancer treatment or by diseases such as leukemia, lymphoma, multiple myeloma or AIDS, in which cases many candidal infections can be discerned solely by their symptoms. The candidal infection of the *heart valves* can cause fever and heart murmurs, while that of the *brain* encephalitis. The kidney and the brain bear the brunt of a hematogenous infection, but lesions can also occur in the thyroid, the myocardium, the endocardium, the pancreas, the adrenals and the liver. The visceral lesions are granulomatous nodules or abscesses containing both mycelia and yeast-like cells. Other invasive illnesses, such as hepatosplenic candidiasis with enlargement of the spleen can also be present. The candidal endophthalmitis is the sign of a widespread disseminated candidiasis. The candidal infection of the retina can cause blindness.
Diagnosis: for a definite diagnosis microscopic examinations of the fungi are necessary. The culturing of the samples of the urine, the mucous membrane, the skin and the blood or the spinal fluid may also reveal the presence of the Candida fungi.
Differential diagnosis: happens by the distinguishing it from other fungal infections.
Treatment: if candidiasis is to be found only in the mouth or in the vagina, antifungal drugs may be applied locally on the area and Fluconazole can be administered orally. Candidiasis, spreading throughout the body, is a severe, progressive and potentially fatal disease that has to be treated usually with intravenous Amphotericin B, though in some cases Fluconazole, Itraconazole or Ketoconazol can also be effective.
RFR method: detects and eliminates the candida, the first step however, is to administer antifungal drugs.
The most frequent resonances are: 293, 380-390, 443-453, 572-586 kHz
Its other resonance frequencies are: 295, 297, 332, 345, 352-359, 372, 380-390, 396-397, 403, 410, 440-453, 520, 554-559, 572-586 kHz
This list might not be complete yet; as there are other subspecies, too, with different resonance frequencies.

7.3.2. Geotrichosis

Geotrichum species are found in soil, water, air, as well as in plants, cereals and dairy products and even in the normal human flora, sputum and fece, too. As a normal inhabitant of the pharynx and the intestines, it may produce colonies on the mucous membranes. Geotrichosis is an opportunistic disorder regarding immunocompromised people. It affects the mouth, the bronchi and the intestinal tract, from where the *Geotrichum candidum* can

be isolated. This fungus has not yet been established as a human pathogen, the validity of geotrichosis as a disease entity remains still questionable. Though this fungus is not a primary pathogen, it may have a role in allergic and asthmatic processes of human beings.
The most frequent resonances are: 297-298, 351-358, 362-364, 377, 392-396, 412-432, 544-554 kHz

7.3.3. Infections Caused by Malassezia Species

Malassezia is a lipophilic yeast, commensal on skin and body surfaces of people and animals and may occasionally cause superficial or deeper mycoses.

7.3.3.1. Dandruff

This fungus, which is requiring fat for its growth, is found in areas containing many sebaceous glands thus on the scalp, the face and the upper parts of the body (chest and back). This rapidly growing yeast disturbs the natural renewal of the cells and dandruff will appear causing itching.

7.3.3.2. Tinea Versicolor (Pityriasis versicolor)

Tinea versicolor is a yeast infection of the *Malassezia* species (such as *Malassezia furfur*, *Malassezia globosa* and *Malassezia sympodialis*) that causes white to light brown patches on the skin. This infection is quite common, especially among young adults. The sometimes itching patches prevent these affected areas of the skin from tanning. People with a naturally dark skin might notice pale patches; while naturally fair-skinned ones may get dark patches. The patches are present mostly on the chest and the back and may scale slightly. Over time, the small areas can adjoin and form large patches.
Therapy: Dandruff shampoos, containing selenium sulfide, usually cure tinea versicolor. These shampoos are applied full strength to the affected areas at bedtime, left on overnight, and washed off in the morning. This treatment is to be usually continued for three or four nights. People, who get skin irritations caused by this treatment, may have to limit the time, when the shampoo is in contact with their skin, to 20-60 minutes, or they may need to turn to prescription medications.
Its resonant frequencies are: 311-317, 358, 453-459, 501-503 kHz
This list is night not be complette yet.

7.3.3.3. Seborrhoeic Dermatitis

These *Malassezia species* are widely present in seborrhoeic dermatitis occuring either among otherwise healthy persons or, more often, concerning patients immunocompromised by AIDS, diabetes mellitus, cancer, transplant etc., though the cause of this illness is still unknown.
RFR method: detects and eliminates the fungi.
The most frequent resonances are: 314-319, 452-480, 500-510, 558-560 kHz

7.4. Human Pathogenic Dermatophytes

There are three dermatophytes genera in the family of Arthridermataceae, such as the Epidermophyton genus, the Trichophyton genus and the Microsporum genus. For human beings pathogenic species of these genera can cause superficial and cutaneous mycosis, such as tinea corporis (ringworm), tinea cruris (jock itch), tinea pedis (athlete's foot) and tinea unguium (onychomycosis), a fungal infection of the nail bed. The most common species causing such fungal diseases among people are the *Epidermophyton floccosum*, the *Microsporum canis*, the *Trichophyton rubrum*, the *Trichophyton mentagrophytes var. interdigitale*. The natural reservoirs of the *Microsporum canis* are cats and dogs.

The ringworm, which is a fungal skin infection of the body, caused by some species of the *Trichophyton* genus or by *Epidermophyton floccosum* and *Microsporum canis*, has generally to be classified by its location on the body.

The body ringworm (Tinea corporis) is an infection of the skin caused mostly by Trichophyton species. The infection generally produces a pink to red rash that sometimes forms round patches with clear areas in their center. Body ringworm can develop anywhere on the skin.

The so-called athlete's foot (Tinea pedis) is a common fungal infection generally appearing in warm weather. It is usually caused either by Trichophyton or by Epidermophyton, which can grow in the warm, moist spaces between the toes. These fungi produce either a mild scaling without any other symptoms, or a more severe scaling with itchy, raw, painful rashes between the toes and on the skin of the feet. Fluid-filled blisters can also often be observed.

The jock itch, or the groin ringworm (Tinea inguinalis) is caused by a variety of fungi and yeasts. It affects commonly men, rather than women, and develops more frequently in warm and humid weather. This infection produces red, ring-like areas, sometimes with small, itchy and painful blisters in the skin around the groin and on the inner upper thighs as well.

The scalp ringworm is caused mostly by the species of the Trichophyton or the Microsporum. They may produce a red scaly rash that can be somewhat itchy, or may produce hairless patches without rashes.

The nail ringworm (Onychomycosis) is an infection of the nails caused mostly by the Trichophyton genus. The fungus invades the newly-forming parts of the nail, resulting in yellowish, thickened, lustreless and deformed nails. This infection is much more common on toenails than on fingernails.

The beard ringworm does only seldom occur. The skin infections in the beard area are caused either by bacteria or by fungi.

Treatment: by using antifungal creams, or taking systemic antifungal drugs.

RFR method: detects and eliminates the fungi.

The most common resonant frequencies in case of infection of the nail caused by Trichophyton species are: 288-309, 321, 370-374, 382-384, 384-399, 406-420, 429-440, 453, 474-480, 544 kHz

The resonant frequencies of Trichophyton mentagrophytes are: 318, 422 kHz

The resonant frequencies of Trichophyton rubrum are: 384, 472 kHz

The resonant frequencies of Trichophyton tonsurans are: 391, 464 kHz

The most frequent general frequencies of other Trichophyton species are: 308-310, 351, 413-415, 536 kHz

Their other resonance frequencies are: 293-305, 309-312, 321, 370-373, 380-400, 406-407, 410-418, 422-440, 453, 464, 536-540 kHz

Human Pathogenic Parasitic Infections

The parasite organisms live and do harm on or inside an other (host's) organism. Parasites infecting human beings include *protozoa* and *helminths*, i.e. worms. Protozoa reproduce by cell division inside the human body. Helminths produce eggs or larvae which, before becoming capable to infect people, develop in the environment. For their development in the environment they have to involve an other animal, which will act as an intermediate host. Some protozoa and some helminths have complex life cycles. Some are transmitted by insect vectors. Parasitic infections usually occur in the rural parts or in the developing areas of Africa, Asia and Latin America, but are to be found, less commonly, in industrialized areas as well.

8. PROTOZOAN DISEASES

The *most common protozoan diseases* are the *amebiasis*, the *malaria*, the *trypanosomiasis*, the *leishmaniasis* and the *toxoplasmosis*. They all remain to be one of the major causes of human sickness and death in the world today. The pathogens, responsible for these diseases, are unicellular possessing a true, or membrane-limited nucleus. The morphologic differences in the cytoplasmic organelles of locomotion are useful for the separation of protozoa, pathogenic to human beings, into four major groups: flagellates, ciliates, amebas and sporozoa. The structures, concerned with the motility of the first two groups, are self-evident. Amebas move by means of pseudopodia, while the sporozoa generally lack any specific locomotive structures. The flagellates, ciliates and amebas reproduce by asexual binary fission, while the sporozoa have alternating cycles of asexual and sexual reproduction. In the process of the asexual multiplication, the nucleus of the intracellular trophozoite first divides into several portions to form a schizont. Afterwards a cytoplasmic division will occur, resulting in the formation of daughter cells, i.e. merozoites. These invade the new host's cells in which they become trophozoites, completing thus the asexual cycle. After one or more such cycles some merozoites initiate the sexual phase of the reproduction by differentiation into male and female gametocytes. These mature and effect fertilization. The fertilized zygote, upon encysting, is named oocyst. Sporozoites, formed within the oocysts, get released, penetrate into tissue cells, and begin another asexual cycle as trophozoites. The way of transmission of these protozoa depends upon the fact, which certain area of the body they inhabit: f.i. in the human gastrointestinal tract or the genitourinary tract they pass from man to man either directly, as in case of *Trichomonas vaginalis*, or, indirectly, by the ingestion of contaminated food or water. In the former case the infecting agent is the vegetative form, i.e. the trophozoite; in the latter case, it is a cyst which is capable to survive in the external environment for prolonged periods.

8.1. Amebiasis (Entamoeba histolytica)

Amebiasis is an infection of the large intestine caused by *Entamoeba histolytica*, a single-celled parasite. The life cycle of the Entamoeba histolytica covers trophozoites (the feeding stage of the parasite) that live in the host's large intestine and cysts which pass in the host's feces. People are infected by ingesting cysts, most often via food or water contaminated with human fecal materials. Amebiasis is more likely to spread among institutionalized people under poor sanitary practices, and by sexual contact, particularly among male homosexuals. The indirect transmission of the cysts is more common in areas where sanitation is poor, f.i. in migrant labor camps. Fruit and vegetables may be contaminated if grown in soil fertilized by human stool, if washed in polluted water, or if prepared by someone who is infected. The trophozoites can destroy the tissues that line the host's large intestine. Of the species of amoebae, capable to get into the human gastrointestinal tract, the *Entamoeba coli*, the *Entamoeba dispar* and the *Entamoeba moshkovskii* are usually commensals, while the Entamoeba histolytica is potentially the most pathogenic.

The symptoms of infected people are in most cases intermittent and mild, including colitis, diarrhea, increased flatulence and cramping abdominal pain. Some may have a slight fever. The invasion of trophozoites in the appendix and the surrounding intestine may cause a mild form of appendicitis. The invasion towards the intestinal wall by trophozoites may cause a large granulomatous lump, the socalled *ameboma*. The ameboma may obstruct the intestine, and can be mistaken for cancer. In more severe cases *Amebic Dysentery* is going to develop, resulting in a severe diarrhea concomitant with mucus and blood in the feces. In some cases the trophozoites enter the circulatory system, infect other organs, most often the liver *(hepatic amoebiasis)*, or may penetrate into the gastrointestinal

tract, resulting in *acute peritonitis*; causing severe abdominal pain. Such cases require immediate medical attention and are often even fatal. Likewise an abcess, filled with trophozoites, may form in the liver. Symptoms include pain, or a feeling of discomfort in the area of the liver, intermittent fever, sweats, chills, nausea, vomiting, weakness, loss of weight, and, occasionally, a mild jaundice may develop. Trophozoites can spread through the bloodstream, seldom causing infection in the lungs, the brain and other organs.
Diagnosis: by demonstration and identification of cysts or trophozoites in stool samples, by serological tests, PCR.
Treatment by administering both tissue drugs such as Metronidazole, Tinidazole, and lumenal drugs such as Iodoquinol, Paromomycin, Diloxanide etc.
RFR method: detects and eliminates the amoebae.
The resonant frequencies are: 303-307, 315, 323, 339-340, 381-403, 488 kHz

8.2. Giardiasis (Giardia lamblia)

Giardiasis is an infection of the small intestine caused by *Giardia lamblia*, (synonyms *Giardia intestinalis*, *Giardia duodenalis*) a single-celled parasite. Giardia is a pear-shaped, multiflagellar protozoan that invades the human duodenum and jejunum, where it multiplies by longitudinal fission. It is actively motile but may attach itself to the intestinal mucosa by means of a large ventral sucker. Encystation occurs in transit through the colon. The resulting ovoid cysts are the infective forms of the parasite, transmitted by the fecal-oral route.
Giardia lamblia trophozoites live in the small intestine of the host. Cysts, which are resistant to adverse environmental conditions, are passed in the feces of an infected host, (one person can pass millions of G. lamblia cysts each day) and most infections probably result from the ingestion of water or food contaminated with human sewage. Open sewers in city streets and the contamination of drinking water with this sewage cause undoubtedly many infections. However, in some countries the use of human fecal material („night soil") as a fertilizer is also an important source of infection. The so-called „traveler's diarrhea" is also often caused by Giardia. In developing countries even the potable water can be contaminated with small amounts of sewage, especially when septic systems are built too close to wells. Every year many people suffering from giardiasis, return from camping trips but the source of these infections remains uncertain. Campers contract giardiasis mostly by drinking stream water contaminated with giardia cysts defecated by beavers (hence the term „beaver fever"). Some authorities, however, believe that these infections result from streams contaminated with human feces. This is why stream water should be boiled before drinking. Boiling will kill the Giardia cysts. There are commercially available filters to remove the cysts from water. Giardiasis occurs worldwide and is especially common among children and in places where sanitation is poor.
Giardiasis is **diagnosed** by finding cysts or trophozoites in the feces, as both life cycle stages have a characteristic appearance. The trophozoites average about 15 μm in length, have a distinct „tear-drop" shape and two nuclei at the anterior end. The characteristic shape of the trophozoite is particularly interesting when viewed with a scanning electron microscope. People, who observe these *Giardia lamblia* under the microscope, often say that it looks as if the trophozoites were „staring back at them". The trophozoites contain a dark transverse rod, the axostyle, which seems to be a supportive element. The cysts (average about 13 μm in length) are oval and contain two nuclei and remnants of the axostyle. Because of these unique characteristics, G. lamblia is one of the most easily diagnosable one of the intestinal protozoans infecting human beings.
Unlike the Entamoeba histolytica, which can invade the tissues of the large intestine, G. lamblia does not invade the tissues of the intestines. However, the trophozoites can adhere

closely to the lining of the small intestine. In case of heavy infections a great part of the lining of the small intestine can be covered with trophozoites.
The **symptoms,** associated with giardiasis, range from none (in case of infections) to *severe, chronic diarrhea* (in case of heavy infections), but not like dysentery. Children are three times more likely to be infected than adults, they probably experience more prominent clinical symptoms. The symptoms can include *intermittent nausea, belching, increased gas-formation, abdominal discomfort, bulky, foul-smelling stools and diarrhea,* though generally the infection is asymptomatic. The chronic giardiasis may lead to malabsorption of carbohydrates, fats and vitamins. In addition, *lactose intolerance* and disaccharidase deficiencies caused by giardiasis easily and often come about.
Diagnosis: by the microscopic examination of the stool, ELISA test.
Treatment: by administering tronidazole, Furazolidine, Quinacridine or Atabrine.
RFR method: detects and eliminates the parasite.
The resonant frequencies are: 340, 414-417, 425-427, 514-517 kHz

8.3. Malaria

Malaria is a protozoan infection of the red blood cells caused by *Plasmodium species*, a single-celled parasite, transmitted to human beings by the bite of an infected female Anopheles mosquito, by the transfusion of infected blood and even by an injection with a needle previously used by an infected person. This disease is widespread in tropical and subtropical regions, including parts of America, Asia and Africa. Four species of these parasites, *Plasmodium vivax, P. ovale, P. falciparum* and *P. malariae,* can infect people and cause malaria. The life cycle of a malarial parasite begins when a female mosquito bites a person suffering from malaria. The blood of the infected mosquitoes contains malarial parasites, which move to the mosquito's salivary glands. When the mosquito bites a person, the parasites are injected along with the mosquito's saliva. Inside the bloodstream of the person, the parasites move to the liver, where they multiply within the red blood cells, eventually causing the infected cells to rupture. *Plasmodium vivax* and *P. ovale* may remain in the liver cells periodically releasing mature parasites into the bloodstream, causing attacks of malarial symptoms. *P. falciparum* and *P. malariae* do not remain in the liver. If the infection is not or inadequately treated, the mature form of the *P. falciparum* may persist in the bloodstream for months, and that of *P.* the *malariae* may remain in the bloodstream for years, causing repeated attacks of malarial symptoms. *Plasmodium falciparum* invades red blood cells regardless of the patient's age, and may cause an extremely severe parasitemia. This infection is characterized by rigors, fever, splenomegaly, anemia, and has a chronic relapsing course.
The first **symptoms** are a mild fever that comes and goes, headache, muscle aches, chills and a feeling of illness. The first attacks may often be severe, but the repeated incidences become milder, though the debilitation may be progressive. In untreated cases, the attacks may persist for weeks. *Hepatomegaly*, mild *icterus* and *edema* can be often observed, especially in case of *P. falciparum* infections. Urticaria is common among patients suffering from chronic malaria.
In case of *P. falciparum* malaria, abnormal brain function may occur, that is a complication called *cerebral malaria.* Symptoms include fever, headache, drowsiness, delirium and confusion. Cerebral malaria can be fatal. It can lead to hemiplegia, convulsions, delirium, hyperpyrexia, coma, and death might rapidly set in. When the pulmonary circulation is involved, coughing may be experienced concomitant of bloodstreaked sputum, leading to the combination of many other diseases of the lung. In case of patients with predominantly gastrointestinal manifestations, usually a cold, clammy skin, hypotension, a profound weakness, and repeated syncopal attacks, the so-called algid malaria is diagnosable. A slight hepatomegaly, with or without jaundice and acute renal failure are also common. Pernicious syndromes should be anticipated if more than 5 percent of the red blood cells

are parasitized. The rupture of infected red blood cells releases hemoglobin into the bloodstream. The hemoglobin excreted into the urine turns the urine dark.
In case of *P. vivax* malaria, delirium may set in when the fever is high.
Sometimes malaria symptoms include *apathy, periodic fatigue and attacks of chills and fever*. See Chapter 19.1.4.1.
Diagnosis: the identifying of the parasites in a blood sample confirms the diagnosis.
Treatment: by administering Mefloquine, Doxycyclin and Pyrimethamine-sulfadoxine.
RFR method: detects and eliminates the parasite.
The resonant frequencies of Plasmodium falciparum are: 345, 372-388, 510-514, 521 kHz
The resonant frequencies of Plasmodium vivax are: 436-450 kHz
The resonant frequencies of Plasmodium cynomolgi are: 416-426 kHz
The resonant frequencies of other Plasmodium species are: 291, 310-330, 364-366, 378-380, 417-426, 464, 562-576 kHz

8.4. Toxoplasmosis

Toxoplasmosis is an infection caused by *Toxoplasma gondii*, a single-celled, obligate, intracellular protozoan parasite. Toxoplasma gondii has a very low host specificity, and it probably infects almost any mammals or birds and is found in every country of the world. Like most Apicomplexa, Toxoplasma is also an obligate intracellular parasite. Its life cycle includes two phases, the so-called intestinal (or enteroepithelial) phase and the extraintestinal phase. The intestinal phase develops only in cats, producing oocysts, while the extraintestinal phase regards every infected animal (including cats). In the latter case tachyzoites and, eventually, bradyzoites or zoitocysts are being produced. The disease toxoplasmosis can be transmitted by ingestion of oocysts (in cat feces) and bradyzoites (in raw or undercooked meat). In regard to most people infected with Toxoplasma, the disease is asymptomatic. However, in some conditions, toxoplasmosis can cause serious pathology, including hepatitis, pneumonia, blindness and severe neurological disorders. This is especially true concerning individuals having a compromised immune system (e.g. AIDS patients). Toxoplasmosis can also be transmitted transplacentally, leading to a spontaneous abortion, and be the cause of a stillborn child, or a child born mentally and/or physically severely handicapped.
The sexual reproduction of this parasite occurs only in the cells lining the intestines of cats. Eggs, or oocysts, are shed in the stool of a cat. People become infected by eating raw or undercooked meat containing the dormant form of the parasite or by being exposed to soil containing oocysts from cat feces.
Symptoms can include *enlarged,* usually not tender *lymph nodes* of the neck and of the armpits, a *feeling of illness, muscle pain, and a fluctuating,* eventually disappearing *low fever that can last for weeks or months*. The increased number of blood lymphocytes and the slightly abnormal results of the liver function tests can be some other signs of the infection. A mild lymphatic toxoplasmosis may resemble an infectious mononucleosis.
The chronic toxoplasmosis produces an inflammation of the eye. The acute disseminated toxoplasmosis can cause rashes, high fever, chills and extreme exhaustion. Concerning some people, the infection causes inflammation of the brain and of other organs, such as meningoencephalitis, hepatitis, pneumonitis, or myocarditis.
Regarding people suffering from AIDS the toxoplasmosis can spread throughout the whole body. In which cases encephalitis will often occur, paralyzing maybe the half of the body, and lead to severe mental retardation.
Connatally infected infants may be born prematurely or at term, be stillborn or alive suffering from fever, rashes, icterus, hepatomegaly, splenomegaly, chorioretinitis, convulsions and xanthochromic spinal fluid, in various combinations. Newborn infants may have none of the signs mentioned, though, subsequently, hydrocephaly or

microcephaly, chorioretinitis, psychomotor retardation, cerebral calcifications and convulsions may appear, either singly or in combination.
Disseminated toxoplasmosis has increasingly come to take its place alongside to the herpes viruses, cytomegalovirus, Epstein-Barr Virus, varicella, Pneumocystis carinii, and various fungi and bacteria as the frequent causes of death among those with profoundly debilitating diseases or who receive immunosuppressive treatment. In these instances toxoplasma most often affects the brain (diffused or localized), the myocardium and the lungs.
Diagnosis is usually made by blood tests that reveal antibodies against the parasite. CT and MRI can help to establish the diagnosis of a toxoplasmal process of the brain.
Treatment: by administering Spiramycin, Sulfadiazine, Pyrimethamine and Doxycyclin.
RFR method: detects and eliminates the protozoa.
The resonant frequencies are: 313-315, 390-400, 436-444, 500-502 kHz

8.5. Leishmaniasis (Sandfly Disease, Dum-Dum Fever, Kala azar)

Leishmaniasis (Baghdad boil, sandfly disease, Dum-Dum fever, Espundia) designates a human disorder produced by flagellated tissue protozoa of the genus Leishmania. It is transmitted from animal to human beings or sometimes from man to man by the bite of certain sandflies. Two sand fly genera, i.e. Lutzomyia in the New World and Phlebotomus in the Old World transmit Leishmania protozoa to human beings. Most forms of the disease can be transmitted solely by animals (zoonosis), some, however, can spread from human to human as well. Human infections can be caused by about 30 species of the Leishmania genus infecting mammals.The infection may be either visceral or cutaneous. The visceral form of the disease is named kala azar, and is characterized by chronic recurrent fever, splenomegaly, pancytopenia, loss of weight and high mortality. Cutaneous leishmaniasis may appear as single or multiple chronic skin ulcers, destructive mucocutaneous lesions, or as a disseminated infection resembling leprosy.
Leishmania species appear to be morphologically identical and must be differentiated on serologic, immunological and behavioral grounds, which are not entirely satisfactory. There are four main groups of this genus generally recognized: *Leishmania donovani*, *Leishmania tropica*, *Leishmania mexicana* and *Leishmania brasiliensis*.

8.5.1. Leishmania Donovani Complex

The Leishmania donovani complex with its three species *(such as Leishmania donovani, Leishmania infantum and Leishmania chagasi)* causes **kala azar**, the visceral form of leishmaniasis. Infecting generations, kala azar becomes endemic, assuming a more chronic form. The domestic dog becomes an important reservoir of the parasite in China, Russia, India, Egypt, Sudan, East Africa, Greece, Crete, Malta and several other countries.
Due to the increased international leisure and military-related travels the total incidence of this infectious disease increased recently. Owing to the alteration of the habitat of its vector and caused by some concomitant factors, such as *HIV infection* and malnutrition the susceptibility to this infection has increased. The coexistence of leishmaniasis with *HIV* and *other HTLV* infections is a serious danger to mankind. Leishmaniasis is spreading in several areas of the world due to the rapidly spreading epidemic of AIDS so that the co-infection with HIV has lead this typically rural disease into urban areas. Leishmaniasis can accelerate the onset of AIDS in HIV patients by a cumulative immunosuppression and by stimulating the replication of the virus. More over, HIV can change asymptomatic Leishmania infections into symptomatic ones. The sharing of needles by intravenous drug users can spread not only HIV but also leishmaniasis.
Mycoplasmal infections do also promote the spreading of Leishmaniasis. In this case the most important immunological feature is the developed marked suppression of the cell-

mediated immunity to leishmanial and mycoplasmal antigens. T-helper cells predominate in persons with asymptomatic self-resolving infection, but years later a symptomatic disease will develop due to the immune suppressive effect of *mycoplasma*. An overproduction of specific and nonspecific immunoglobulins does also occur. This increased amount of gamma globulin leads to the reversal of the albumin-globulin ratio commonly associated with this illness.
Leishmaniasis is a disease involving the reticuloendothelial system. These species multiply extensively in the macrophages of the spleen, the liver, the bone marrow, the lymph nodes, the skin and the small intestines accounting for many of the manifestations of the disease.
Symptoms: include fever, progressive weakness, pallor, loss of weight and tachycardia. Physical findings include enormous splenomegaly, lymhadenopathy, hepatomegaly with signs of portal hypertension, and often edema as well. Hyperpigmentation is noted among light-skinned, infected individuals.

8.5.2. Leishmania Tropica Complex

The Leishmania tropica complex *(Leishmania tropica, L. major and L. aetiopica)* causes cutaneous leishmaniasis. The *L. aethiopica species* can cause oriental sores and diffuse cutaneous leishmaniasis in Ethiopia.
Symptoms: The healing of the indolent, granulomatous ulcer may require a year or more. Occasionally it does not heal, resulting in lesions closely resembling lupus vulgaris.

8.5.3. Leishmania Mexicana Complex

The Leishmania mexicana complex *(Leishmania amazonensis* and *Leishmania venezuelensis)* produces cutaneous leishmaniasis. The *Leishmania brasiliensis* is the cause of the *American mucocutaneous leishmaniasis*, also known as **espundia**.
Symptoms: Fever, anemia, and loss of weight accompany the mucosal complications. The destruction of the nasal septum produces a characteristic deformity called tapir nose or camel nose.
Each complex contains a variety of strains which have been recorded asseparate species or subspecies with different resonance frequencies.
Diagnosis: by buffy-coat preparations of peripheral blood or aspirates from marrow, spleen, lymph nodes and by complement tests
Differential diagnosis: Mycobacterium phlei
Treatment: by admininstering Amphotericin B, Doxycyclin, miltefosine and paromomycin.
RFR method: detects and eliminates the Leismania.
The resonant frequencies of L. donovani are: 318, 398-404, 506-508, 536-537 kHz
The resonant frequencies of L. brasiliensis are: 321, 400-407, 510, 538 kHz
The resonant frequencies of L. tropica are: 322, 402-410, 513, 540 kHz
The resonant frequencies of L. mexicana are: 320, 400-410, 508-511, 534-544 kHz
Antibiotics Resistant Leishmaniasis (ARL)
The often administered antibiotic therapies resulted in the development of **new, antibiotics resistant species of Leishmania.** There are more and more antibiotic derivates which have no effect on these species anymore. Indian researchers identified a certain mechanism of the drug resistance developing in the parasites causing visceral leishmaniasis and they suggested an effective way to reverse it. They identified in these resistant parasites an enzyme named trypanothione reductase aswell as a gene, MRPA (multidrug resistance protein A). The development of a method to create certain defined *mutants* of Leishmania parasites lacking genes which confer the resistance to antibiotics shows experimental and practical benefits. ARL mutants, deficient in specific virulence genes, are potential attenuated live vaccines, though this fact can only be of clinical relevance if the antibiotic resistance genes used for the selection of the mutants are subsequently removed. In

addition, the limited number of antibiotic resistance genes that can be used for genetic manipulation of Leishmania means that a system for recycling them for subsequent use would be highly beneficial if multiple genetic modifications are wanted.
Drug-resistant leishmaniasis may respond to *immunotherapy* (inoculation of parasite antigens together with an adjuvant) aiming the stimulation of the body's own immune system to kill the parasite. Upto now, the potential vaccines developed to prevent visceral Leishmania can still be deadly.
I think, that RFR method has a good eliminative effect on the drug-resistant Leishmania protozoa.
The most frequent resonances of ARL are: 318-325, 398-407, 507-515, 537-539 kHz
The most frequent resonances of Mycoplasma fermentans are: 442-451 kHz
Several different resonant frequencies of *HTLV* are also frequently present in case of this disease. As to the frequencies, see their special Chapter.

8.6. Trypanosomiasis

Trypanosoma, a genus of protozoa with many species belongs to the Trypanosomatidae family and is a parasitic of the blood, the lymph and other tissues of invertebrates and vertebrates, including human beings. Most trypanosoma species live in one part of their life cycle in the intestines of insects and other invertebrates, but in their flagellate stage they can only be found in vertebrate hosts. Some species cause serious diseases in domestic animals. Some species of *minor pathogenicity* are f.i. the T. avium (in birds), T. binneyi (in platypus), T. calmetti (in ducklings), T. diazi (in capuchin monkeys), T. dimorphon (mostly in domestic animals), T. gallinarum (in fowls), T. melophagium (in sheep), T. minasense (in monkeys, e.g. marmosets), T. nabiasi (in rabbits), T. primatum (in chimpanzees, gorillas), T. rangeli (in human beings, dogs, cats), T. ariarii, T. guatemalense, T. saimiriae (in squirrel monkeys), T. sanmartini (in squirrel monkeys), T. theodori (in pigs) T. equiperdum (in horses), T. lewisi (in rats) etc.
The resonant frequencies are: 320-323, 358, 368-372, 412-413, 520-522 kHz
This list is not complet.

8.6.1. Trypanosomiasis in Africa (Sleeping sickness)

The most serious Trypanosoma species, transmitted by the saliva of tsetse flies cause *nagana* (named also Animal African Trypanosomiasis), which is the sleeping sickness of cattles, goats, sheep, horses, dogs and cats. The serious human trypanosomiasis caused by the subspecies of *Trypanosoma brucei* is the socalled sleeping sickness occuring in some areas of Africa. The transmitter of these T. brucei species is the saliva of tsetse flies, while the reservoir hosts of T. brucei are wild ruminants. The subspecies *Trypanosoma brucei gambiense (syn. , Trypanosoma hominis, Trypanosoma nigeriense, Trypanosoma ugandense)* cause chronic diseases among people and in cattles and goats, as well. The subspecies *Trypanosoma brucei rhodesiense* cause a serious disease in case of human beings but only mild ones if affecting ruminants, other domestic animals and monkeys (see sleeping sickness also in Chapter 10.28.4.).
The resonant frequencies are: 347-357, 370, 408-415, 518-526 kHz

8.6.2. Trypanosomiasis in America (Chagas disease)

An other human trypanosomiasis is the American trypanosomiasis (otherwise named Chagas disease) caused by the flagellate protozoa *Trypanosoma cruzi* (syn. *Trypanosoma escomeli*), the reservoirs of which are pigs, mice, rats, guinea pigs, rabbits dogs and cats as well as many wild animals. This protozoon causes disease among the above mentioned hosts and may prove fatal to dogs. *Trypanosoma cruzi* is the only trypanosoma species which is transmitted by the feces of adult and nymphal invertebrate, blood sucking, insect

vectors, f.i. Triatoma infestans, Rhodnius prolixus and Triatoma dimidiata. The particularly frequent oral transmission occur by eating food contaminated by feces of infected insects. The transmission by skin penetration can occur via the bite or the microlesions caused by scratching. The pathological processes of the organs (f.i. the heart, the oesophagus and the colon) caused by this parasite are sequentially induced inflammatory responses, cellular lesions and fibrosis as well. Myocytolysis of the myocytes and autonom denervations of the nervous cells are the most often cellular lesions caused by this intracellular protozoa.

The Symptoms: vary depending on the phase of the illness. The early stage is usually asymptomatic, and is only a local swelling at the place of the infection. Seldom Lymphadenopathy, hepatospenomegaly and myocarditis does occur but rarely. In the chronic stage, there develop, mostly years (even 10-20 years) after the inoculation, characteristic heart symptoms, f.i. arrhythmic syndrome, hypertrophy of the heart muscles, dilatative cardiomyopathy, apical aneurism, etc. If left untreated, the cardiomyopathy can be fatal. Systemic and pulmonary thromboembolism are frequent complications of the Chagas disease. Dilatation of the digestive tract and malnutrition are less common symptoms of the disease.

Diagnosis: the definitive diagnosis depends upon the finding of trypanosomes in the blood, and in the aspirate of the lymph nodes. By blood films with Giemsa's stain.

Prevention: Fighting the vector by using insecticides

Treatment: in case of Chagas disease by administering benznidazol, nifurtimox, amphotericin B and symptomatic.

RFR method: detects and may eliminate the trypanosoma!

The resonant frequencies of T. cruzi are: 460-465 kHz

The resonant frequencies of T. brucei brucei: 423-431 kHz

The resonant frequencies of T. brucei gambiense are: 321-326, 352-359, 368-375, 393-398, 410-416, 522 kHz

The resonant frequencies of T. brucei rhodesiense are: 423-428 kHz

The resonant frequencies of T. equiperdum are: 434-451 kHz

The resonant frequencies of T. lewisi are: 424-426 kHz

The frequencies of some Trypanosoma species unidentified yet are: 300-340, 356-372, 392-420, 520-560 kHz

The frequencies of antibiotic resistent groups may be increased up to 1-10 kHz

Use the RFR method with antibiotics!

In the chronic form of trypanosomiasis may co-infections be present, the pathogens of which may inhibit the host's immune system and immune response.

8.7. Babesiosis

Babesiosis is a parasitic infection of the red blood cells caused by *Babesia bigemina, B. canis* and *B. microti.* The members of the Babesia genus belong to a group of the Apicomplexa referred to as „piroplasms". The Piroplasms have two life cycles involving ticks and mammals as their hosts. In the mammalian hosts the microorganisms reproduce themselves asexually in the host's red blood cells. The hard-bodied ticks (the same deer ticks and Ixodes ricinus ticks, which transmit the Lyme disease) transmit Babesia parasites. Although the infection of animals is common, human beings are rarely infected. This disease is a typical disease of hunters. The vector for *Babesia bigemina* is a hard tick (Boophilus sp.), the parasite infects a variety of ruminants. In cattle this parasite causes a disease known as Texas cattle fever or red-water fever. The parasite often occurs in pairs in the host's red blood cells, hence the name bigemina. These parasites can cause massive destruction of the red blood cells, resulting in red urine (hemoglobin compounds in the urine). The disease can kill cattles within a week. Similar species occur in dogs (B. canis) and rodents (B. microti).

Symptoms include low fever and anemia, caused by the breakdown of the red blood cells. In case of patients, whose spleen had been removed, the risk of death is high. Concerning these patients, the infection closely resembles falciparum malaria, producing high fever, anemia, hemoglobin in the urine, jaundice and kidney failure. Patients with functioning spleens get a milder form of the illness, usually healing on its own within weeks or months.
Diagnosis: is made by blood smear with Giemsa staining and by laboratory tests to identify the parasites.
Differential diagnosis: by differentiating it from Malaria, Rickettsial infections, and from infections caused by Eperythrozoon species.
Treatment: by administering Clindamycin.
RFR method: detects and may eliminate Babesia.
The resonant frequencies are: 291, 311, 369-371, 385, 405-406, 442 kHz

8.8. Sarcosporidiosis (Sarcocystis)

Sarcosporidiosis is a disease caused by Sarcocystis, an intracellular protozoan parasite, which predominantly affects animals. This protozoan parazite can rarely be found in human skeletal and cardiac muscle as the patients are only intermediate or accidental hosts. However, after ingesting cysts in raw or undercooked beef or pork, human beings can also serve as definitive host for this parasite. After the invasion of the gastrointestinal tract, the infective sporozoites replicate themselves and are subsequently eliminated in the stool. These oocysts, when completing their life cycle, are ingested by intermediate hosts (usually cows or pigs). Most of the sarcosporidiosis cases attacking people occur in Southeast Asia, Malaysia and the USA. The disease has two distinct forms. In the first form, the ingestion of water or food contaminated with sporocysts from the feces of a carnivore (e.g. a dog or a wolf) is followed by the penetration of the sporocysts into the intestinal wall. Their proliferation in the vascular endothelium and their subsequent hematogenous dissemination leads to the invasion of the skeletal and cardiac muscles. These sporocysts subsequently disintegrate with an accompanying vasculitis and fibrosis of the tissue (myositis). The second form of the sarcosporidiosis in case of human infection occurs after ingesting meat contaminated by infective oocysts. The oocysts undergo a sexual reproduction and maturation in the intestinal tract being then shed into the stool (enteritis). Systemic phases and subsequent tissue phases do not occur in this form of the infection. Histopathologic findings show inflammatory cells, intense eosinophilic infiltrate surrounding the muscle cyst and localized vasculitis with the fibrosis of the muscle.
The symptoms: include *in case of the myositic form* of sarcosporidiosis painful muscle swellings accompanied by erythema, muscle tenderness, generalized muscle weakness and fever. Bronchospasm can also occur. The cardiac involvement is asymptomatic, though sarcosporidiosis can cause an atrioventricular block ofsecond-degree, in case of sheep. Within a day after ingesting contaminated beef or pork, people who develop *the enteritis form* of this infection get diaphoresis, chills, fever, nausea and diarrhea. Dehydration and diffuse abdominal tenderness occur in case of patients ingesting oocysts.
Diagnosis and differential diagnosis: In the intestinal form sporulated sporocysts can be found by flotation technique in freshly voided stool. The sporocysts contain 4 sporozoites. There is eosinophilia present in the blood smear. In cases of myositis the creatine kinase levels of the serum may be elevated. By using PCR techniques a Toxoplasma gondii infection can be excluded. A muscle biopsy is useful by diagnosing myositis.
Treatment: if administering Metronidazole and cotrimoxazole there is no specific outcome known. Corticosteroids can reduce the inflammation associated with the muscular involvement.
RFR method: detects and eliminates the pathogens. RFR method should be used simultaneously with the antibiotic treatment.
Its resonant frequencies are: 359-361, 379-380, 447-456 kHz

8.9. Trichomoniasis

This common, sexually transmitted disease is caused by the single-celled protozoan parasite *Trichomonas vaginalis*. This protozoon infects primarily the urethra and the vagina and the tip of the penis of uncircumcised men.

The **Symptoms** generally appear 4 to 20 days after being infected. A profuse, yellow-green or gray vaginal discharge, an unpleasant vaginal odor, vulvovaginal itching and discomfort are characteristic. Vulvovaginal swelling and a feeling of discomfort during the sexual intercourse often come about. Painful urination as well as abdominal pain can also be present. The complication of the infections in case of pregnant women may be the cause of premature birth, or small for date infants. In case of men the symptoms are rare, but if prsent, it includes a pale, white discharge from the penis and a painful or difficult urination. Prostatitis and cystitis are the most common complications.

Diagnosis: by microscopic examination of the vaginal fluid, Pap smear and urinanalysis in case of women or by the culturing a sample of the discharge.

Treatment: by administering metronidazole or tinidazole also for the sexual partner.

RFR method: detect and eliminate.

Its resonant frequencies are: 312, 321, 354, 375-388, 500-503 kHz

8.10. Dientamoebiasis

Dientamoeba fragilis is a nonflagellate trichomonad parasite, one of the smallest parasites that can live in the human large intestine. The infection among people occurs if they are in their trophozoite stage. The diameter of this pleomorphic trophozoite ranges from 5-15 mm. The way of transmission is believed to be through direct fecal-oral spread and through coinfection of the eggs of *Enterobius vermicularis* (i.e. pinworm). *Dientamoeba fragilis* species can cause diseases among human beings regardless of their immune state. The most often infected persons are children aged from 5-10 years.

Symptoms: abdominal pain and diarrhea (1-4 stools per d) are the main signs of the infection. Nausea, vomiting, loss of weight, flatulence, headache, fatigue, nervosity, pruritus or urticaria can also come about.

Diagnosis: by the examination of immediately preserved smear of fresh, stained feces, and by the detecting of the *D fragilis* trophozoites. (The immediate preservation is necessary as, the morphologic characteristics of the trophozoites do not persist in unpreserved feces, they become granular at room temperature within 15 minutes.)

Treatment: by administering Metronidazole, Iodoquinol, etc.

RFR method: detects and may eliminate the parasites.

The most frequent resonances are: 318-323, 401-406, 510-513 kHz

8.11. Cyclosporiasis

Cyclospora cayetanensis, a coccidian protozoan parasite, can cause intestinal infections, the socalled „traveller's diarrhea". Cyclospora species are ubiquitous, they infect various animals, including vipers, moles, rodents and myriapods. People are the only known hosts of *C. cayetanensis* as yet. Cyclospora is endemic in Bangladesh, Brazil, Chile, China, Cuba, the Dominican Republic, Egypt, Guatemala, Haiti, India, Indonesia, Jordan, Mexico, Morocco, Nepal, Nigeria, Pakistan, Peru, Puerto Rico, Romania, Saudi Arabia, Tanzania, Thailand, Turkey, Venezuela, Viet Nam, Zimbabwe. Ilnesses caused by Cyclosporiasis can occur seasonally in Guatemala (from May to August), in Haiti (from January to March or April), in Nepal (from May to August) and in Peru (from December to May), often disappearing at times for months. In several countries Cyclospora species has been found encountered in source waters as well.

The human infection can occur by ingesting sporulated oocysts of *C. cayetanensis*. The oocyst excysts in the small intestines, usually in the jejunum, invading the intestinal epithelial cells. The next process is schizogony, beginning with the formation of trophozoites which grow into a mature schizont containing 8-12 merozoites. These latter then are released, presumably by cell ruptures, and invade other epithelial cells repeating the whole process. These merozoites are named type I meronts, which are asexual forms.
After several cycles of type I schizogony, type II meronts, the sexual forms will develop, in this form each cell contains 4 merozoites. After invading the epithelial cells, some of these then form single macrogametes, others are dividing multiple times, forming microgametes. If released, a microgamete fertilizes a macrogamete, which develops into a zygote. The zygote, in turn, develops into an oocyst having an environmentally resistant wall. The oocyst passes into the environment in the feces as a nonsporulated noninfectious oocyst. In such a way, human-to-human transmission does not occur. The oocysts are continuously excreted during the infection. Getting in the environment, the oocysts sporulate, becoming infectious for human beings. During the sporulation, sporonts divide into 2 sporocysts, each containing 2 sporozoites. The course of time of being in the environment lasts from days to weeks. Contamination of food or that of the drinking water can lead to human ingestion and infection. Cyclospora undergo both sexual and asexual reproduction. They appear microscopically as nonrefractile, double-walled spheres 8-10 μm in diameter. Some of them resist staining.
Symptoms: are characterized by cyclical diarrhea, accompanied by fatigue, malaise, anorexia, nausea, loss of weight and abdominal cramps interspersed with periods of remission. Low-grade fevers and malabsorption may come about. If left untreated, the diarrhea may continue for weeks to months. Cyclospora infection affects both immunocompetent and immunocompromised individuals as well.
Diagnosis: Cyclospora oocysts are difficult to identify by microscopic examination (high dry, 400X) without special techniques: acid-fast staining, safranin staining, direct wet smear examined by using a fluorescent microscope or by using a differential interference contrast microscope, as well as by lacto-phenol cotton blue staining. By PCR.
No serologic tests are currently available to detect antibodies to Cyclospora species.
Prevention: by avoiding untreated, contaminated water and unpeeled fruits and vegetables, even when traveling.
Treatment: Trimethoprim-sulfamethoxazole has proven bo be effective by the treatment of Cyclospora infections in case of immunocompetent and immunocompromised hosts as well. Norfloxacin, metronidazole, tinidazole, quinacrine and azithromycin are also effective.
RFR method: detects and may eliminate the parasite. It is advised to combine RFR method with the trimethoprim-sulfamethoxazole therapy.
The most frequent resonances of the Cyclospora species are: 322-325, 360-362, 507-509, 547-556 kHz
Immunocompromised hosts require the oral antibiotic therapy for a longer time, followed by prophylaxis to prevent recurrence. It is necessary to examine the cause of the immunocompromised state and to treat the eventually found other pathogens, too.

9. HUMAN PATHOGENIC HELMINTHS (WORMS)

The human pathogenic helminthic parasites are sorted into three major groups; roundworms (nematodes), tapeworms (cestodes) and flukes (trematodes). The helminths are large, multicellular organisms having excretory, nervous and reproductive systems. The trematodes are the most highly differentiated helminths, possessing fully developed male and female sexual organs capable of producing an enormous number of offsprings in form of eggs and larvae. The differences in the life cycles of worms have a determinative influence on the epidemiology of helminths.

The **pathogenesis** of helminthic diseases is, as relating to their life cycles, variable. The *Diphyllobothrium latum* species competes with the host for nutriments. *Strongyloides stercoralis* and *Capillaria* species interfere with the absorption of food across the intestinal mucosa. Hookworms cause loss of iron, that essential mineral. The flukes such as *Clonorchis* and *Schistosoma* compromise the function of important organs by obstructing and provoking secondary bacterial and viral infections. These helminthic parasites often suppress the immune function of the host, their long-term infections can also be carcinogenic. A disease can result from a simple mass effect too, as can be observed in case of echinococcosis. An actual tissue invasion and destruction by larval forms occur in case of many helminthic infections. Immunological mechanisms are undoubtedly also responsible for the tissue damages and for the clinical manifestations regarding many helminthic diseases. The eosinophilia in the blood presumably reflects the immune response of the human body to the complex foreign proteins of the worm and is even more marked in the early stage of the migration and the invasion of parasites into the tissue. Once the migration ends and the worm matures to adulthood, the eosinophilia may diminish or disappear.

The **diagnosis** of worm infections present a difficult problem, their examination produces many false-negative results. Although eosinophilia has already long been recognized as a fact of the presence of helminthic infections, the failure of eosinophilia does not exclude this diagnosis.

The definitive diagnosis usually rests upon the recovery and the morphologic identification of the parasite in stool, urine, sputum, blood, or tissues. Helminths being antigenically rather complex, the serological tests and the skin tests are much less reliable than in case of microbes.

Common instructions concerning the RFR method of the worms: The adult worms must first of all be treated with antihelminthic drugs, if needed, even repeatedly until they are killed. After eliminating the adult worms, the RFR method can eliminate all the other life-cycle forms, such as the cysts, the larvae, etc. Without this adviced method the hypermotility of the adult worms might cause complications, f.i. the adult ascaris can climb into the ductus hepatopancreaticus and by closing it cause cramps and icterus. The worms in the brain for instance will move more intensively if treated with radiofrequency causing thus seizures, swellings and epileptiform cramps. In case of cysticercosis of the brain neither antiparasitic treatment, nor RFR method is recommended.

Worms as Virus Carriers: The worms are able to be the reservoir of viruses and thus may carry different viruses into their host's organism; likewise a parasitic infection can be an indirect cause of an additional viral infection. The type of the virus species seems to be specific to the carrier worm. For example, a Eurytrema species often carries Epstein-Barr viruses, while an Ascaris species is frequently a Herpes Zoster Virus carrier. This means that the parasite may originally be infected with a virus, and that a helminthic infection of people may, likewise cause a simultaneous viral infection.

9.1. Human Intestinal Roundworm Infections

The nematodes or roundworms are unsegmented, bilaterally symmetric, triploblastic protostomes with a complete digestive system. Most free-living nematodes are microscopic, though a few parasitic forms can grow even to more than one meter in length. The nematodes commonly parasitic on human beings include ascarids, pinworms, hookworms, whipworms, trichina worms and filarids.

9.1.1. Ascariasis (Ascaris lumbricoides)

Ascariasis is a human intestinal roundworm infection caused by *Ascaris lumbricoides*. This worm is one of the largest and most common parasites found in human beings. The adult females of this species can grow to 12-18 inches i.e. cc. 30-45 cm (its males are generally shorter), it is estimated that 25% of the world's population is infected with this nematode. The adult worms live in the small intestine, their eggs pass in the feces. One single female Ascaris worm can produce up to 200000 eggs per day! People are infected by ingesting infective eggs from with human feces contaminated soil, vegetables and water. About two weeks after the eggs pass in the feces, they undergo an infective larval or juvenile stage. The eggs become hatched and the juvenile worms penetrate the small intestine, enter the circulatory system, or may be carried by the lymphatic vessels or the blood vessels eventually reaching and getting into the lungs. There, they pass into the air sacs, (alveoli), ascend up to the respiratory tract into the pharynx where they are being swallowed. After which the larvae mature in the small intestine, growing into adult worms. Just why *Ascaris lumbricoides* undergoes such a migration through the body only to end up where it started is unknown. Such a migration is not unique concerning the Ascaris genus, as its close relatives undergo a similar migration in the body of their hosts.

Human ascariasis can cause significant pathologies. The migration of the larvae through the lungs causes haemorrhage in the blood vessels of the lungs causing there inflammation and edema. This accumulation of fluids in the lungs results in pneumonia, which can be fatal. The large size of the adult worms is also problematic, especially if the worms physically block the gastrointestinal tract. Ascaris is notorious for its migration within the small intestine, if a large worm begins to migrate there is nothing to stop it. Ascaris can migrate into the bile duct or the pancreatic duct blocking them, or they can perforate the small intestine which results in an acute (and fatal) peritonitis. Ascaris species seems to be especially sensitive to anesthetics, there are documented cases telling about patients in surgical recovery rooms, who had worms which migrated from their small intestine into their stomach, to pass out, eventually, through the patient's nose or mouth.

The life cycle of *Ascaris suum* species, pathogen in pigs, is identical to that of A. lumbricoides. If a human being ingests eggs of *A. suum*, the larvae will migrate to the lungs and die. This can cause a particularly serious form of ascaris pneumonia. Adult worms of this species do not develop in the human intestine. Domestic pets often get Ascaris infections, so that children are not allowed to clean up any vomit or mess left by an animal.

Ascaris lumbricoides has an important antigen which can cause an allergic sensitivity to histamine. At this stage this special sensitivity may increase as high as thousand fold. Ascaris can provoke allergic sensitivity in the host so that it leads in certain circumstances to respiratory problems f.i. to allergic asthma. The cause of this kind of allergic asthma is a combination of ascaris antigen (or other worm antigens) and of microorganisms with pollutants. Asthma patients are often allergic to air pollutants, such as pollen, animal antigens and fungal antigens, smoke, etc. The production of histamine in the lungs plays a leading role in the development of allergic asthma. The invasion of the lungs by Ascaris results in a higher histamine production as well as in high eosinophil counts. As asthma patients have usually both, they have to be routinarily checked for worms.

The Symptoms of ascariasis, characterized by fever, coughing and wheezing, are caused by the migration of larvae through the lungs. A heavy intestinal infection may cause abdominal cramps and, occasionally, intestinal obstruction. The malabsorption of nutrients may be caused by a great amount of worms. Adult worms occasionally obstruct the appendix, the biliary tract, or the pancreatic duct. The infection caused by adult worms is usually diagnosed by identifying eggs in a sample of the stool. Occasionally, eosinophilia in blood tests reveal adult worms in the stool, or in the 0vomit, or larvae in the sputum.
Prevention requires using adequate sanitation and the avoidance of unwashed vegetables. Cats, dogs and other domestic animals infected with ascaris must be promptly treated to eliminate the parasite.
Treatment by administering pyrantel pamoate, or mebendazole. However, mebendazole cannot be taken by pregnant women because of its potentially harmful effects on the fetus. The treatment must be repeated until all life cycles of the worm have been eradicated.
RFR method: the first step is the administering antihelminthic drugs, followed by mebendazole treatment, which eliminates the ascaris larvae in the tissues.
Its resonant frequencies are: 308, 384, 402-410, 452, 584-590 kHz
The general range of the Ascaris species is: 402-410 kHz

9.1.2. Enterobiasis

Enterobiasis (also known as Seatworm infection, Threadworm infection or Oxyuriasis) is a disease caused by pinworms, such as the human pathogenic *Enterobius vermicularis*. This small roundworm grows and reproduces within the intestines itself. Pinworms infect more than 10% of human beings in many a country of the world (especially North America and Europe). It is the most common human pathogenic nematode parasite. The adult pinworms live in the large intestines; the males and the females are about 5 and 10 mm long, respectively. After copulation the males die. When the female is ready to lay eggs, she crawls out of the anus and deposits more than 10 000 eggs on the perianal skin during the early morning hours. Having layed eggs, the female also dies. The eggs are deposited in a sticky, gelatinous substance. This substance and the movements of the mother pinworm cause itching. The quickly hatched worms develop at body temperature in about six hours and become infective juvenile worms which can migrate back to the rectum and into the lower intestine. When eggs are ingested by a person, they hatch in the small intestine, the juvenile worms grow into adult, sexually mature worms in about a month. Pinworms are highly contagious. Bed linens, clothing, carpets, etc. can be contaminated with eggs. The infected person's hands can, invariably, be contaminated with eggs, providing a route for reinfection and dispersion of eggs. For this reason, if one member of a family is infected by pinworms, the whole family has to be treated. Pinworms are the most common parasites infecting children living in temperate climates.
The *Dientamoeba fragilis*, a protozoan human parasite, can by being in the eggs of pinworms also be transmitted to people. In this way, pinworm infections, as well as Dientamoeba infections may occur simultaneously.
Symptoms: The pinworm infections can be asymptomatic, can result in mild gastrointestinal upsets, most often causing a perianal itching and sleeping disturbances. The scratching of the perianal skin can lead to bacterial infections resulting in more violent itching. This circulus vitiosus can result in extreme discomfort. Children infected with pinworms often undergo changes in their behaviour, including restlessness, irritability and insomnia. Pinworms blocking the appendix may sometimes cause appendicitis, too. In case of girls, pinworm infection can cause vaginal itching and irritation.
Diagnosis by finding the eggs or worms obtained by patting in the early morning the skin folds around the anus with the sticky side of a strip of transparent tape, before the child wakes up. The eggs and worms on the tape can be identified under microscope. Eosinophilia in the blood smear is the sign of the antihelminthic immune response.

Treatment: by administering Mebendazole and Pyranten. Despite the drug therapy, reinfection is common even after treatment, as the live eggs continue to shed +in the feces for even up to a week after treatment. Clothing, bedding, and toys should be machine-washed frequently to eliminate eggs.
RFR method: do not use this method; antihelminthic drugs are preferable.
Its resonant frequencies are: 336, 395-400, 420-430, 536 kHz

9.1.3. Hookworm Infections (Ancylostoma duodenale and Necator americanus)

There are many species of hookworms that infect mammals, but there are only two intestinal *roundworms* that infect human beings. One of them named *Ancylostoma duodenale* predominates in the Middle East, North Africa, India and formerly in southern Europe; too. The other one named *Necator americanus* predominates in countries of America Sub-Saharan Africa, Southeast Asia, China and Indonesia. About one fourth of the population of the world is infected with hookworms.
These hookworms are about 10 mm in length and live in the small intestine of the host. The males and females mate, the female produces eggs, which pass into the feces. Depending on the species, female hookworms can produce 5,000-25,000 eggs per day. About two days after their passage, the eggs of the hookworm get hatched, and the juvenile worm (or larva) gets infective in about five days. In case of *Necator americanus* the next host will be infected when an infective larva penetrates the host's skin. Then the juvenile worm migrates through the host's body and finally ends up in the host's small intestine, where it grows to sexual maturity. In case of *Ancylostoma duodenale* the portal of their entry is usually the ingestion rather than the skin.
The presence of hookworms can be demonstrated by finding the characteristic eggs in the feces; the eggs however can not, however, be differentiated according to species. The mouthparts of hookworms are modified into cutting plates. The attachment of hookworms to the host's small intestine causes hemorrhages, the hookworms feed on the host's blood causing anaemia. Hookworm diseases can have devastating effects on human beings, particularly as regards children, due to the loss of excessive amounts of blood. The larvae as well as the juveniles of dog hookworms and cat hookworms (such as the Ancylostoma braziliense, the Ancylostoma caninum etc.) can infect people, though they do not mature into adult worms, there. They remain in the skin migrating for weeks, or in some cases, even for months, resulting in a condition known as *cutaneous or dermal larva migrans, or „creeping eruption.*" Hence, one has to remember the importance of not allowing dogs and cats to defecate indiscriminately.
The eggs of every kind of hookworms are discharged into the stool and hatched in the soil after an incubation time of a few days, then they become larvae and live in the soil. A person can become infected by walking barefoot through a field contaminated by human or animal feces as the larvae are able to penetrate the skin. The larvae travel through the lymphatic vessels and the bloodstream to the lungs and other organs. The presence of larvae in the lungs causes an increased amount of secretions, with which the larvae travel from the lungs, are coughed up, and swallowed. Thus, about a week after penetrating the skin, the larvae pass into the intestines, where they mature into adult worms. The larvae attach themselves by their mouths, being sharp, curved, hook-like, to the lining of the upper small intestine and there suck blood. Hookworm larvae may enter the bloodstream through the wall of the intestine.
Symptoms: in the locus, where the larvae penetrate the unbroken skin, itchy, flat rashes may develop. Fever, cough, and wheezing may be caused by the migration of the larvae through the lungs. The adult worms often cause pain in the upper abdomen. Anaemia, caused by iron deficiency, eosinophilia and hypoproteinaemia in the blood can come about due to intestinal bleeding.

Diagnosis: hookworm eggs can be detected in the stool by microscopy.
Treatment: by administering pyrantel pamoate or mebendazole. Check the resistance to antihelminthic drugs. Repeat the treatment after one week.
RFR method: the first step must be the administering antihelminthic drugs. Then use RFR method between two antihelminthic treatments to eliminate the remaining parasite larvae. Detects and after repeated treatments eliminates the larvae in the tissues.
The frequencies of Ancylostoma duodenale are: 380-403, 506-511 kHz
The frequencies of Ancylostoma braziliense (Dog and cat hookworm, the larva of which is the most common cause of the cutaneous larva migrans aka creeping eruption) **are: 319, 508 kHz**
The frequencies of Ancylostoma caninum are: 307-312, 318, 489-498, 507 kHz
The resonant frequencies of other non-differentiated Ancylostoma species are: 332, 346-352, 364-374, 383-406, 423-432, 482-488 kHz

9.1.4. Strongyloidiasis

Strongyloidiasis is an infection in human organs resulting from the invasion by roundworm larvae, such as larvae of Strongyloides stercoralis, an unusual parasite, having two life cycles. In its parasitic life cycle, the female worms are found in the superficial tissues of the human small intestines; where are apparently no parasitic males. The female worms produce larvae parthenogenically (without fertilization), which then pass into the host's feces. The presence of this nematode larvae in the fecal sample is characteristic of strongyloidiasis. Once they are in the feces, some of the larvae develop into free living larvae, while others develop into parasitic larvae. The free living larvae will complete their development in the soil and mature into free living, non-parasitic males and females. These free living males and females mate, produce more larvae, and the cycle, described above, repeats itself, and, eventually, the new larvae become either free living or parasitic worms. As one can imagine, this life cycle constitutes an important reservoir for human infections.
The parasitic larvae infect the human host by penetrating the skin. The larvae migrate to the lungs via the circulatory system, penetrate the alveoli, then passes into the small bronchioles, are then coughed up and swallowed. Once they get into the small intestine, the larvae mature into parasitic females. Strongyloides stercoralis can infect people also via autoinfection. In some circumstances, f.i. chronic constipation, the larvae, produced by the parasitic females, will remain in the intestinal tract long enough to get to their infective stage. These larvae will penetrate the tissues of the intestinal tract and develop in a way as if they had penetrated the skin. Autoinfection can also occur, if larvae remain penetrating the perianal skin. Autoinfection often leads to very heavy worm burdens for human beings. Since the parasitic females live in the superficial tissues of the small intestine and can be present in high numbers, they can cause a significant pathology.
The larvae penetrate the intestinal wall and travel to the internal organs, to the brain and to the tissues of other organs. If the infection reaches the brain and its membrane lining, meningitis can develop. Vision disorders and hearing disorders accompanied by headache may be experienced. The lungs, the pleura and the heart may also become inflamed, resulting in fever, cough, or wheezing. Other possible complications can include skin rashes, such as hives, or rash in a linear pattern, spleen enlargement, eosinophilia, radiating pain in the pit of the stomach and diarrhea.
Diagnosis: by microscopic examination of feces.
Treatment: by administering Thiabendazole
RFR method: following the treatment with Thiabendazol
Its resonant frequencies are: 357, 369-385, 389, 390-411, 424, 432, 564, 569 kHz
The resonant frequencies of its larvae are: 318, 411, 507 kHz

9.1.5. Trichuriasis

Trichuriasis is an infection caused by *Trichuris trichiura*, an intestinal roundworm. There are only two of the approximately sixty species of whipworms that can infect human beings i.e. the *Trichuris trichiura*, and the canine whipworm, i.e. the *Trichuris vulpis*. These two species show a high host specificity, but canine whipworms can, on rare occasions, infect people too. Whipworms derive their name from the characteristic shape of their adults. The adults live in the host's large intestine with their ends embedded in the cells that line the intestine; each female can produce in excess of 10,000 eggs each day, and the worms can live for several years. The eggs are passed in the host's feces, and become infective in about three weeks. If an infective egg is eaten by the appropriate host, it hatches in the small intestine, from where the juvenile worm migrates to the large intestine, reaching there its sexual maturity. Most infections of the whipworms are probably asymptomatic. However, as the worms live for a long time and a person can be constantly reinfected, heavy worm burdens can develop.

The Symptoms of a whipworm infection can include abdominal pain, diarrhea, dysentery and anemia. Concerning children, the heavy infections can cause mental and physical retardation.

The diagnosis done by demonstration of the barrel-shaped eggs, which can have a characteristic appearance in the stool samples examined under a microscope.

Treatment: by administering mebendazole.

RFR method: The first step is to administer mebendazole or other antihelminthic drugs.

Its resonant frequencies are: 321-324, 380-410 515-517 kHz

9.1.6. Trichinosis

Trichinosis is a roundworm infection caused by *Trichinella spiralis*.

Unlike many parasites that demonstrate a high degree of host specificity, *Trichinella spiralis*, the trichina worm, can be found in many species of carnivores and omnivores. Animals are infected by *T. spiralis*, when ingesting infective larvae (juveniles) present in raw or undercooked meat. The larvae mature into adults in the host's small intestine in a few weeks, and the female worms give birth to larvae. (The males die after fertilizing the females, and the females die after producing larvae.) The larvae enter the blood stream of the host and, eventually, end up in the host's muscles. Here the larvae mature into infective larvae, so that the next host will be infected by eating these larvae. In the muscles, the larvae cause a severe host reaction that results in soreness and tenderness of the muscles. Although this parasite probably only rarely causes fatalities in human beings, it can cause extreme discomfort.

Trichinella spiralis is best known as a parasite that people contract by eating raw or undercooked pork. Because of its low host-specificity, almost any wild meat should be suspected, and hunters should be careful if they prepare meat from their victims.

Trichinosis occurs in most parts of the world, but is rare or absent in regions where pigs are fed root vegetables. Hunters who eat wild pigs or bears, deer and/or rabbits may develop a very high infection rate. Although the infection most often results from eating raw or inadequately cooked pork or pork products, it can also result from eating the meat of bears, boars and, in rare cases, of some marine mammals. Any one of these animals may contain a cyst form of the larvae of trichinae. If the cyst wall is digested in the stomach or the duodenum, it releases larvae that penetrate the wall of the small intestine. The male worms play no further role in the causing of infection. The females burrow their way into the intestinal wall and, by the seventh day, begin to discharge living larvae. Every female may produce more than 1000 larvae. The tiny larvae are carried round the body by the lymphatic vessels and the bloodstream. Only those larvae survive, which reach the skeletal muscles. They penetrate the muscles, and cause inflammation. By the end of the third month, they form cysts. Certain muscles, such as the tongue, the muscles of the eye and the muscles between the ribs, are particularly suited to be infected. Larvae that reach the heart

muscle are killed by the intense inflammatory reaction and the allergic reaction provoked by them. In rare cases, the larvae may get into the brain or into other internal organs.
Symptoms: Bleeding at the whites of the eyes and at the back of the eyes, pain in the eyes and sensitivity to light can be caused. Muscle soreness and pain, together with a skin rash and bleeding under the nails may develop shortly afterwards. Great difficulties in breathing may follow, sometimes causing even death. Some additional symptoms may include profuse sweating, fever, chills and weakness. An increased number of eosinophilic cells in the blood is frequently noticed. As the immune system destroys the larvae outside of the muscles, of the lymph nodes, as well as outside of the brain their membrane linings may become inflamed, vision or hearing disorders may develop. The lungs, the pleura and the heart may also become inflamed. Heart failure may develop within the fourth and eight weeks.
Diagnosis by confirmation with a biopsy of the infected muscle and by examinating under microscope.
Treatment: by administering mebendazole and thiabendazole. Testing in bed helps to relieve the muscle pain; analgesics, corticosteroids, f.i. prednisolone, may be of use to reduce the inflammation of the heart or that of the brain. The treatment must last for a long time and has to be repeated.
RFR method: the first step to take is the treatment with antihelminthic drugs. RFR detects and eliminates the parasite in the tissues.
Its resonant frequencies are: 350-354, 400-408, 420, 537-541, 552 kHz

9.1.7. Filariasis

Filariasis is a group of disorders produced by being infected with the thread-like nematodes of the *Filaroidea* superfamily. These worms invade the lymphatics and the subcutaneous and deep tissues of human beings, producing reactions ranging from acute inflammation to chronic scarring. The viviparous females discharge microfilariae into the blood or into the subcutaneous tissues, where they live for weeks or months until they are taken up by hematophagous arthropods. Within these vectors they are transformed into filariform larvae, which will then infect a new host when the arthropod takes another blood meal.
The clinical picture, produced by various species of this group, is more or less specific. The term **lymphatic filariasis** is commonly used to designate the disease caused by *Wuchereria bancrofti* and *Brugia malayi*. These organisms are responsible for the lymphatic blockade which causes elephantiasis. *Loa loa* causes **loiasis,** a disease characterized by transient subcutaneous swellings. *Onchocerca* volvulus causes blindness and also pruritic skin rashes, typical of **onchocerciasis**. *Mansonella ozzardi*, *Mansonella perstans* (formerly named *Dipetalonema perstans*) and *Mansonella streptocerca* (formerly named *Dipetalonema streptocerca*) cause infections of questionable clinical significance concerning human beings and animals. These parasites are identified by the location, the periodicity and the morphologic characteristics of their microfilariae.
The presence of adult worms in the lymphatics causes pathologic changes which can be divided into two categories: an inflammatory and an obstructive one. The inflammatory response, marked mostly by being found around the molting larvae and the dead or dying adult worms as well, occurs due to the infiltration with lymphocytes, plasma cells and eosinophils. This is followed by a granulomatous reaction which may lead to lymphatic obstruction. Hyperplasia of the lymphatic endothelium, acute lymphangitis and thrombosis are also present. The clinical manifestations, including edema, ascites, hydrocele of the scrotum, pleural effusion and joint effusion. Lymphadenopathy will develop for a long time. The lymphadenitis almost always accompanies or sometimes precedes the lymphangitis. Inguinal, femoral and epitrochlear nodes are involved usually. The abscesses formed around the affected lympatics and lymph nodes may discharge to the surface,

resulting in persistently draining sinus tracts. In some cases there can even develop an elephantiasis.
Patients with pulmonary manifestations show obstructive and restrictive abnormalities as regards their pulmonary functions and will possibly develop an irreversible pulmonary hypertension with tropical eosinophilia.
Diagnosis: by the demonstration of the parasites and of the hyper-eosinophilia caused. The skin tests and the serologic tests are group-specific, lack of sensitivity and may often produce false negative results concerning other nematode infections. However, in absence of microfilariae and other helminthic infections, they may be helpful in establishing a diagnosis in clinically suspect cases.
Treatment: by administering Diethylcarbamazine, Suramin, and Niridazole.
RFR method: is to be used concurrently with an antiparasitic drug treatment, in order to detect and eliminate the parasites.
Its resonant frequencies are: 318, 355-368, 385, 408, 421, 436-446, 488 kHz

9.1.7.1. Onchocerciasis („River Blindness")

The onchocerciasis is a form of cutaneous filariasis caused by *Onchocerca volvulus*. It is characterized by subcutaneous nodules, pruritic skin rashes and ocular lesions.
Onchocerciasis is found in many a country of the world, f.i. in parts of Africa, Arabia, Central America, northern South America and Mexico. It is estimated that solely in Africa more than 30 million people are infected with this parasite. The life cycle of this parasite is similar to those of other filarial parasites. The adult worms live in the skin of human beings. The adult worms (which may be up to 50 cm in length) are surrounded by the fibrotic tissue of the host, resulting from the host's response to the parasite, the fibrotic capsule often appears as a nodule under the skin. The female worms produce microfilariae (advanced embryos), which remain in the skin. The vector of this parasite is a black fly (i.e. Simulium sp.), which becomes infected when it feeds on an infected man. The microfilariae develop into infective larvae in the vector, the disease is transmitted to another person via the infective larvae. Once the infective larvae get into the human skin they do not anymore migrate. They develop there into adults at the spot of the vector's bite. An inoculated larva matures into a male or a female one within approximately one year. Though larvae do not multiply within the human host, heavy parasite loads are still the result of the parasitic repeated infections. The adult worms are found coiled together in the fibrous subcutaneous nodules. The gravid females, which may live for 10–15 years, release unsheathed, motile microfilariae, migrating in the skin, in the subcutaneous tissue, and in the eyes for up to 30 months or until they are ingested by a feeding Simulium. The subcutaneous nodules which enclose the adult worms are usually 2 to 3 cm. in diameter when fully developed. ")
Unlike most other filarial infections in which the adult worms cause the problem, (e.g. the Bancroftian filariasis), in this disease the microfilariae of *O. volvulus* are the cause of most problems. River blindness, that is Ophthalmic Onchocerciasis, is the world's second leading infectious cause of blindness.
Symptoms: The microfilariae enter the eye and die, and eventually cause blindness. In some endemic areas, 30–40% of the adult population becomes blind. (The vector breeds in rivers, which is why the disease occurs most often in areas near to rivers; hence the name "river blindness.")
Onchocerciasis can be characterized by the following phases or types: "Erisipela de la costa, Mal morando and Sowda".The significant pathologic changes occur as a result of a hypersensitive reaction to the dead or dying microfilariae. The skin lesion may appear as an erysipelas-like reaction over the face or like a pruritic papular rash over the extremities. In chronic cases thickening, lichenification, and depigmentation may be present. The depigmentation, in combination with the nodules containing the adult worms, result in

severe disfigurement. The most serious complications of onchocerciasis are the eye lesions, which are usually found in patients repeatedly infected on the upper part of the body. A punctate keratitis, iridocyclitis, or, less commonly, a chorioretinitis can occur, leading to blindness.

Prevention: Chemoprophylaxis might not be necessary for the protection of people, but protective clothing has to be worn and insecticides has to be used.

Diagnosis: can be established by the clinical symptoms, and by one of the filarial serologic tests. ")

Differential diagnosis: by excluding other parasitic infections.

Treatment: by administering Thiabendazole, Pyrantel pamoate, Mebendazole, Praziquantel, Niclosamide, Diethylcarbamazine, and Antihistamines. Mass therapy and nodulectomy are seldom useful..

RFR treatment: detects the parasites, and, after treatment with antiparasitic drugs, is able to eliminate them definitely. ")

Its resonant frequencies are: 347-351, 430-447, 557-560 kHz

9.1.7.2. Dirofilariasis

Dirofiliasis is a parasitic infection caused by *Dirofilaria immitis* (affecting dog) or *Dirofilaria tenuis* (affecting raccoons) and other dirofilariae. *Dirofilaria immitis* is a large filarial microorganism infecting dogs and living in their right ventricle and their pulmonary arteries releasing its microfilariae into the peripheral blood of the animal. The parasite is also called canine heartworm. It is transmitted by several types of *mosquitoes*. Human infections have been occasionally reported. The worm does not mature in the human body, hence microfilaremia is not present there. Although cardiac infections are noted at autopsies, most human infections appear as well-defined pulmonary nodules.

Symptoms: The patients may cough and may complain of chest pain, or, less commonly, suffer from hemopthysis, fever, chills and myalgia.

Diagnosis: is usually made by microscopic examinations of the excised pulmonary nodules.

Treatment: the nodules are to be removed by surgical excision. Dietylcarbazine, Praziquantel, Yomesan and other antiparasitic drugs are not effective enough

RFR method: may be attempted.

Its resonant frequencies are: 318-327, 338, 350, 355-365, 385, 408, 421, 436-446, 488, 518,
558 kHz

9.1.7.3. Mansonellosis (former Dipetalonemiasis)

Mansonella perstans (formerly named *Dipetalonema perstans* or *Acanthocheilonema perstans*) and *Mansonella streptocerca* (formerly named *Dipetalonema streptocerca*) are filarial parasites of human beings and other primates inhabiting tropical areas. The adult worm lives encysted in the subserosal tissues of the pericardium, the pleura, and the peritoneum, and most particularly in the tissues of the mesentery. The unsheathed microfilariae, found in the peripherial blood all throughout the day, have four to six nuclei in their tails. They are transmitted from host to host by *blood-sucking gnats of the genus Culicoides.* Most infections are asymptomatic, their principal significance lies in the fact that they may be confused with other, more serious forms of filariasis.

Symptoms: Some patients complain of fever, pruritus, Calabar swellings, erysipelas like rash and abdominal pain. Peripheral eosinophilia is common, and the filarial complement fixation tests are usually negative.

Diagnosis: by examination of the characteristic microfilariae in the peripheral blood.

Treatment: by administering Dietylcarbazine, Suramin, Mebendazole, Niclosamide and other antihelminthic drugs.

RFR method: should only be used together with the taking of antiparasitic drugs.
The most frequent resonances of Mansonella (Dipetalonema) are: 420-460, 467-515 kHz

9.1.7.4. Loiasis

Loiasis is a disease caused by *Loa loa,* prevalent in Africa and other tropical countries. The infection is transmitted by *deer flies of the genus Chrysops*, and other mosquito flies. The adult worms, which may live for 15 to 20 years, migrate continuously through the subcutaneous tissues.
Symptoms: Localized, allergically inflammated areas, the socalled Calabar swellings are the hallmark of the disease, an immune reaction caused by the secreted toxins of the worms being injured by a minor or major force affecting the skin. Occasionally the adult worms may be seen crossing the eye subconjunctivally, causing intense lacrimation, pain, and anxiety; thus this worm is often called eye worm. A marked general and local eosinophilia is usually present in case of loiasis.
Diagnosis: is established by positive filarial complement laboratory tests, eosinophilia and parasite examinations. Diagnosis can be done by finding the adult worm or by demonstrating the distinctive, sheathed microfilariae in the contents of the Calabar swellings or in the bloodstream axamined during the day.
Differential diagnosis: by differentiating it from other parasitic infections.
Treatment: by administering Praziquantel, dietylcarbamazine and other antiparasitic drugs.
RFR method: use it in conjunction with antiparasitic drugs.
Its resonant frequencies are: 360-365, 550-554 kHz

9.1.8. Haemonchus Contortus

Haemonchus contortus is a red stomach worm and a very common parasite, one of the most pathogenic nematodes infecting ruminants. The adult worms are attached to abomasal mucosa, feeding on blood. Females may lay over 5,000 eggs a day, which are passed via the faeces. The life cycle of Haemonchus contortus is as follows: after hatching their eggs, the larvae molt several times, getting into the form L3, which is infectious to animals. They can take up these larvae when eating grass leaves. The L4 larvae, formed after another molt, suck blood in the abomasum of the infected animal, potentially giving rise to anaemia and oedema of the host, which eventually lead to death. The infection, named Haemonchosis, can cause large economic losses among farmers all over the world, especially for those living in warmer climates. Antihelminthics are used to combat these worms, and other worm infections as well, but the resistance of these parasites against these chemicals is growing.
Haemonchus is most common in tropical or subtropical regions or in those with a lot of rain in summer. The latter species is predominant in temperate zones.
Haemonchosis is very rare among human beings, the course of this infection can be hyperacute, acute, or chronic. The acute form of the disease is characterized by severe anemia accompanied by generalized edema. Anemia is characteristic also of the chronic infection, often of low worm burdens, accompanied by a progressive loss of weight. is not a sign of Haemonchosis does not cause diarrhea. The acute disease is characterized by severe anemia accompanied by generalized edema; anemia is also characteristic concerning chronic infections, often with low worm burdens, and is accompanied by a progressive loss of weight, too. The abomasum is edematous and, in the chronic phase, the pH increases, causing gastric dysfunctions.
Diagnosis: and differential diagnosing of trematode eggs in feces including other trematodes.
Treatment: by administering Praziquantel, or Albendazole.

9.2. Human Intestinal Tapeworm Infections

The parasitic tapeworms, (named also Cestodes, flatworms or Woods) are segmented, ribbon-shaped hermaphroditic worms that can inhabit the intestinal tract of many kind of vertebrates. Unlike other helminths, these parasites lack a digestive tract, they absorb food through their entire body-surface. Their attachment to the host's intestinal mucosa is effected by means of sucking cups or grooves (named scolex), located on this head. In regard to some species, the head is also armed with hooklets which help their attachment. Behind the globular scolex there lies a short, narrow neck from which segments or proglottids develop, one at a time, to form the chain-like strobila of the worm. These proglottids mature progressively as they are displaced further and further from the neck by the formation of new segments. Once each section reaches gravidity, it releases its mass of eggs by passing them through a uterine pore, by splitting open, or simply by disintegrating. The eggs of many a tapeworm appear to be identical, the identification of the species depends on the morphologic characteristics of the scolex, or on the gravid proglottids. Other species of this genus infect dogs and cats, so that people are likely to encounter them, by noting the presence of their proglottids in the feces of these pets. All species of Taenia have similar life cycles.

The most common tapeworms infecting human beings are the Pork tapeworms (*Taenia solium*), the Beef tapeworms (*Taenia saginata*), the Fish tapeworms *(Diphyllobothrium latum)* and the Dwarf tapeworm (Hymenolepis Nana).

The infections involving the adult pork tapeworms and beef tapeworms are called **taeniasis**. An infection with the larval stage of these worms causes **cysticercosis**.

Tapeworms, belonging to the *Echinococcus* genus, can infect so animals as people, causing the most harm to intermediate hosts, such as sheep and cattle. The infection with this type of tapeworm is referred to as Echinococcosis or hydatid disease.

9.2.1. Taeniasis and Cysticercosis (Taenia Saginata and Taenia Solium)

There are several species of the Taenia genus, that people are likely to get ill from. Human beings serve as the only definitive host for the *Taenia saginata* (nowadays often referred to as *Taeniarhynchus saginatus*), i.e. the Beef tapeworm; and also for the *Taenia solium*, i.e. the Pork tapeworm. People get infected by eating contaminated raw or undercoocked meat. The adult tapeworms live in the jejunum of the definitive host. The proglottids, which contain the eggs, break off the posterior end of the tapeworm. The proglottids either pass intact in the host's feces or dissolve in the host's intestine and the eggs pass in the feces. The eggs of Taenia genus have a characteristic appearance, but they can not be differentiated according to their species. These eggs are not capable to invade the tissues of the definitive host. The intermediate host will be infected by ingesting the eggs which, once ingested, are capable to hatch out. The larvae of *Taenia solium* and *Taenia saginata* are able to invade the tissues by entering the bloodstream in the small intestine. From there, they spread to many organs such as to the striated muscles, the heart, the eyes, the brain and the spinal cord, forming there cysts named cysticerci. In this state they cannot grow into adult worms remaining encapsulated in these tissues. The definitive host can be infected by eating the intermediate host (such as uncooked pork or beef) infected with cysticerci.

Symptoms: *Infections caused by adult tapeworms* rarely cause symptoms in the definitive host's small intestine, though, there may occur complications, f. i. if the tapeworms block the intestinal tract, due to their large size, or if the proglottids become lodged in the

appendix resulting in appendicitis. The proglottids of Taenia are large and muscular, so that the single proglottids or their long chains might, occasionally, crawl out of the anus of an infected person. Though the adult tapeworms found in people usually do not cause any symptoms except a feeling of discomfort in the small intestine, persons, infected with cysticerci (f.i. by eating uncooked pork or beef or drinking contaminated water), will have an illness with significant pathology. Such an infection is referred to as *cysticercosis,* in which case cyst in the muscles cause a painless swelling or nodules under the skin. Cysts in the eye can impair the vision by causing swelling and the detachment of the retina. Cysts in the heart can cause abnormal rhythms. An encystment in the CNS i.e. the neurocysticercosis can cause seizures, headaches, confusion, lack of attention, dizziness and the compression of the brain tissue leading to hydrocephalus and even to death. An encystment in the spinal cords can cause weakness and paralysis, as well. If the parasite dies, swelling and then scarring will develop in the locus of the cysticercus.

Diagnosis: Infections caused by adult Taenia species are diagnosed by recovering eggs or proglottids in the feces of the host. The eggs of *T. saginata* and *T. solium* are virtually identical, but the species can be differentiated based on the morphology of their proglottids and scolex (holdfast). However, since the same drugs are used for treating both species, a differentiation is generally unnecessary. In case of cysticercosis not only the observed symptoms, but antibody tests, biopsy, CT-scan, MRI examinations can also be helpful.

Differential diagnosis: The proglottids of *canine and feline species* of the *Taenia* are rectangular and larger than those of the *Dipylidium caninum.* Dogs serve as definitive hosts for this species of cyclophyllidean cestodes, while rabbits most often serve as intermediate hosts. Dog owners often encounter this parasite when the proglottids are passed in the stools of their pet. Many biological aspects of these cestodes are similar to each other.

There are some species of the cyclophyllidean cestodes in the *Multiceps* genus which produce in their metacestode stage cenurus, similar to cysticercus, though they containmany scoleces. The intermediate host of these species is a rabbit or a hare, and the definitive host is a dog or an other canine. These cenuri cause illness among the intermediate hosts. Human infections (acquired by accidental ingestion of *Multiceps* eggs) seldom occur, but if, then mostly among children, causing diarrhea and restlessness.

Treatment: the administering of anti-parasitic drugs such as Praziquantel and Albendazole is controversal, as only dead or dying parasites and not the living ones do invoke an inflammatory response and cause seizures and not the live ones. The administering of corticosteroids may be helpful in case of swelling or uncontrolled immune responses. Surgical actions may also be necessary f.i. the removing of cysts.

9.2.2. Diphyllobothriasis, Sparganosis (Fish Tapeworm infections)

The Fish tapeworm infection (**diphyllobothriasis**) is an intestinal infection caused by the adult tapeworm *Diphyllobothrium latum.*

Many fish-eating vertebrates can serve as the definitive host for *Diphyllobothrium latum* (the broadfish tapeworm), including humans, dogs, foxes, cats, mink, bears, and seals. The adult tapeworm lives in the host's small intestine, and in humans the tapeworm can reach a length of 10 meters (>30 feet) and produce over a million eggs a day! The life cycle of D. latum involves two intermediate hosts. The first intermediate host is a copepod, the second intermediate host is a fish, often a pike or a salmon, and the definitive host is infected by eating raw or undercooked fish. In case of human beings, this tapeworm is more prevalent in areas where people consume significant quantities of fish, including Scandinavia and the areas bordering the Great Lakes in the US. Dogs and cats are often infected when they are fed the offal remained after cleaning fish. Occasionally, people are infected with the plerocercoid stage of cestodes. Such infections are referred to as **sparganosis.**

People also can serve as intermediate hosts; the eggs reach the stomach either when a person swallows them or when proglottids are regurgitated from the intestine to the stomach. The Taenia solium embryos are released inside the stomach. They then penetrate the intestinal wall and travel to the muscles, the internal organs, the brain, and the tissue under the skin, where they form cysts. Live cysts cause only a mild tissue reaction, whereas dead ones invoke a vigorous reaction.

The life cycle of *Dipylidium caninum*, the „cucumber tapeworm," involves dogs or cats (rarely humans) as the definitive host and fleas or lice as the intermediate host. The perianal region of the dog or cat becomes contaminated with eggs when these are passed in the feces, after which the flea or louse ingests the eggs. The dog or cat (or human) is infected if they ingest a flea or louse infected by a cysticercus being in a metacestode state (cysticercoid). Hence the importance of controlling fleas on pets.

The feces of an infected dog or cat (or human being) may contain proglottids (often incorrectly referred to as segments) that are shed from the tapeworm, having a characteristic size and shape (more like rice grains than cucumbers). The diagnosis of this species depends on finding proglottids or egg packets (see below) in the feces. The proglottids of the other common tapeworms of dogs, i.e. the *Taenia pisiformis*, are much larger and rectangular in shape.

9.2.3. Echinococcosis (Hydatid disease)

The life cycle of *Echinococcus granulosus* includes dogs (and other canines) as its definitive hosts, and a variety of species of warm-blooded vertebrates (sheep, cattle, goats, and human being), as its intermediate hosts. The adult worms are very small, usually consist of only three proglottids (their total length is 3-6 mm), and they live in the small intestines of dogs. The eggs are liberated in the host's feces, and if they are ingested by the intermediate host they hatch in the host's small intestine. The larvae in the eggs penetrate the gut wall and enter the circulatory system. The larvae can be distributed throughout the intermediate host's body (although most of them end up in the liver) and can grow into a stage referred to as hydatid cyst. Hydatid cysts are able to grow to be fairly large: cysts of the size of golf balls are not uncommon, while cysts as large as basketballs are seldom found. The pathology associated with the hydatid disease in the intermediate host depends on the size of the cyst and its location. One or two small cysts in the liver of a host might go unnoticed for years. However, even one single large cyst in the liver might prove to be fatal. The hydatid disease is far more serious if the cysts are found in other parts of the body, particularly if found in the brain. The infection is transmitted to the definitive host when the hydatid cyst is eaten.

As one might suspect, this species of parasite is more common in areas of the world where dogs are used to herd sheep. Under most circumstances human beings are a „dead end" in the life cycle of the microorganism, though concerning people the hydatid disease still remains to be a serious pathological problem. The interior of a hydatid cyst is filled with protoscolices, everyone of which is able to grow into an adult worm if ingested by a canine host. A small cyst might contain hundreds of protoscolices; while a large one tens of thousands! This tremendous reproductive potential poses a problem as regards the intermediate host (particularly concerning people). If a hydatid cyst breaks up, each protoscolex could grow into a new hydatid cyst. How can this happen? A sharp blow to the abdomen might rupture a cyst in the liver. Quite a number of cases have been reported according to which cysts have been damaged by routine surgery, when the contents of the cyst were let to leak into the patient's abdominal cavity.

Symptoms: The infection with the adult worm usually does not cause any symptoms. A heavy infection with cysts may cause muscle pain, weakness and fever. If the infection reaches the brain and its membrane linings, they may become inflamed.

Diagnosis: In case of an infection caused by an adult worm, eggs may be seen around the anus or in the stool. The proglottid or the head of the worm must be looked for and found in the stool and be examined under microscope in order to be able to distinguish the pork tapeworm from other tapeworms. Live cysts in tissues like those in the brain can best be seen by CT and MRI.
Treatment: by administering Praziquantel, Niclosamide and by the surgical removal of cysts; while in case of Echinococcus: it can be treated by administering high doses of mebendazole.
RFR method: the first step to take is to treat the disease with Praziquantel or Yomesan; after which, in order to detect the parasite residuum and eliminate the larva, RFR method can be used.
Some of the varieties of cysticercus consist of many heads, and each head contains even more heads, which might have different resonant frequencies. To produce the desired effect they must all be eliminated. Since bacteria and viruses are released by the eliminating of their host tapeworms, this process should always be followed by using the sweep method (i.e. by sweeping them out).
The resonance frequencies of the tapeworms are often extremely weak, possibly due to their being encased in a cyst. Search between 400 and 550 kHz. Patients may be disappointed that they do not to feel any difference in their condition after being rid of a number of tapeworms and their pathogens. Evidently, the presence of the tapeworms itself does not make a person sick; it is simply a condition, similar to that of a wart, the presence of which does not mean illness. Nevertheless viruses in tapeworms are well able to cause illness, and depending on the type of the virus it can make one very sick or not sick at all. Different viruses invade different organs, and some of them can turn into warts.
The resonant frequencies of Taenia pisiformis cysticercus are: 470-510 kHz
The resonant frequencies of the Taenia pisiformis eggs are: 460-470 kHz
The resonant frequencies of Taenia saginata cysticercus are: 470-490 kHz
The resonant frequencies of Taenia solium cysticercus are: 470-480 kHz
The resonant frequencies of the Taenia solium scolex are: 410-520 kHz
The resonant frequencies of the Diphyllobothrium mansonoides (erinacei) scolex are: 460-490 kHz
The resonant frequencies of the Diphyllobothrium latum scolex are: 450-480 kHz
The resonant frequencies of the Dipylidium caninum proglottid composite are: 440-460 kHz
The resonant frequencies of the Dipylidium caninum scolex are: 450-510, 612 kHz
The resonant frequencies of undefined tapeworms are: 312-318, 411, 431-440, 444, 534-554 kHz
The resonant frequencies of Echinococcus granulosus are: 318-320, 450-500, 554 kHz
The resonant frequencies of the Echinococcus granulosus cysts are: 318-322, 450-465, 474 kHz
This list is not yet complete.

9.2.4. Hymenolepiasis

Hymenolepiasis is an intestinal tapeworm infection of human beings, mice and rats caused by *Hymenolepis nana*. The infection is particularly common among children, in whom it is usually asymptomatic. Its life cycle is unique, as the larval phases as well as the adult phases occur in the same host. The eggs are immediately infective, ingested by a new host, the freed oncospheres penetrate the intestinal villi, becoming cysticercoids. Then the larvae migrate back to the intestinal lumen, attach themselves to the mucosa, and mature into adult worms. The eggs hatch before passing in the stool, causing an internal autoinfection. This tapeworm infection is characterized by the presence of many adult worms in the host's intestine.

Symptoms: If the infection is massive, or if it is in a chronic stage, diarrhea and abdominal pain will set in.
Diagnosis: by examining the stool microscopically.
Prevention: the contamination of food by rats or mice should be prevented.
Treatment: by administering Niclosamide, Mebendazol, Yomesan, or Praziquantel.
RFR method: For this infection RFR method is of no importance. Do not use it without administering antiparasitic drugs.
The most frequent resonances are: 440-490 kHz

9.3. Human Pathogenic Flukes

9.3.1. Schistosomiasis (Bilharziasis)

Schistosomiasis is possibly the most important one of the helminthic diseases because of its worldwide distribution and the extensive pathologic changes produced by these parasites. Schistosimiasis (bilharziasis) designates a group of diseases caused by three closely related species of digenetic trematodes, or blood flukes, belonging to the Schistosomatidae: *'Schistosoma mansoni*, *Schistosoma haematobium* and *Schistosoma japonicum*. In tropical and subtropical countries the schistosomal blood flukes can inhabit the circulatory system of human beings and animals, where they deposit large numbers of eggs, many of which are retained within the body of the host, producing inflammatory lesions. The most frequently affected organs and tissues are the colon, the urinary tract, the bladder, the liver, the lungs and the central nervous system. The adult worms grow and mature within the portal venous system of the liver, measuring 1 to 2 cm in length. After copulation the male carries the female against the flow of portal blood to the small mesenteric vessels. If they can travel no further, the female deposits her eggs in clusters, slowly retreating down the vessel in front of them. The eggs, viable for three weeks, secrete an enzymatic substance which destroys the surrounding tissue. If the eggs lie close to the mucosal surface, they rupture into the lumen of the gut and are carried outside in the urine or feces. Reaching fresh water, the embryonated eggs quickly hatch, liberating ciliated miracidia.
Schistosoma mansoni cause intestinal bilharziasis, or schistosomal dysentery. The *intermediate snail hosts* belong to the genera Biomphalaria and Tropicorbis. Human beings are heldt to be the principal hosts, though baboons in Africa have been found to be infected naturally.
Symptoms: The clinical manifestations can be divided into three phases:

1) an early stage, in which cercariae penetrate the skin, and the resulting shistosomula are carried by the blood to the liver, mature into adult parasites within the intrahepatic portal veins;
2) an intermediate stage, dealing with the duration of the oviposition and the egg extrusion; and
3) a late stage, characterized by tissue proliferation and fibrosis in response to the eggs that have been retained in the tissue. Within a few hours of their penetrating the unbroken skin, most cercariae die, producing dermatitis characterized by round-cell infiltration, pruritus, and papular eruption, the result of the sensitization to the cercarial antigen, rarely occuring in case of a primary infection.

The rash is commonly followed by headache, myalgia, abdominal pain and diarrhea, which manifestations are presumably related to the migration of the schistosomula. The illness is characterized by high raging fever, chills, cough, urticaria, lymphadenopathy, enlarged, tender liver and spleen, and occasionally melaena as well. In the bowels, these changes produce congestion, thickening, and, in case of severe infections, sessile or praecancerous pedunculated polyps. Clinically there can be observed abdominal pain, anemia, leukopenia,

thrombopenia, jaundice, ascites, diarrhea with or without blood and a mild protein-losing enteropathy. Rarely experienced complications are the intestinal obstruction and the rectal prolapse.
Schistosoma japonica species produce early symptoms, f.i. pruritic dermatitis, cough, angioneurotic edema, diarrhea and fever resulting from the penetrative migration of the schistosomulas, similarly to that observed in case of schistosomiasis mansoni.
Diagnosis: depends on the finding and identification of the ova.
Treatment: by administering Niridazole (Ambilhar)
RFR method: detects and eliminates the parasite.
Its resonant frequencies are: 325-330, 336-346, 350-357, 428, 433-445, 456-504 kHz
The resonant frequencies of Schistosoma mansoni (blood fluke causing Hepatitis C like symptoms) **are: 316, 336 kHz**

9.3.2. Swimmer's Itch (Schistosome cercarial dermatitis)

Cercariasis is a parasitic infection caused by *Schistosoma* and its related genera schistosomes.
The definitive host is infected when free-swimming cercariae penetrate the host's skin. In case of human schistosomal infection the cercaria penetrates the skin rapidly and transforms into a schistosomule (larva), which then enters the circulatory system and leaves the skin. The penetration of the skin by cercariae of human schistosomes may not cause any symptoms or cause only a mild inflammation of the skin (dermatitis), especially in hosts exposed previously to cercariae. Schistosome cercariae are unable to differentiate between the skin of animals and human beings. If Schistosome cercariae which do not affect human beings come in contact with the human skin, they will penetrate it just as if it were the skin of their normal host. If this occurs the schistosomules can cause a dramatic inflammatory response, especially concerning hosts previously exposed to cercariae, because (1) human beings are abnormal hosts, (2) the schistosomules remain in the skin for an extended period of time and eventually die there. The inflammatory response that results if schistosome cercariae, which do not affect human beings penetrate the skin of humans is named „swimmer's itch" or schistosome cercarial dermatitis. The cercariae of bovine and avian schistosomes are probably responsible for the majority of cases of swimmer's itch, already known and reported throughout the world. Assuming that somebody swims, bathes, plays, etc., in a natural large body of water in which these cercariae are found, there is virtually no way to prevent an infection. The risk of infection can be lessened by spending less time in the water or by drying oneself completely after coming out of the water. (Some people mention that covering ones body with some petroleum jelly will also decrease the risk of infection, but most people would find this less than appealing.) Schistosome cercarial dermatitis can be extremely uncomfortable and annoying, up to the point of ruining one's vacation, though the schistosomules will not develop into adult worms, and the infection is not life-threatening.
Treatment: by administering Mebendazole and Thiabendazole
RFR method: is to be used after anthelminthic treatment in order to detect and destroy the parasites.
Its resonant frequencies are: 325, 331, 336-348, 352-357, 422, 433-445, 460-480 kHz
The resonant frequencies of Schistosoma haematobium are: 300, 325, 376, 433-443 kHz

9.3.3. Fluke Infections

The trematodes that affect human beings are long-lived parasites producing progressive damage to the tissues of their hosts.

Flukes disease is an infection caused by intestinal fluke, liver fluke, sheep liver fluke, or pancreatic fluke.

Fasciolopsis buskii lives in the small intestine of human beings and pigs. Measuring up to 80 mm in length, it is one of the largest trematodes found in human beings. This parasite is found in numerous countries of the Orient and, as in case of many other parasites that infect human beings, pigs serve as their reservoir host. The life cycle of this parasite is similar to that of *Fasciola hepatica*. The worms produce eggs (up to 25000 eggs/worm/day) that are passed in the host's feces. The first intermediate host is a snail, and the cercariae that emerge from the snail encyst during vegetation. Human beings are infected if they eat vegetation contaminated with metacercariae. Chronic infections caused by this parasite lead to inflammation, ulceration, hemorrhage and abscesses of the small intestine, and which, ultimately can lead to the host's death. The diagnosis of the disease is based on the recovering of eggs in the host's feces. Several books and a number of web sites state that this parasite is either the direct cause or is associated with an increased risk of cancer, HIV, or any number of other diseases affecting human beings. But there is absolutely no evidence whatsoever that this parasite causes cancer, HIV, or any other disease in human beings.

The common name of this parasite, the sheep liver fluke, is somewhat misleading since this parasite is found also in hosts other than sheep (including cattle and human beings), and the parasite resides in the bile ducts inside the liver rather than in the liver itself. This species is the most common parasite of sheep and cattle and, therefore, relatively easy to contract. Thus, in introductory biology or zoology courses, it is often used as „THE" example of a digenetic trematode. *Fasciola hepatica* has been studied extensively by parasitologists, thus there is probably much more known about this species of digenetic trematode than about any other species. The adult parasites reside in the intrahepatic bile ducts, produce eggs there, which are then passed in the host's feces. After passing through the first intermediate host (i.e. snail), cercariae encyst on vegetation. The definitive host is infected by eating contaminated vegetation. The metacercaria excysts in the definitive host's small intestine, after which the immature worm penetrates the small intestine and migrates through the abdominal cavity to the host's liver. The juvenile worm penetrates and migrates through the host's liver, and finally ends up in the bile ducts. The migration of the worms through the host's liver, as well as the presence of the worms in the bile ducts, are responsible for the pathology associated with fascioliasis.

Fasciola hepatica is found in some parts of the United States, in Great Britian, Ireland, Europe, the Middle East, the Far East, Africa, and Australia. Fascioliasis in sheep and cattle causes low milk production and the animals do not gain weight. In many countries, liver originating from animals infected with *F. hepatica* is unsuitable for human consumption, which fact does not only cause a significant economic loss to ranchers and farmers, but results in the loss of an important source of protein, as well. The infection can be diagnosed by finding eggs in the feces of animals and human beings.

Eurytrema pancreaticum may cause an acute or chronic pancreatitis.

Symptoms of Fascioliasis usually include abdominal pain, diarrhea and intestinal obstruction; while liver flukes cause inflammation of the gallbladder and the liver tissues.

Treatment: by administering Praziquantel, Niclosamide, such as Yomesan and Bithionol.

RFR method: the first step to take is to administer anthelminthic drug.

The resonant frequencies of Fasciolopsis buskii are: 333-336, 426-439, 443, 533, 544, 550, 560 kHz

The resonant frequencies of Fasciola hepatica are: 292, 320, 346, 390, 420-430, 484, 560 kHz

The resonant frequencies of Eurytrema pancreaticum are: 418-424, 511-524, 532 kHz

9.3.4. Paragonimiasis

Paragonimiasis is a chronic infection of the lungs caused by the trematodes of the genus *Paragonimus*. Clinically, the disease is characterized by cough and hemoptysis. Ectopic worms may cause a variety of other signs.
The infection is acquired by the ingestion of cysts in the second intermediate host, a crab or a crayfish. The metacercariae excyst in the duodenum, burrow through the intestinal wall into the peritoneal cavity, migrating then usually through the diaphragm into the lung or other organs. The worms may also be found in the liver, the pancreas, the kidney, the mesentery, the skeletal muscle, the subcutaneous tissues, the central nervous system and, particularly, in the brain. Dogs, cats, pigs, rats, raccoons and other wild carnivores, and human beings as well, are definitive hosts for the parasite.
An eosinophilic granuloma is formed about the adult worm, leading eventually to the formation of a fibrous cyst. Pulmonary lesions develop frequently communicating with a bronchiole, resulting in a secondary bacterial infection. The picture shows a kind of chronic bronchitis and bronchiectasis and the production of brownish sputum and hemoptysis. Fibrosis and calcification occur, presenting a picture of tuberculosis. An abdominal mass, pain, and dysentery characterize these intestinal or peritoneal infections. Various types of paralysis and epilepsy occur in case of cerebral involvement.
Diagnosis: by peripheral blood eosinophilia. Definitive diagnosis depends upon finding the characteristic operculated ova in the sputum, stool, pleural fluid, or tissues. Eggs may be rare or totally absent from the sputum. Ziehl-Nielsen staining, often carried out because of a suspected tuberculosis, usually does not show eggs.
A complement fixation test is available, its result correlates well with active infection. CT and MRI are used for brain examinations.
Treatment: by administering Bithionol, Praziquantel, Yomesan, and other anthelminthic drugs.
RFR method: detects the parasite, and after administering antiparasitic drugs, it is to be used in order to eliminate them.
Its resonant frequencies are: 347-351, 430-447, 557-560 kHz

9.3.5. Clonorchiasis

Clonorchiasis is an infection of the biliary passages caused by *Clonorchis sinensis*, one of the most important liver flukes infecting human beings.
Symptoms: though the infection is usually asymptomatic, heavy worm loads may produce manifestations of biliary obstruction.
Diagnosis: by using complement tests, antibody examinations.
Treatment: by administering Chloroquin, Prasiquantel and Yomesan.
RFR method: detects the parasite.
The resonant frequencies of Clonorchis sinensis are: 340, 420-430, 541 kHz

9.3.6. Fascioliasis

Fascioliasis is a parasite infection. This disease is caused by *Fasciola hepatica*, which, like Clonorchis, inhabits the bile ducts of the definitive host. Infections are contracted by the ingestion of the encysted forms of the fluke attached to edible aquatic plants, such as watercress. The larvae excyst in the duodenum, migrate through the intestinal wall, pass into the peritoneal cavity, penetrate the liver capsule, and finally reach the bile ducts, where to mature. Occasionally, larvae may migrate to and mature in ectopic locations, including the subcutaneous tissues, the chest cavity, or the brain.
Fascioliasis produces the so-called liver rot in sheep, being the principal definitive host. Sometimes hares, rabbits, pet rabbits, or other herbivorous animals are the hosts.
Symptoms: Early clinical manifestations are related to the migration of the larval form to the *liver* and within it. Epigastric pain, fever, diarrhea, jaundice, urticaria, pruritus,

arthralgia and peripheral blood eosinophilia may be observed in this stage. Fibrosis of the liver appears similar to that found in clonorchiasis but only after a prolonged residence of many adult worms in the bile ducts. Obstruction of the bile ducts occurs frequently and can be the obvious manifestation of the disease. A *pharyngeal form* of the disease, called halzoun, can result from eating infected raw liver; in this form the young adults attach themselves to the pharyngeal mucosa, occasionally interfering with respiration. The pneumonial form, in which parasites are infiltrated nodes, is rare. The *brain form* causes various symptoms produced by the parasite.

Diagnosis: is based on finding eggs in the feces or in the duodenal contents. Complement fixation, hemagglutination, and precepitin tests, or fluorescent methods have been reported to be helpful. Skin tests are also available.

Differential diagnosis: by distinguishing it from fasciolopsis buskii

Prevention: to prevent infection, aquatic plants such as watercress should not be eaten; vegetables from fields irrigated with polluted water should be boiled before eaten and clean drinking water should be provided for. The liver of sheep, rabbits, deer and other animals in areas where fascioliasis infection is prevalent should never be consumed.

Treatment: by administering Dehydroemetine dehydrocloride, Praziquantel, Yomesan, Bithionol, Metrifonate and other antiparasitic drugs.

RFR method: use after antiparasitic drug treatment, or concurrently with it.

The resonant frequencies of Fasciola hepatica are: 292, 320, 346, 390, 420-430, 484, 560 kHz

This method may be successful in treating brain infections and other forms of infection when antiparasitic drugs are unsuccessful, though it might cause brain edema.

9.3.7. Fasciolopsiasis

Fasciolopsiasis is caused by the large intestinal fluke *Fasciolopsis buskii*, which inhabits the upper part of the intestine of its definitive host. In India, China and other countries pigs are the principal hosts. The large adults attach themselves to the intestinal mucosa, which parts may later ulcerate. The infection is usually asymptomatic in the beginning.

In case of heavy infections, diarrhea and abdominal pain appear soon. Later on, asthenia with ascites, anasarca and toxicosis will occur. Fasciolopsis has the same life cycle as other worms: egg, miracidia, redia, cercaria, metacercaria and adult worm. Fasciolopsis lives in the intestines producing eggs. The eggs hatch in water, can swim vigorously having cilia; the larvae must find an intermediate snail host in about one to two hours as, if not, they may be too exhausted to invade. They develop then within the miracidia in form of little balls, until they are expelled. These are the mother redia, bearing each daughters redia; continue to reside within the snail for up to eight months, living on the fluids in the lymphatic spaces. Similarly to the mother redia, the daughter redia produce cercaria continually. These have tails, enabling the cercaria to exit from the snail and swim to a plant. If the snail is feeding on the plant, cercaria can latch onto plants with their sucker mouths and start to encyst within minutes. Then the tail breaks off and swims away to dissolve. Metacercaria have a two-walled cyst, the outer wall of which is very sticky. Being ingested by a host the cyst-wall protects it against being chewed up, and the keratin-like coat prevents it from being digested by stomach juices. Reaching the duodenum, the contact with the intestinal juices dissolves away the cyst-wall and makes it free. It then fastens itself to the intestinal lining and begins to develop into an adult.

Diagnosis: by the examination of eggs in the feces.

Differential diagnosis: by distinguishing it from other worms, f.i. fasciola hepatica.

Treatment: by administering Praziquantel, Yomesan and other antiparasitic drugs.

RFR method: is to be done together with drugs for this parasitic infection.

Its resonant frequencies are: 333, 346, 426-438, 443, 533 kHz

9.3.8. Metagonimus Yokogawai

Metagonimus yokogawai species less-commonly cause human intestinal fluke infections. Intestinal flukes usually cause inflammation, ulceration and intensive mucous secretion at the locus of its attachment. In case of certain severe infections the flukes cause intestinal obstruction or malabsorption, leading to hypoalbuminemia, protein-losing enteropathy, and even to an impaired vitamin B-12 absorption. *M. yokogawai* is 1-2.5 mm in length and 0.4-0.75 mm in width. Its life cycle is similar to that of *Heterophyes heterophyes.* The adult worm produces eggs in the human intestine which are excreted in the feces. The eggs, getting into water, infect their first intermediate hosts, snails, fishes, (f.i. ayu, golden carp) in which the eggs undergo their developmental cycle becoming cercariae. Cercariae infect their second intermediate hosts, the freshwater fishes, in which they become metacercariae. Metacercariae infect people, who ingest raw or undercooked fish. The flukes invade the mucosa of the small intestines, causing there inflammation and ulcerations. Flukes eventually become encapsulated. Patients infected with *M yokogawai* suffer from mucous diarrhea and vague abdominal symptoms, f.i. ulcer-related abdominal pain, dyspepsia, nausea, vomiting, diarrhea and a loss of weight as well. The prognosis of this disease is usually good, except in case of embolization. The illness may be asymptomatic; too.

Its resonant frequencies are: 436-443 kHz

INFECTIONS OF SPECIAL ORGANS

10. DISORDERS OF MENTAL HEALTH, BRAIN AND NERVES

The disorders of mental health involve the disturbances in thinking, emotion and behavior. These disorders are caused by complex interactions between physical, psychological, social, cultural and hereditary factors and influences.
The diseases of the peripheral nervous system proved to be the most difficult subjects in neurology. Since the structure and the function of this system are relatively simple, one might suppose that the knowledge of the affecting diseases would be complete. Thus far only very limited possibilities are known in the treatment of these disorders with RFR method.

10.1. Depression

Depression is a feeling of intense sadness. Its causes are not yet cleared up. Many factors can contribute to make a person feel depressed, f.i. familial troubles, existential uncertainty, side effects of certain medicaments, an introverted personality, emotionally upsetting events, particularly which involve some loss. Depression may come about or get worse without showing any apparent or significant signs.
Changes in hormone levels can create a change of mood shortly before menstruation. Some other types of depression include: grief, reactive depression, manic-depressive psychosis, involutional melancholia and hypochondriasis.
There are three main types of depression the physician should be acquainted and become familiar with, without reverting to an elaborate classification. The first, the reactive depression, is by far the most common form of it and is typified by grief reaction. The manic-depressive psychosis is the second type. It represents the standard model for the psychotic depression and thus gets often an incorrect diagnosis made by non-psychiatric physicians. The third type, the involutional melancholia deserves recognition and attention since it is frequently encountered in general practice and carries an excellent prognosis if given a proper treatment.
Depression following a tragic event, f.i. the death of a loved one, is named situational depression or exogenous depression. Depression without an apparent precipitating causative event is the so-called endogenous depression. However, these distinctions are not too important in regard to all cases concerning the effects and the treatment of the illness.
Depression may also occur caused by a number of physical diseases and disorders. A person falling into depression may appear to be slow, sad, irritable and anxious. Someone, who tends to withdraw, speaks and sleeps little, stops eating, has a so-called vegetative depression. Someone who becomes very restless, for example wrings his/her hands, etc. experiences the so-called agitated depression.
A feeling of insecurity and worthlessness may lead the severely depressed people to believe that they are being watched and persecuted. These kinds of depressions concomitant with delusions are termed psychotic depressions.
Depression can be caused by:
I.A. Infectious diseases, f.i.
AIDS
Influenza
Epstein-Barr Virus e.g. Infectious mononucleosis
Viral hepatitis
Viral pneumonia
Some other viral infections
Shigella flexneri

Tuberculosis
Mycoplasmosis
Lyme borreliosis
Syphilis - late stages
Trypanosomiasis

I.B. Infectious diseases in a larger sense, such as some autoimmune diseases and tumours:

Rheumatoid arthritis
Systemic lupus erythematosus
Multiple Sclerosis
Parkinson's disease
Abdominal cancer
Brain tumours

II. Neurological disorders

Head injuries
Sleep apnea
Stroke
Temporal lobe epilepsy

III. Nutritional disorders

Pellagra
Pernicious anemia
Vitamin Other B deficiencies

IV. Hormonal disorders

Addison's disease
Cushing's syndrome
Low levels of pituitary hormones
Low levels of thyroid hormones
High levels of parathyroid hormones

V. Side-effects of drugs

Amphetamines
Antipsychotic drugs
Beta-blockers
Cimetidine
Contraceptives
Cycloserine
Indomethacin
Mercury substances
Methildopa
Ranitidine
Reserpine
Thallium
Vinblastine
Vincristine

The conventional treatment of depression:

Tricyclic and similar antidepressants are, as follows: Amitriptyline, Amoxapine, Bupropion, Clomipramine, Desipramine, Doxepin, Imipramine, Maprolitine, Nefazodone, Nortriptyline, Protriptyline, Trazodone, Trimipramine, etc.

Serotonin-norepinephrine reuptake inhibitors (SNRI) are f.i.: Fluoxetine, Fluvoxamine, Paroxetine, Sertraline, Venlafaxine, etc.

Monoamine oxidase (MAO) inhibitors are f.i.: Isocarboxazid, Pargyline, Phenelzine, Tranyl-cypromine, etc.

Psychostimulants are f.i.: Dextroamphetamine, Methylphenidate, etc.

The RFR method can be used in those cases of depression that have their origin in certain infectious diseases. Concerning these depressions specific pathogens (mostly viruses or bacteria) can be found in patients and very often in the family of the patients, too.
For example, Shigella flexneri infections often cause a certain type of depression. As the Shigella flexneri is the causative agent of this depression of infectious origin, it can easy be eliminated with RFR method. The depression can be caused by the Shigella toxin itself, though an other pathogen's infection (e.g. the *Epstein-Barr Virus* and other viruses), frequently associated with *Shigella* or *Borrelia* groups, can also be involved in the persistance of the depression. Depressive states or diseases can often be traced in the family tree of these patients, so that it seems as if they had inherited them. Indeed, some kind of predisposition in the family does certainly play a role in the appearance of the disease, but this might only be referable to the cumulative occurrence induced by multiple factors. The presence of the depression as a disease indicates a specific deficiency of the person's immune system, allowing the Shigella to manifest a chronic depression.
The resonant frequencies of Shigella flexneri are: 313, 318, 389-396, 403-410, 423-425, 499 kHz
The resonant frequencies of Borrelia groups are: 378-387 kHz
The resonant frequencies of Epstein-Barr Virus are: 339, 342-347, 370-385, 397-398, 403, 422, 491, 518, 528 kHz
The Epstein-Barr Virus can often be present as a latent infection in the brain for several years.
As to the depressive states caused by other pathogens mentioned above, the RFR method is effective by using the resonance frequencies given in the Chapters dealing with the relating pathogens.

10.2. Bipolar Disorder

Bipolar disorder, named also Manic-depressive illness (MDI), is one of the most common, severe and chronic mental illnesses, characterized by periods of prolonged, deep and profound depression that alternate with periods of an excessively elevated and/or irritable mood known as mania. Bipolar disorder has a number of contributing factors, including genetic predisposition, biochemical neurotransmitters, psychodynamic effects, different environmental elements and certain chronic infections, such as *Borreliosis, certain Streptococcus infections, Mycoplasmal infections, Toxoplasmosis,* and several various viral infections caused f.i. by *HSV, CMV, EBV, Parvovirus B19* and *HTLV.*
The symptoms of mania include a decreased need for sleep, pressured speech, increased libido, reckless behavior without regarding consequences, grandiosity, and severe thought disturbances, which may or may not include psychosis. In the time between mania and depression, patients can usually lead a productive life, but in some cases, depression and mania may rapidly alternate, which is named rapid cycling. Extreme manic episodes can sometimes lead to psychotic symptoms such as delusions and hallucinations. This disorder can be sorted into bipolar disorder type I, bipolar disorder type II, cyclothym and other types, based on the nature and severity of the experienced mood episodes. Episodes of abnormality are associated with distress and disruption, and an elevated risk of suicide, especially during depressive episodes. In some cases it can be a devastating long-lasting disorder; in others it can be associated with creativity, and positive achievements.
Depressive suicide episodes: depressed patients have a very high rate of suicide. They are those who attempt and succeed in killing themselves. Query patients whether they have any thoughts of hurting themselves (suicidal ideation) and any plans to do so. The more specific the plan, the higher the danger.

Manic agression episodes: persons in manic period can be openly combative and aggressive. They have no patience or tolerance for others. They can be highly demanding, violently assertive, and highly irritable. The homicidal element particularly emerges if these individuals have a delusional content to their mania. They maintain the grandiose belief that others must obey their commands, wishes, and directives. If their delusions become persecutory, they may defend themselves against others in a homicidal fashion.

Infectious Agents in Schizophrenia and Bipolar Disorders: the idea that schizophrenia and bipolar disorders can be caused by infections is not a new one.

New researches are being continued in this field, increasingly aided by an impressive technologic development in microbiology and virology. Reports of the past decades document the presence of an infection caused by *influenza virus, rubella virus, bovine disease virus*, and other infectious agents in patients with schizophrenia and bipolar disorders, and show the presence of certain infectious agents in pediatric autoimmune neuropsychiatric disorder associated with *streptococcal* infections (PANDAS) and in Obsessive-Compulsive Disorders.

An additional important reason to look for infectious agents in case of schizophrenia and bipolar disorders is that CNS infections caused by certain specific pathogens frequently mimic the clinical symptoms of primary psychiatric diseases. For example, Caroff and his colleagues reviewed 108 cases of psychiatric disorders resulting from suspected or confirmed CNS viral infections; while in 62 cases, a specific virus was found to be implicated, including *HIV, HSV1, HSV2, EBV, CMV, Measles, Mumps, Coxsackie and Influenza viruses*. The fact that the spirochete bacterium *Treponema pallidum* can cause the symptoms of schizophrenia in case of syphilis, is well known to the psychiatric clinicians. Infections caused by the spirochetes Borrelia Burgdorferi sensu lato *can also be* associated with schizophrenia-like symptoms in some persons.

Two additional studies reported an increased level of Toxoplasma gondii antibodies in the late-pregnancy serum of women giving birth to infants in whom, later on, schizophrenia developed.

Syphilis and Lyme disease (combined with a *Mycoplasma fermentans* infection) **may cause neuropsychiatric illnesses such as neurosyphilis and neuroborreliosis,** as well as a broad range of psychiatric reactions associated with Lyme disease, including paranoia, dementia, schizophrenia, bipolar disorder, panic attacks and others. In case of bipolar disorders the pathologic reaction of the involved immune system is autoimmunity. Autoimmune diseases develop usually when the patient's immune system becomes confused and can no longer distinguish between the body's own tissue antigens and those which belong to an outside threatening agent. As a consequence of this immune confusion, the body's own tissues get attacked by the person's immune system. However, there is no clear evidence that autoimmunity is necessarily involved in bipolar disorders. Mycoplasmal, borrelial and several different bacterial and viral antigens are adsorbed to the brain tissues where an autoimmune response to the neurotransmitter system of the brain does develop.

Genetic predisposition: bipolar disorders, especially the bipolar disorder type I, has a significant genetic component. Numerous genetic studies suggest that multiple different genetic loci, each of them of small effect, contribute to the affected phenotype. According to the recently published 3 largest studies 2 protein- coding genes are implicated that either regulate or are but subunits of ion channels ANK3 and *CACNA1C*. These findings suggest that the bipolar disorder, similar to epilepsy, might be, in part, an ion channelopathy. Another candidate gene thought to be associated with mania is the *CLOCK* gene involved in circadian periodicity.

Biochemical factors of MDI

Calcium channel blockers are used to treat mania, which symptom can be also a result of the disruption of calcium regulation in neurons. The supposed disruption of calcium

regulation may be caused by various neurologic insults such as excessive glutaminergic transmission or ischemia. Interestingly, valproate up-regulates specifically the expression of a calcium chaperone protein GRP 78, being one of its chief mechanisms concerning the cellular protection.
The levels of several different neurotransmitters change in case of MDI, as well as the number of receptors of neurotransmitters.
The subtypes of MDI are:
Bipolar disorder type I: For the diagnosis of Bipolar disorder type I there are one or more manic or mixed episodes required. A depressive episode is not required for the diagnosis of Bipolar disorder type I, though it does frequently occur.
Bipolar disorder type II I is characterized by hypomanic episodes rather than by actual manic episodes, and by at least one major depressive episode. Hypomanic episodes do not reach the full extremes of mania, which fact can make the Bipolar disorder type II more difficult to diagnose, since the hypomanic episodes may simply appear as a period of successful high productivity and is reported less frequently than a distressing, crippling depression. Concerning the disorders Bipolar disorder type I and II, a number of specifiers indicate the form and course of the disorder, including "chronic", "rapid cycling", "catatonic" and "melancholic".
Cyclothymia involves the presence or the history of hypomanic episodes with periods of depression that do not own the criteria for major depressive episodes. The diagnosis of Cyclothymic disorder requires the presence of numerous hypomanic episodes, intermingled with depressive episodes that do not fully meet the criteria for major depressive episodes.
Bipolar Disorder NOS, sometimes called "sub-threshold" Bipolar Disorder, is a "catch-all" diagnosis that is used to indicate bipolar illness that does not fit into any of the formal DSM-IV bipolar diagnostic categories.
Rapid cycling, however, is a course specifier that may be applied to any of the above mentioned subtypes. It is defined as having four or more episodes per year and is found in a significant group of individuals with bipolar disorder.
Diagnosis: symptomatically. Although there are no biological tests which confirm bipolar disorders, tests should be carried out to exclude organic illnesses such as hypo- or hyperthyroidism, metabolic disturbances, systemic infections or chronic diseases (such as Syphilis, Borreliosis, Toxoplasmosis, Mycoplasmal infections, HIV or other HTLV infections), By special braine examinations, such as EEG, CT scan, functional magnetic resonance imaging (fMRI) and PET.
The diagnosis of bipolar disorder in children is particularly difficult. Patients showing some bipolar symptoms tend to have a rapid-cycling or mixed-cycling pattern that may not meet the MDI criteria.
Differential diagnosis: by distinguishing it from schizophrenia, schizoaffective disorder, drug intoxication, brief drug-induced psychosis, schizophreniform disorder and borderline personality disorders.
Treatment: by administering *Mood stabilizing medicaments*. (f.i. lithium, valproic acid, lamotrigine, gabapentin, topiramate, oxcarbazepine, etc. Except lithium, many of these drugs are anticonvulsants. (People taking anticonvulsants should be closely monitored for new or worsening symptoms of depression, suicidal thoughts and behavior, or any other unusual changes in their mood and behavior.) *Atypical antipsychotic medicaments* (f.i. olanzapine, aripiprazole, quetiapine, risperidone and ziprasidone) are sometimes also used to treat the symptoms of bipolar disorder. *Antidepressant medicaments* (f.i. fluoxetine, paroxetine, sertraline and bupropion) are sometimes used to treat symptoms of depression in bipolar disorder. People with bipolar disorder who take antidepressants take often also a mood stabilizer. The taking of only an antidepressant can increase the person's risk of

switching to mania or hypomania, or of developing rapid cycling symptoms. Lamotrigine seems to be helpful in controlling the depressive symptoms of bipolar disorder.
Antidepressants are safe and popular, but some studies suggest that they may have unintentional effects on some people, especially concerning adolescents and young adults.
Antibiotic treatments: As to the treatment of these special infections, see their special Chapters. The antibiotic treatment of Syphilis and Borreliosis (Lyme disease) may cause a **Jarisch-Herxheimer** reaction, occuring when large quantities of toxins of dying spirochetes are released into the body. These toxins can increase the symptoms of MDI and cause highly irritable, manic aggression, temporary psychosis and other psychotic symptoms. Non steroid inflammatory drugs, antipsychotic medicaments, antidepressants and anxiolytic drugs (f.i. meprobamate) can sometimes decrease these symptoms.
RFR method: detects and can eliminate all pathogen microorganisms.
As to the most frequent resonances, see the special Chapters of infection. Jarisch-Herxheimer reaction does occur in case of spirochetal infections. If in case of MDI of infectious origin solely a pharmacotherapy is given, it does not eliminate all the microorganisms (such as viruses and antibiotic resistant microorganisms) in the brain. The regulation of bipolar mood swings by pharmacotherapy together with RFR method is based on the use of medicaments facilitating the regulation of these mood swings and of certain neurochemicals in order to restore a normal mood and cognition state.
MDI is optimally treated first of all in Psychiatric Institutes.
See the frequencies the special Chapters.

10.3. Delirium and Dementia

The delirium is a potentially reversible condition that usually comes on suddenly; the patient has a diminished ability to pay attention, is confused, disoriented, and unable to think clearly. Delirium can be caused by many a factor. The RFR method is only effective if the delirium is caused dominantly by bacterial or viral infections.
Dementia is a decline in the mental ability usually progressing slowly, when memory, thinking, judgment and the ability to pay attention and to learn are impaired. The patient's personality may also deteriorate. Dementia can develop due to a severe injury, diseases, toxic substances, or the anoxia of the brain.
The most common cause of dementia is the Alzheimer's disease.
RFR method: is useful in detecting and eliminating the bacterial or viral pathogens being causative cofactors of dementia.

10.3.1. Alzheimer's Disease

The most common form of dementia is the Alzheimer's disease. The real and only causes of the Alzheimer's disease are not known, certain genetic factors can play a role. The illness seems to run in some families and be caused or influenced by several specific gene abnormalities. An other cofactor causing Alzheimer's disease may be one of the species of the *Aspergillus fungi* (which one, yet it is not known). Its spreading in the brain results in a chronic process of the disease characterized by the building-up a certain toxic level of amyloidal proteins in the brain and by the destruction of those regions next to the place of the accumulation (see Aspergillosis infection in the brain, Chapter 7.2.8.). The organism of patients suffering from Alzheimer's diesease is unable to break down these amyloidal fibers. The accumulation of these fibers, the so-called amyloid plaques reach neurotoxic levels, compress the nerve fibers lying in their path and lead to the formation of brain lesions characteristic of the Alzheimer's disease. The destruction of these cerebral tissues causes thus behavioral changes associated with Alzheimer's dementia.
An other type of fungi which may play a role in the development of the Alzheimers's disease are those of the *Cryptococcus group*. These fungi may also be found in the brain of

Alzheimer's patients (see also Chapter 7.1.3). In case of Alzheimer's disease, some parts of the brain degenerate, destroying cells and reducing the responsiveness of the remaining ones concerning the chemicals transmitting signals in the brain. Abnormal tissues, named senile plaques, neurofibrillary tangles and abnormal proteins will appear in the brain.
The neurodegenerative Alzheimer's disease can also be associated with chronic pathological calcifications (see Chapter 6.22.). *Nanobacteria* secrete a slimy, calcium-containing biofilm around them that subsequently hardens, creating a calcium wall. They can cause apoptosis or cell-death in all brain tissues they come in contact with. Nanobacteria divide very slowly, so that the Alzheimer's neurodegenerative process can take 20-40 years to develop.
The **Diagnosis** happens usually by testing the mental status, CT or MRI scan can be helpful.
By the **conventional treatment** the disease can not be cured, nor can it stop the development and progression of dementia. Tacrine and donepezil (centrally-acting cholinesterase inhibitors) may slacken the progression of the disease for a short period of time (i.e. for about a year).
The **RFR measuring** can find the pathological resonances.
The most frequent frequencies of Alzheimer's disease are: 287-289, 291-293, 303-308, 317-318, 324-326, 328, 341-356, 372, 382-387, 390-394, 401, 403-410, 442-451, 466-468, 477, 482, 492-496, 504-505, 520, 534-536, 568-578 kHz
As can be seen, there are more pathogen agents involved in Alzheimer's disease. It is also very important to note that patients suffering from Alzheimer's disease have low frequencies as well, indicating the presence of molds and Aspergillus fungi. The Aspergillus species contain very strong toxins which can damage the liver and the brain.
The list of possible participants that may play a role in the pathological process of Alzheimer's disease includes: species of *Borrelia Burgdorferi s.l., Chlamydia, Molds, Aspergillus, Herpes viruses (EBV, CMV, HZV), Measles viruses*, other unknown viruses and *Shigella bacteria.*
The species of Borrelia Burgdorferi sensu lato and Chlamydia can cause autoimmune processes as well.
It is crucial to stop the mental deterioration before it becomes irreversible.
RFR method: can detect and eliminate the pathogen agents thus stopping the autoimmune cascade.

10.3.2. Alzheimer's-like Dementia in Neuroborreliosis

Neuroborreliosis can show similar symptoms to those seen in demyelinating and inflammatory autoimmune neurologic disorders such as Multiple Sclerosis, systemic lupus erythematosus, Alzheimer's disease and other cerebrovascular diseases.
Borrelial encephalomyelitis can show prominent pyramidal, sensorial and cerebellar syndromes with MRI lesions resembling demyelination and vasculitis as well, so that the patients suffering Lyme disease may be mistakenly diagnosed as if they had Multiple Sclerosis or other diseases mentioned above.
The Alzheimer's-like brain damages caused by Lyme disease among adults is similar to those experienced in case of meningovascular and cerebrovascular syphilis, CNS microvasculitis and CNS macrovasculitis causing clinical symptoms and MRI changes as well. In case of progression symptoms include confusion, anger, mood swings, language breakdown, long-term memory loss and general withdrawing of the sufferer as his or her senses weaken. Among the common neuropsychiatric manifestations are irritability and labile affects, leading to crying and outbursts of unpremeditated aggression and physical violence, even among patients whose life-long behavior had been formerly peaceful. These patients can have illusionary misidentifications and other delusional symptoms. Because of this communication deficit together with delusions, patients often resist if their caregivers

attempt to take care of them. The talking of the patient may be reduced to simple phrases or even single words, or even to the complete loss of speech. Though aggressiveness can still be present, an extreme apathy and exhaustion are much more common. Urinary and defecation incontinency can also develop.
Alzheimer's-like damages develop if the *Borrelia* infection is coinfected with *Mycoplasma*. *HTLV* is also frequently associated with such Alzheimer's like diseases.
Diagnosis: by detecting borrelia using ELISA, Immunoblot, PCR, etc. Dementia is by definition a clinical condition, not an exact diagnosis. Alzheimer's disease is usually diagnosed clinically from the patient's history, collateral history from his/her relatives, and clinical observations, based on the presence of characteristic neurological and neuropsychological features. Examinations by CT, MRI, SPECT scan and PET are generally used to help to diagnose the subtype of dementia and to exclude other cerebral pathologies. Memory testing, intellectual functioning tests can characterize the severity of dementia.
Treatment: see Chapter 6.20.3. (Borreliosis) and symptomatically.
RFR method: detects and eliminates the Borrelia. Use the RFR method together with administering iv. antibiotics given in effective doses for a long time.
The most frequent resonances of Borreliosis are: 377-387 kHz
The most frequent resonances of Mycoplasma are: 442-451 kHz
The most frequent resonance of HTLV are: 370-376 kHz

10.4. Insomnia

Sleep disorders are disturbances concerning the falling asleep, staying asleep, or the duration of sleeping or abnormal sleeping behaviors such as experiences of night terrors or waking in sleep. Insomnia means that after awakening people feel as if they had slept less, than enough. Insomnia isn't a disease, only a symptom of different causes, including emotional disorders such as stress, anxiety or nervosity and depression, pain, medical drugs and a variety of infections owing to microorganisms. Awakening in the early morning happens more often among elderly people, nethertheless, it can be a may be a sign of depression concerning any age. Brain damage and infection cause insomnia, such as encephalitis, stroke, Alzheimer's disease, Parkinson's disease, cerebral arteriosclerosis, high blood pressure, *Borrelia infections*, rheumatoid arthritis, Multiple Sclerosis, brain tumor, *AIDS, influenza, syphilis, tuberculosis*), *viral hepatitis or pneumonia, Shigellosis and other* infectious diseases as well.
Diagnosis: by neurological examinations, EEG, the primary insomnia has to be distinguished from the secondary one.
Treatment: by administering sedatives, hypnotics, narcotics, minor tranquilizers, antianxiety, antihistamines and antidepressants.
RFR method: look for the causal relation concerning infectious insomnia, detect and eliminate the pathogen microorganisms!

10.5. Connatal Rubella Syndrome

(We advise never to treat pregnant women using the RFR method!)
Rubella is a viral illness caused by a toga virus of the genus *Rubivirus*. This disease is mostly affecting children, its symptoms include low-grade fever, headache, malaise, mild coryza and conjunctivitis. If rubella infection occurs during pregnancy, especially during the first trimester, a transplacentar fetal infection is likely to happen, and can cause connatal rubella syndrome (CRS), ending in abortions, miscarriages, stillbirths and even severe birth defects. If the infection develops within 0-28 days before conception, there is a cc 40% danger that the infant will be affected. But, if the infection develops within 0-12 weeks after conception, there is an about 50% risk that the infant will be affected.

However, if the infection develops within 13-26 weeks after conception, there is an about 23% of danger that the infant will be affected. Usually, infants are not affected if they contract rubella during the third trimester of pregnancy, or, within 26-40 weeks after conception. The most common connatal defects are cataracts, heart diseases, sensorineural deafness and mental retardation.
The illness of infants, having CRSs, should as early as possible be diagnosed, in order to prevent the further spreading of the virus. Moreover, an early diagnosis will facilitate the early intervention against specific disabilities. Infants with CRS may shed the virus for a prolonged period and have to be considered to be infectious until they are at least 1 year old, or, until their urine and pharyngeal viral cultures, taken every month, remain to be repeatedly negative concerning rubella.
CRS can result in serious birth defects such as: mental retardation, microcephaly, meningoencephalitis, malformations of the heart (especially patent ductus arteriosus, peripheral pulmonary artery stenosis), brain damages, deafness, spleen, liver and bone-marrow problems, eye defects (especially cataract, glaucoma, pigmentary retinopathy and microphthalmia), thrombocytopenic purpura, hepatomegaly and low birth weight. Presence of any of these defects or laboratory data consistent with connatal rubella infection are the diagnostic criteria of the disease.
Children, exposed to rubella in their mother's womb have to be closely watched as they are aging after birth, for the appearance of any of the following signs: developmental delay, schizophrenia, growth retardation, learning disabilities, diabetes, glaucoma.
Prevention: The goal of a rubella vaccination program is to prevent CRS.
Diagnosis: by isolating the rubella virus, or by finding of rubella-specific immunoglobulin M (IgM) antibodies, or by finding persisting high level antibodies of the infant for a longer period than expected in case of a passive transfer of maternal antibodies. By experiencing rubella-specific IgM antibodies in the infant's cord blood or sera. As concerns infants with CRS, IgM antibodiespersists for at least 6-12 months.
Documentation of the persistence of serum rubella IgG titer beyond the time expected from the passive transfer of maternal IgG antibody; isolation of the virus, which may be shed from the throat and the urine for a year or longer; or the detection of rubella virus by PCR and by immunofluorescent antibody assay (IFA), which is a rapid and sensitive methos.
Vaccination: although the use of rubella vaccine is contraindicated in case of pregnant women or women planning to get pregnant within 3 months, an inadvertent administration of the vaccine to pregnant women can occur.
RFR method: it is not yet allowed to treat pregnant women with the RFR method!
Two months before pregnancy the rubella virus must be detected and eliminated.
The most frequent resonances of the rubella are: 372, 402, 440, 450-451, 468, 520-530 kHz
The most frequent resonances of the vaccine are: 425, 438-439, 468-473, 516-520 kHz

10.6. Reye's Syndrome

The Reye's syndrome is a life-threatening disorder causing inflammations of the brain and a rapid accumulation of fat in the liver. The illness develops typically after a viral disease, particularly f.i. in case of an upper respiratory tract infection, influenza, varicella, or gastroenteritis, if *aspirin* is administered during the infection. Some discoveries of the inborn errors of metabolism show manifestations similar to those of the Reye's syndrome. By administering less aspirin to children the occurrence and the diagnosis of the Reye's syndrome became more rare.
In case of this syndrome an acute inflammatory or noninflammatory encephalopathy will develop with an altered level of consciousness, cerebral edema, hepatic dysfunction and abnormality. It is characterized by a pathological fatty metamorphosis and a more than 3-fold increase in the alanine aminotransferase (ALT) levels, the aspartate aminotransferase

(AST)levels, and/or the ammonia levels of the serum. Its other processes include fatty-acid oxidation defects, amino-acidopathies and organic acidopathies, urea-cycle defects, and disorders of the carbohydrate metabolism. The pathogenesis of the disease is a mitochondrial dysfunction which inhibits the oxidative phosphorylation and the fatty-acid beta-oxidation and is present in a virus-infected, sensitized host, in whom the administered salicylate drugs will stimulate the expression of inducible nitric oxide synthase and provoke an autoimmune process.
The histologic changes are cytoplasmic fatty vacuolization in hepatocytes, edema in astrocytes and loss of neurons in the brain, as well as edema and fatty degeneration of the proximal tubules in the kidneys. The affected cells have pleomorphic, swollen mitochondria reduced in number, glycogen depletion and there is a slight inflammation in the affected tissues to be found. The hepatic mitochondrial dysfunction causes hyperammonemia, which is thought to induce astrocytic edemas, cerebral edemas causing an increased intracranial pressure.
Influenza B, Influenza A and *Varicella-Zoster Virus* are the most often involved viruses in this illness. Other viruses f.i. the *Parainfluenza viruses*, the *Adenoviruses*, the *Coxsackie viruses A and B*, the *ECHO viruses*, the *Epstein-Barr Virus*, the *Rubella virus*, the *Measles virus*, the *Cytomegalovirus*, the *Herpes Simplex Virus* and the *Polioviruses* are less commonly involved. Reye's syndrome can also develop after vaccination with live viral vaccines.
The symptoms of the disease include headache, sleepiness, vomiting, diarrhea, hyperventilation, irritability, restlessness, delirium, seizures starting 12 hours to 3 weeks after the viral illness, and then even lethargy, dehydratation, jaundice, flaccid paralysis, decerebrate rigidity and coma can develop. Other triggering components besides the salicylate may be the insecticides, herbicides, aflatoxins, paints, paint thinners, hepatotoxic mushrooms, hypoglycin in akee fruit, margosa oil, paracetamol, outdated tetracyclines, valproic acid, zidovudine, didanosine, antiemetics. The most frequent drug classically associated with Reye's syndrome is the aspirin. These above mentioned substances will transform to hapten molecules in the patient's body becoming a new antigen.
Diagnosis: can be done by using laboratory examinations f.i. liver function testing, enzyme examinations, neurological detections, CT, EEG.
Treatment: can only be done symptomatically. The administering ammonia detoxicants, anti-inflammatory drugs and antiviral agents may help.
RFR method: detects and may eliminate the infective viruses.
As to the frequencies of **Influenza viruses,** see Chapter 5.1.1. Treat the illness on the frequencies of the actually infecting influenza virus!
As to the frequencies of Human T-cell Lymphotropic Viruses: see Chapter 5.1.10.
The frequencies of Herpes Zoster Virus are: 293, 339, 402, 409-410, 416-421, 450, 467 kHz
As to the frequencies of Herpes Simplex Virus-1 and 2: see Chapter 5.2.4.1.
The frequencies of Cytomegalovirus are: 305, 327, 345-350, 406-412, 530-536 kHz
The frequencies of Coxsackie virus are: 287-290, 294-303 kHz
The frequencies of Rubella virus are: 372, 402, 440, 450-451, 468, 520-530 kHz
The frequencies of Measles virus are: 364-373, 381-387, 390, 402-407, 450-456, 478, 492, 522-536, 564 kHz
The frequencies of ECHO viruses are: 317-319, 369, 397-405, 470-471, 526 kHz
The frequencies of Epstein-Barr Virus are: 337-339, 342-347, 372-382, 422, 441, 518 kHz
The frequencies of Mycoplasma fermentans are: 442-444, 447-451, 493-495 kHz

10.7. Connatal Syndromes with Vascular Defects

The **Sturge-Weber syndrome** (SWS), named also encephalotrigeminal angiomatosis, is a very rare connatal neurocutaneous disorder with angiomas. The disease is a phakomatosis („mother-spot" disease) consisting of hamartomatous malformations, which are initially connatal, light pink-coloured, macular lesions which later on become dark-red or purple nodular lesions. These lesions usually affect the skin of the face, or even other areas of the body and are associated with lesions in the choroidal vessels of the eye or the leptomeningeal vessels of the brain. The characteristic inflammations and calcifications in the external layers of the cerebral cortex which are to be found under the angiomatosis, associated with ipsilateral, cortical atrophy, develop frequently, progressing with age. These alterations occasionally extend anteriorly to the frontal and temporal lobes.
The clinical manifestations of the SWS have a common embryological basis. The primary defect is a developmental insult affecting the precursors of tissues originating from the promesencephalic and the mesencephalic neural crest. These affected precursors give then rise to vascular and other tissue malformations of the meninges, the eye, and the skin.
Though the exact nature of the insult is not known, it is said that a somatic mutation of these precursors may lead to the overproduction of an angiogenic factor. The cause of this disease may be an infection of the mother caused by *Herpes simplex virus*, or *HZV,* or *Coxsackie virus*, or *Adenovirus*, or *Cytomegalovirus* or/and by *Mycoplasma species*, which pathogen agents infect the embryo through the placenta, too. These pathogens can be demonstrated both in case of the mother and her newborn. This infection is combined by one of these viruses with an other pathogen, such as the mycoplasma.
An incomplete SWS results from the same developmental defect, but does affect only those cells, the clonal progeny of which are destined for the affected tissues. In contrast with other phakomatoses in which clear-cut hereditary patterns are often evident, the influence of heredity in SWS has not been documented. Up day, no gene defect had been associated with the syndrome. Several types of chromosomal abnormalities are reported, but most patients with SWS have normal karyotypes. Most patients suffering from SWS have a sporadic, nonfamilial disease.
The SWS is caused by residual embryonal blood vessels and their secondary effects on the surrounding brain tissues. A vascular plexus develops around the cephalic portion of the neural tube, under the ectoderm destined to become the facial skin. Normally, this vascular plexus is formed on the sixth week and regresses cc. on the ninth week of gestation. A failure of this normal regression results in residual vascular tissues, which form the angiomas of the leptomeninges, the face, and the ipsilateral eye.
The secondary effects on the surrounding brain tissue result in neurologic dysfunctions including hypoxia, ischemia, venous occlusion, thrombosis, infarction, or vasomotor reactions.
Symptoms: at the child's birth, seizures accompanied by a large port-wine stain birthmark on the forehead and the upper eyelid of one side of the face indicate the diagnosis of *Sturge-Weber syndrome*. The birthmark can vary in color from light pink to deep purple, and is caused by an overabundance of capillaries around the ophthalmic branch of the trigeminal nerve, just beneath the surface of the face. There are malformations of the blood vessels in the pia mater overlying the brain, at the same side of the head as the birthmark is. These cause calcifications of the tissues and cause the loss of nerve cells in the cerebral cortex. The neurological symptoms include seizures beginning in infancy which may worsen with age. Convulsions, varying in severity, usually happen on the side of the body opposite the birthmark. Moreover, there may be even muscle weakness on the same side. Some children will have developmental delays and mental retardation; most of them will have glaucoma (increased pressure within the eye), present at birth or developing later on. An increased pressure within the eye can cause enlarged eyeball and bulge out of its socket (buphthalmos). The Sturge-Weber syndrome rarely affects other organs of the body.

The **Klippel-Trénaunay-Weber syndrome** consists of port-wine stains on the extremities and the face, as well as hemihypertrophy of the soft tissues and the bony tissues, and in addition to all this, the characteristics of SWS. This syndrome is sporadic just like SWS. In case of Klippel-Trénaunay-Weber syndrome, an association exists between hemihypertrophy and solid visceral tumors, mostly affecting the kidney, the adrenal gland, or the liver.

The **Beckwith-Wiedemann syndrome** is characterized by a facial port-wine stain, macroglossia, omphalocele and visceral hyperplasia. There is certain associated risk of visceral neoplasia. Severe hypoglycemia resulting from pancreatic islet-cell hyperplasia is very common too and may be life-threatening.

In case of the **Dyke-Davidoff-Masson syndrome** one of the cerebral hemispheres is partially or completely atrophic as a result of an intrauterine or perinatal carotid artery infarction. Since the cerebral atrophy in SWS occurs also during infancy, changes similar to those in the Dyke-Davidoff-Masson syndrome, including cerebral hemiatrophy with ipsilateral calvarial diploic space enlargement, may be seen.

Though it is possible that the birthmark and the atrophy in the cerebral cortex is present without any symptoms, there will develop among most of the infants convulsive seizures during their first year of life. There is a great likelihood of intellectual impairment if the seizures start before the age of 2 and are resistant to treatment.

Diagnosis: The MRI is better than the CT scan in detecting the malformations affecting the central nervous system in case of SWS. The diagnosis is often obvious on the plain skull x-ray film. The MRI examination can find abnormal venous drainages and pial contrast enhancements associated with SWS. The angiomatous malformations confirm the diagnosis even in case of very young children. The MRI could show a cerebral volume reduction and an ipsilateral choroid plexus enlargement as well. By using intravenous contrast material, in addition, one may demonstrate the curvilinear posterior contrast enhancement in case of an ocular choroidal angiomatosis.

On the other hand, the CT scan is better than MRI in detecting the characteristic double-lined gyriform pattern of calcifications being parallel with cerebral convolutions, which are named railroad track sign by radiologists. The calcifications can usually not be detected if the patient is younger than 1 year or even for several years.

A-scan ultrasonography and B-scan ultrasonography can be useful diagnostic aids diagnosing diffuse choroidal hemangiomas. The B-scan ultrasonography characteristically shows a solid echogenic mass, while the A-scan ultrasonography shows a high internal reflectivity.

The fluorescein angiography can be a useful complementary examination.

Treatment: There being no specific treatment, the therapy of Sturge-Weber syndrome is symptomatic. Laser treatment may be used to lighten or to remove the birthmark. Anticonvulsant medications can be used to control seizures. Monitoring for glaucoma, and surgery may be performed in more serious cases. Physical and educational therapy is often prescribed. Medical therapy can be used to lower the intraocular pressure, etc.

Prevention: To detect and eliminate the pathogen microorganisms before pregnancy would be effective.

RFR method: detects and may eliminate the viral and mycoplasmal infection befor pregnancy. Convulsive seizures might develop mostly during the infant's first year of life, the immediate detection of the viral and mycoplasmal infection is thus necessary.

The most frequent resonances of SWS are as follows: 288-293, 344-345, 371-387, 393-394, 403, 408-411, 416-420, 440-451, 468, 544-545 kHz

10.8. Rett Syndrome

Rett syndrome is a neurodevelopmental disorder of childhood, characterized by a normal early development followed by the loss of the purposeful use of the hands, distinctive hand movements, slowed brain growth and head growth, gait abnormalities, seizures and mental retardation. It affects girls almost exclusively. Affected girls are said to be placid infants, low toned and with subtly slowing development. The course of the illness is characterized by a period of developmental stagnation followed by a period of regression. Males with this disorder show also symptoms, ranging from severe congenital encephalopathy, dystonia apraxia and retardation to psychiatric illnesses with mild mental retardation.
Rett syndrome is primarily a neurologic disorder but affects many other organ systems as well. Before the discovery of it, incidents were mistaken for many other neurologic disorders.
This syndrome is caused by many a factor, including *genetic errors, defective immunologic responses* and *infections.*
Rett syndrome is a *genetic* disorder hindering the normal neurodevelopmental process rather than being a progressive process. The gene for this syndrome is located on the X chromosome. This gene is encoding the Methyl-CpG-binding Protein-2 (MECP2). Females with 1 mutated MECP2 gene are more likely to survive as one X chromosome is activated randomly in each cell. The symptoms and the severity of the illness may depend on the percentage of activated defective genes and the type of mutation. Rett syndrome is the first discovered human disease being caused by defects in a protein, regulating the gene expression by the interaction with methylated deoxyribonucleic acid (DNA). The disease can also be inherited from phenotypically normal mothers who have a germline mutation in the gene encoding Methyl-CpG-binding Protein-2, MECP2.
An atypical form of Rett syndrome, characterized by infantile spasms or early onset epilepsy, can also be caused by a mutation to the gene encoding cyclin-dependent kinase-like 5.
Is it an Inherited disorder? Though Rett syndrome may be a genetic disorder – resulting from a faulty gene or genes – less than one percent of the recorded cases are inherited or passed from one generation to the next.
The role of *infections* in the etiology of this disease can be significant. Patients with Rett syndrome are frequently infected by *Mycoplasma fermentans*, *Epstein-Barr Virus* and *Human T-cell Lymphotropic Viruses.* Every one of these infections can cause an *abnormal immune response* concerning vaccination, according to my conception. According to Fiumara's theory the pertussis vaccination may cause Rett syndrome. A girl infant 2 months of age, developed an acute encephalopathy with a destructive brain damage 12 hours after acellular pertussis vaccination. Following this vaccination, the peripheral lymphocyte subset analysis revealed the existence of T lymphocytes double positive for CD4 and CD8 markers. This pattern was normalized over the following 3 months. Months later, the girl showed a Rett syndrome phenotype. DNA screening of the MECP2 gene could not be revealed either in the child or in her parents. This previously unreported association emphasizes the notion that Rett syndrome phenotypes can result from different (either genetic or environmental) causes.
There is also an other hypothetic causal association such as between the measles-mumps-rubella (MMR) vaccine and autism. I think, that a normal immune response does not cause a Rett syndrome following vaccination, but this syndrome may develop in case of a damaged immune response, f.i. damaged due to *mycoplasmal* or *HTLV infection.*
The symptoms of the clinical stages of this illness are, as follows:
Stage I – Developmental arrest affecting typically infants aged 6-18 months of age. Parents may report gross motor development delay, disinterest to playing and the loss of eye contact.

Hypotonia may be noted. Hand wringing, a hallmark of the disease, appears typically. Infants may be reported as placid and calm, if compared to healthy infants. Early symptoms are often vague and nonspecific.
Stage II – Rapid deterioration or regression typically concerning children aged 1-4 years.
Deterioration may be rapid. Parents can sometimes report specific dates, following which their child was no longer healthy. Regarding other cases, deterioration may be slow in onset.
This stage can last from weeks to months and may be characterized by reports of autistic-like behavior, such as a loss of social interaction and communicative skills, loss of oral language, and loss of purposeful finger and hand use. Parents may note stereotypic hand movements during wakefulness, which are usually moderate consisting of hand wringing, clapping, washing, or hand-to-mouth movements. Parents also may report episodes of breathing irregularities, such as hyperventilation or breath holding. Patients with Rett syndrome also may have seizures and vacant spells that look like seizures. Other problems that may be noted are sleep disorders, intermittent strabismus and irritability.
Stage III – Pseudostationary, typically affecting children aged 2-10 years.
There may occur some improvement in behavior, hand use, and communication skills. The patient's eye contact will get better so that they are able to make their intent known by whatever communicative skills that remained. Despite these improvements, there will exist a continued mental impairment, the hand stereotypias will continue. Increasing rigidity, bruxism, involuntary tongue movements, motor dysfunctions and seizures can be still experienced. Episodes of hyperventilation or breath holding may continue. Even though the child has a good appetite, weight gain is poor. Feeding may become more difficult, and almost all individuals with Rett syndrome show some degree of oral motor dysfunction.
Stage IV – Late motor deterioration, typically concerning children older than 10 years.
There does not occur any deterioration of cognitive skills, communication skills, or hand skills; nethertheless, motor problems such as hypertonia, dystonia and Parkinson symptoms: f.i., bradykinesia, rigidity, retropulsion may increase. Some patients do not walk any more. Their seizure frequencies may be reduced.
Other Symptoms of Rett syndrome (that are similar to autism) include: screaming fits, panic attacks, inconsolable crying, avoidance of eye contact, lack of social/emotional reciprocity, general lack of interest, markedly impaired use of nonverbal behaviors to regulate social interaction, loss of speech.
Diagnosis: By examining the mutations in the MECP2 gene identifying a wide spectrum of clinical phenotypes (including girls with classic Rett syndrome, girls with its variant forms, girls with autism, healthy females (carriers), males with severe infantile encephalopathies, males with classic Rett syndrome, and males with X-linked neurologic problems).
Treatment: Symptomatically, as no specific medication is available to treat persons with this syndrome.
RFR method and prevention: detects and eliminates the pathogen present before vaccination so that the removal of pathogen microorganisms will stop the development of a damaged immune response.
In case of Rett syndrome, I found *Mycoplasma fermentans*, *Epstein-Barr Virus* and *HTLV-1* frequencies.
The most frequent resonances are: 370-376, 442-451, 493-495 kHz

10.9. The Tuberous Sclerosis or Bourneville's Disease

The clinical triad of convulsive seizures, mental deficiencies and adenoma sebaceum manifests this disease. The earliest lesions are foliate hypopigmented spots over the trunk, which are seen most clearly under ultraviolet light. The mental deficiency may be relatively stationary or progressive. The seizures are usually generalized, but may be focal,

too. Retinal tumors, optical atrophy and cataracts, syndactylism, spina bifida and other visible malformations may also be present. The latter are fine, wartlike lesions predominantly in a butterfly distribution on the cheeks and the forehead. The lesions of the skin are pathologically fibromas or like adenoma. Some other lesions are rather vascular looking like telangiectasia. The brain lesions consist of areas of malformed cortex with extensive astrogliosis and a peculiar mixture of glioblasts and monster nerve cells. Calcification may or may not be present. In Bourneville's original case, the death of the patient was due to a rhabdomyoma of the heart. Most cases of this benign tumor of he heart muscles are associated with tuberous sclerosis. This disease is combined with tuberous malformations of the kidney, the liver, the adrenal glands, the pancreas and the tuberous malformations of other organs.
Tuberous Sclerosis has is very complex etiology, family history is frequently unhelpful. There are many primery and secondary infective pathogen microorganisms to be found in case of this disease. The immune state is frequently damaged, there is often a severe immunodeficiency present.
Diagnosis: by roentgenograms, EEG, ECG, CT, combined with mental examinations.
Treatment: symptomatically. The prognosis is depressing.
RFR method: detects and may eliminate the pathogen microorganisms!
The most frequent resonances are: 222, 238, 340-354, 368-372, 396-397, 409-410, 437, 442-451, 525, 557-558 kHz

10.10. Attention Deficit Disorder (ADD) and Attention Deficit Hyperactivity Disorder (ADHD, AD/HD)

The attention deficit disorder (ADD) and the attention deficit hyperactivity disorder (ADHD) can be diagnosed with increasing frequency in children and adults as well. Many of these patients were previously said to be hyperactive or slightly brain damaged so that their terminology changes from a minimal brain damage to a minimal brain dysfunction.
The terms ADHD and the Hyperkinetic Disorder terminology are commonly used, they are considered to be developmental disorders affecting about 5% of the world's population. The disorders affect typically children, and are characterized by a persistent pattern of inattention and/or hyperactivity, as well as by forgetfulness, poor impulse control or impulsivity and distractibility. ADHD is currently considered to be a persistent and chronic condition for which there exists a poor medical cure or none at all. In the past decade the disease is generally diagnosed in childhood and increasingly in adulthood, too.
In case of prenatally occuring rubella viral or measles infections the pathogens are carried by the circulatory system. Hence it follows that virtually every physiological, neurological or vital function may be weakened. The connatal rubella infections and measles infections result from the transplacental transmission of the viruses from the infected mother to the fetus, and may be associated with a minimal brain damage and a slight brain dysfunction, because these viruses can infiltrate and cause damage in different tissues thus f.i. in the tissues of the central nervous system, too. In case of a postviral chronic encephalitis a moderate perivenous demyelination and a minimal damage of the brain function can develop. These damages may be of directly viral origin or can be caused by a postinfectious allergic process.
Regarding children suffering from Lyme borreliosis attention deficit disorder (ADD) and attention deficit hyperactivity disorder (ADHD) provoked by Borrelia B. sensu lato bacteria are often present.
Diagnosis: can be established by their symptoms, nethertheless EEG and other special neurologic and psychologic examinations and tests are needed, as well.
Treatment: there is in case of prenatal viral infections no specific therapy. The generally used drugs applied in case of ADD (such as methylphenidate (Ritalin), and pemoline

(Cylert), have the same Class 2 classification as cocaine and morphine, can cause negative side effects and have certain risk factors.
In case of ADD and ADHD caused by lyme borreliosis, antibiotic therapy will heal the patients.
RFR examining frequencies and the possibly effective frequencies will find: 332-334, 350-352, 364-468, 371-372, 387-390, 402, 440, 425, 442-444, 450-454, 473, 492, 498, 516, 522-528 kHz and 377-387.5 kHz in case of Lyme disease.

10.11. PANDAS or TIC Disorders

Several inflammatory disorders have been associated with the preceding streptococcal infections, including an acute rheumatic fever (ARF), post-streptococcal reactive arthritis, erythema nodosum, post-streptococcal glomerulonephritis and cutaneous vasculitis. The spectrum of the poststreptococcal diseases is expanded with adding Pediatric Autoimmune Neuropsychiatric Disorders Associated with Streptococcal infection (PANDAS). PANDAS is a recently described subgroup of childhood neuropsychiatric disorders, arising great public and physician interest in their pathophysiology, diagnosis and management as well. The hypothesis, that neuropsychiatric disorders, such as the Tourette's syndrome and the Obsessive-Compulsive Disorder (OCD) are related to a preceding streptococcal infection, is certainly controversial. The pediatric rheumatologists should be told to utilize their expertises concerning childhood autoimmune diseases to evaluate the use of immunomodulatory therapies in case of these children.
Clinical criteria proposed to establish the diagnosis of PANDAS include: the presence of OCD, tic disorder; of onset of the disorder in childhood; an abrupt onset of symptoms; exacerbations of symptoms temporally related to a preceding streptococcal infection; and the presence of neurological abnormalities as well. Additionally, the clinical features, the way of treatment and prevention of PANDAS has to be reviewed.
Obsessive-Compulsive Disorder (OCD), tic disorders, Tourette's syndrome (TS) and Sydenham's chorea (SC) are neurobiologic disorders due to basal ganglia abnormalities. Both tics and OCD can be the result of the damage of the basal ganglia, while obsessive-compulsive symptoms occur frequently in case of patients suffering from SC. *Group A beta-hemolytic streptococcus* (GABHS), the etiologic agent, responsible for acute rheumatic fever and SC, was recently proposed to trigger tic disorders and the OCD in genetically predisposed children.
OCD is observed in about one to two percent of school-aged children. Obsessions and compulsions may change in course of time in content and severity with no clear pattern of progression. The disorder is chronic and disabling, but the course may wax and wane with exacerbations and remissions.
TS has a lifetime prevalence of 1: 1000, and are characterized by the childhood onset of chronic motor and vocal tics. TS has a variable course, with symptoms often waxing and waning. Severity varies from a social nuisance to a severely debilitating condition. Neuroleptics are the medications of choice in order to reduce the severity of tics, though the symptoms are rarely entirely eliminated by this therapy. TS is an in an autosomal dominant manner inherited illness, though a mixed genetic way as well as an environmental influence may also play a role in its pathogenesis.
Pathophysiologically, the manifestations of an acute rheumatic fever is likely the result of molecular mimicry, thus the antibodies to GABHS cross-react with the self-proteins inducing an inflammatory autoimmune response. Specifically, antibodies against GABHS cross-react with the neuronal cells producing inflammation particularly within the basal ganglia, resulting in movement disturbances observed in SC.
Sydenham's chorea can occur after a few weeks or even nine months after being infected with GABHS. Clinical manifestations include motor symptoms, emotional lability and behavioral changes.

The biologic evidence that PANDAS is an autoimmune-mediated process is compelling, but not conclusive, though there exists an identified potential B cell marker, the magnetic resonance imaging of the brain demonstrated basal ganglia changes showing an inflammation, moreover, certain immunomodulatory therapies proved to be benefitial concerning some patients. Antibiotic prophylaxis, though being effective in case of ARF, remains questionable in case of PANDAS.
A B lymphocyte cell marker D8/17 was identified as a predictor of ARF and SC, diseases recognized to have genetic susceptibility.
Diagnosis: by clinical symptoms, by finding a D8/17 marker, by the measuring of streptococcus titer, by CT, MRI.
Treatment: by administering antibiotics, corticosteroids, and immunosuppressive drugs or,
alternatively, an immunomodulatory treatment either administering intravenous immunoglobulin (IVIG) or using a plasma exchange may hasten the recovery.
RFR method: detects and may eliminate group A beta-hemolytic streptococcus. This pathogen may prove to be resistant to antibiotics. Group A beta-hemolytic streptococcus can bring about a secondary process in case of infections caused by other pathogen microorganisms and cause an autoimmune process.
The resonant frequencies of the Group A beta-hemolytic streptococcus species are, as follows: 345, 353, 368-375, 386-390, 402, 450-452, 536-544 kHz
Eliminate the other pathogen, too, by using its resonant frequencies. In case of GABHS infections the RFR method decreases the possibility of the development of the PANDAS syndrome, but is not effective concerning the damage of the basal ganglia.
Penicillin prophylaxis is useful in preventing recurrent rheumatic fevers, and its effect can be increased using RFR method.

10.12. Stress Syndrome

Chronic intensive stress effects cause stress syndromes, including severe differential responses such as neurosis, restlessness, uncomfortable as well as enjoyable feelings, easy fatigue, pain, loss of sexual desire, anxiety, difficulty in concentrating, increased irritability, muscle tension, decrease in general immune response, disturbed sleep and other nervous system dysfunctions or/and somatization disorders.
The stress is the biggest challenge of our new age. Although a certain amount of stresses is normal in our life, prolonged bouts of stress can lead to exhaustions and minor illnesses and even to more serious illnesses. The stress produces several, different physiologic effects, it can affect the release of ACTH, causing increased concentration of steroids in the adrenal venous blood, thus decreasing the general immune response. In such a way there can develop several different chronic infections and hypertension as well.
Diagnosis: by psychological examinations.
Treatment: is extremely difficult and complex.
RFR method: detects and may eliminate the pathogen microorganisms causing chronic, secondary infections.

10.13. Anorexia Nervosa and Bulimia Nervosa

Anorexia nervosa is a disorder characterized by a distorted body image, an extremely fearing obesity, refusal to maintain a minimally normal body weight, and, in case of women, by the absence of menstrual periods. Having lost the desire to eat, anorexia is a prominent symptom characterized by various intestinal and extra intestinal disorders. It must be clearly differentiated from satiety and from specific food intolerance.

Bulimia nervosa is a disorder characterized by repeated episodes of binge eating followed by purging (i.e. self-induced vomiting), by taking laxatives, diuretics, rigorous dieting as well as by excessive exercising for the sake of counteracting the effects of bingeing.
Anorexia nervosa and bulimia nervosa are psychiatric disorders and may have very complicated syndromes.
A significant loss of weight is often followed by the stop of menstrual periods in case of women. Both men and women may lose interest in sex. People suffering from anorexia often have a low heart rate, a low blood pressure, a low body temperature, edema, as well as fine, soft hair or, in contrast, excessive body hair and facial hair. Depression is common in both illnesses, but this symptom may be caused by a secondary, bacterial, intestinal infection f.i. by *Shigella*, *E. coli*, *Salmonella*, *Pseudomonas* and *Proteus*, as well as may even develop a regional enteritis.
The hormonal changes resulting from anorexia nervosa and bulimia nervosa include markedly reduced levels of estrogen and thyroid hormones, as well as increased levels of cortisol. Immune mechanisms are also involved in the developing of depression in case of being in these states. Abnormalities regarding 5-hydroxytryptamine, noradrenaline and the corticotropic-releasing hormone function may also be present. Some data show, that the levels of cholecystokinin (which is a brain-gut peptide having an established role in the modulation of both the food intake and the gastric emptying) in the serum of patients suffering from eating disorders differ from its level regarding the control persons.
The disorders, concerning the heart and the changes of electrolyte contents (such as sodium, potassium, calcium and chloride) of the body fluids, are the most dangerous. The patients may become dehydrated and be liable to faint. There may even develop metabolic acidosis. General deficiency in vitamins can also be present. A variety of psychological, sociological, and familial influences are thougth to have influence on the development of anorexia nervosa.
The mortality, associated with anorexia nervosa, is high, the 6-12% of patients eventually succumb to the disorder. Death is usually caused by starvation or by suicide.
Diagnosis: by psychiatric examinations, laboratory examinations and by bacterium culture from the feces using antibiotic resistance tests. By examination of total protein, liver enzymes, creatin kinase, beta human chorionic gonadotropin hormon levels as well as of thyroid hormol levels in the serum. Electroencehalogram, CT scan, and MRI often show enlarged ventricles in the brain and an increased ventricle-brain ratio in case of patients suffering from anorexia compared to the age-matched and sex-matched controls.
Differential diagnosis: by distinguishing it f.i. from panhypopituitarism, malabsorption, regional enteritis caused by other factors, diabetes mellitus, carcinomatosis, Crohn's disease, malabsorption syndrome, miliary tuberculosis, etc.
The treatment: of this condition is very difficult. In extreme cases parenteral or nasogastric feedings are necessary.
RFR method: can help in case of patients having some antibiotic-resistant bacteria found in the bowels.
The resonance frequencies of pathogenic clostridium species in the bowel, see Chapter 6.17.
The resonant frequencies of Shigella flexneri causing most frequently secondary infection in the bowel, are: 393-395 kHz
Friendly bacterial cultures, containing f.i. acidophylus and bifidobacteria have to be administered.

10.14. Autism

Autism is a disorder in which a young child cannot develop any normal social relationship, behaves in a compulsive and ritualistic way, and usually fails to develop a normal

intelligence. Childhood brain disorders suggest an inherited genetic defect. Nethertheless numerous viral and bacterial pollutants are able to pass into the unborn child through the placenta. The cause of autism may be congenital, but also connatal, associated with *Rubella, Measles virus, Cytomegalovirus*, or other *herpes viruses*. These pathogens can often be found in the mother before/under pregnancy befor/and after delivery as well as in her child suffering from autism.

Protection against this disease of the mother by RFR method is only possible before pregnancy. The bacteria and the viruses must be eliminated at least a few months prior to pregnancy. Do not use RFR method if a woman is already pregnant.

The RFR method of the child can be in some cases effective and useful.

10.15. Migraine Syndrome in General

Migraine headache is a recurring, throbbing, intense pain usually affecting one side of the head but sometimes affects both sides. The pain begins suddenly and may be preceded or accompanied by visual, neurological, or gastrointestinal symptoms. The attacks, instead of beginning in childhood and recurring in the usual fashion every few weeks or months with dimishing frequency in middle and late adult years, may begin in adult life or even in middle age or can suddenly increase in frequency during the menopause or when hypertension and a vascular disease had developed. Migrain is a symptom, the mechanism of its pain is not clear yet. The aura of a migraine may consist of neurological symptoms, such as dizziness, tinnitus, irritability, hemiparesis, restlessness, nausea, scotomas, photophobia, or visual scintillations (eg, bright zigzag lines) or consists of seeing jagged, shimmering or flashing lights; and loss of appetite occur in about 20 percent of the people suffering from migraine. The headache previously described as a classic migraine, is now known as migraine with aura, while that described as a common migraine is now termed migraine without aura. Migraines without aura are the most common, accounting for more than 80% of all of them. The pathophysiology of migraine headaches is not clearly understood. However, growing evidence supports the role of serotonin and dopamine receptors in the brain. Some of the symptoms of migraine headaches, such as nausea (80%), vomiting (50%), drowsiness or confusion, yawning, irritability, hypotension and hyperactivity can be associated with dopamine receptor activation. Dopamine receptor hypersensitivity has been by dopamine agonists such as apomorphine, bromocriptine and pergolide experimentally proven. Dopamine antagonists, such as metoclopramide, haloperidol and prochlorperazine are effective in case of migraine headaches.

The target of pharmacotherapy is to prevent attacks or alter migraine attacks reducing its severity and its duration. The preventive therapy encompasses these symptoms, decreases the frequency of the attacks and improves the effect of the treatment.

The patients suffering from migraine syndrome may have herpes viral or some other viral infections.

Diagnosis: by blood pressure measuring, by CT, EEG, RTG, contrast examination, ophthalmologic examination, as well as by other laboratory examinations, f.i. searching for parasitism.

Differential diagnosis: by distinguishing it from brain tumor, cluster headaches, tension headaches, Guillain-Barré syndrome, ophthalmologic migrain, trigeminal neuralgia, glossopharyngeal neuralgia, Bell's palsy, angioma, aneurysm, subdural hematoma as well as from parasitic infections, f.i. from Strongyloides infection.

Treatment: symptomatically. (There is no specific therapy for migraine syndrome.) By administering Sumatriptan, frovatriptan, eletriptan, naratriptan. Selective agonists for serotonin 5-HT1 receptors can be of help administering it into the cranial arteries. This drug can suppress the inflammation associated with migraine headaches. Ergotamine tartrate, having alpha-adrenerg and serotonin antagonist effects causes the constriction of the peripheral and the cranial blood vessels. Dihydroergotamine in form of nasal spray is

more effective if given at once at the beginning of the migraine attack. Isometheptene dichloralphenazone acetaminophen has sympathomimetic properties dilatating cranial and cerebral arterioles, causing the reduction of those stimuli, which lead to vascular headaches. Barbiturates are used in combination with aspirin and acetaminophen to relief pain and to induce sleep. Caffeine is also used to increase GI absorption. Antidopaminergic drugs such as prochlorperazine and droperidol having anticholinergic effects, depress the reticular activating system, and thus relieve of nausea and vomiting.

RFR method: detects the pathologic resonances!

The most frequent resonance frequencies of migrain are: 290-293, 378-385, 394-402, 406-410, 416-421, 447-453 kHz

RFR method has no effect on the acute migrain attack.

10.16. Headache and Migraine Syndrome

Headaches count among the most common medical problems. Headache is a symptom of different diseases and of pathological conditions. Up to 90% of the general population experience headaches at some time of their lives. At the given time, up to 10% of the general population seeks medical treatment in order to get rid off their disabling headaches. **Migraine is a neurological syndrome** and most frequently an inherited disorder, characterized by altered bodily perceptions, headache, nausea, vomiting, dizziness, tinnitus, scotoma, photophobia, or visual scintillations, (increased sensitivity to bright light), and hyperacusis (increased sensitivity to sound), olfactory, or other sensory experiences that are a sign that the migraine will soon occur.The typical migraine headache is recurrent, unilateral and pulsating, lasting from 4 to 72 hours. Migraine sometimes has dramatic features of transient neurological deficits.

Migraine is much more common in women than men, runs in families, and is usually a disorder of young, primarily healthy women. Some 5 to 60 minutes before the headache begins (i.e. the period named aura and prodrome), symptoms of depression, irritability, restlessness, nausea, or loss of appetite occur in about 20 percent of the people. Similar percentages of people lose vision in a specific area (called a blind spot or scotoma) or see jagged, shimmering, or flashing light. Some people experience tingling sensations or rarely, weakness in an arm or leg. Usually these symptoms disappear shortly before the headache begins, but sometimes they merge in it.

Migraine with aura and migraine without aura is far more „common" than migraine with aura. Migraine headaches commonly begin early in the morning but may occur at any time of the day or night. Nocturnal headaches awakening the patient from sleep, characteristically occur in cluster headaches but have also been reported with migraine. However, a patient's recent onsets of nocturnal headaches should by thorough neurological and ophthalmological evaluation and with appropriate imaging studies, be distiguished from brain tumor and glaucoma.

During severe attacks, headaches are lateralized (as to 60-70% of the patients) which headaches are followed by bi-frontal or global headaches (in up to 30% of the patients). There are occasionally other loci of the headache. Pain is often associated with nausea, photophobia, phonophobia and occasional vomiting. Headache is usually gradual at the onset and getting worse and worse persists typically for from 4 to 72 hours, gradually seizing totally. The headache is usually dull, deep and steadies from mild to moderate and becomes throbbing and pulsating if severe. A rapid moving of the head, sneezing, straining, constant motion or physical exertion lead many migrain sufferers to lie down in a dark, quiet room because of the worsening of their migraine headaches. The phenomenon of prodrome should be separated from the aura. Prodromes can last from hours to days, and are usually associated with the patient's changes in mood, appetite and fluid retention. Although autonomic features characteristically occur in cluster headaches, they can also occur in case of 10-20% of migraine patients. These symptoms may include nasal

stuffiness, rhinorrhea, tearing, change in skin color and temperature, and changes of the pupil. Migraine headaches may frequently occur for long periods but then they will disappear for many weeks, months or even years. How do the causes of headaches differ: muscle tension, status migraine, basilar artery migraine, carotidynia, cluster headache, high blood pressure, eye problems (iritis, glaucoma), cholepathy, abnormal bile secretion, gallbladder disease, sinus problems, brain tumor, brain infection (abscess, meningitis), accumulation of blood around brain (subdural hematoma, subarachnoid hemorrhage), accumulation of edema, raised intracranial pressure, distension, traction, and dilatation of the intracranial or extracranial arteries, inflammation, posttraumatic nervous instability, other diseases (viruses, such as arbovirus, arena virus, herpes simplex virus,) cancer, and bacteria (such as syphilis, tuberculosis), cryptococcosis, sarcoidosis, and worms (f.i.strongyloidosis and other brain parasites).

Another type of migraine is a throbbing or pulsating headache that is often one sided (unilateral), associated with nausea; vomiting; sensitivity to light, sound, and smells; sleep disruption and depression. Attacks are often recurrent and tend to become less severe as the sufferer is getting older.

Some women experience migraine headaches just prior to or during their menstruation. These headaches named *menstrual migraines*, may be related to hormonal changes and do not often occur during pregnancy. Other women get migraines for the first time during pregnancy or after their menopause.

In case of migraine serotonin, dopamine, and other vasoactive neuropeptides stimulate an inflammatory cascade affecting the endothelial cells mobilizing endothelin 1-3, the mast cells and platelets. This inflammatory process causes vasodilation and in other places vasoconstriction, functional hypoxy and a perivascular oedemic reaction. Serotonin receptor 5-HT is believed to be the most important receptor playing a role in its development. Neurogenically induced plasma extravasation can play a role in the expression of pain in migraine, though the presence of other stimulators may also be required. The pain process does need not only the activation of nociceptors of pain-producing intracranial structures but also the reduction in the normal functioning of endogenous pain control pathways that gate the pain.

The causes of migraine

The cause of migraine is unknown. The condition may result from a series of reactions in the central nervous system caused by changes in the body or in the environment. The disorder is often familiar, suggesting that migraine sufferers may inherit their sensitivity to triggers producing inflammation in the blood vessels and the nerves around the brain, causing their pain.

According to me, migraine is caused by an infection of the nervous system. Such various causes of headaches or migraine can be viral infections (f.i. *Herpes simplex virus*, *West Nile virus, ECHO, Adeno and Coxsackie viruses*) bacterial infections (f.i. *Borrelia Burgdorferi sensu lato, Treponema pallidum, Nanobacteria, Klebsiella, Staphylococcus and Streptococcus group*). These microorganisms can play an important role also in chronic recidive migraine processes. Macroparasites (f.i. *Strongyloides*) and/or other macro- and microparasites can cause migrain attacks in tropical countries. These chronic infections change the normal biochemical functional pathways and cause irregulate neurological functions and sensitivity to triggers.

Triggers (Trigger is a stimulus initiating a process or a reaction). Identified migraine triggers are the following:

Alcohol (e.g. red wine)

Environmental factors (e.g. weather, altitude and time zone changes)

Foods containing caffeine (e.g. coffee and chocolate), monosodium glutamate (MSG; found in Chinese food), and nitrates (e.g. processed foods and hot dogs)

Glare

Hormonal changes in women
Hunger
Lack of sleep
Medications (over-the-counter and prescription)
Perfume
Stress

The International Headache Society classifies migraine headache: into migraine without aura, migraine with aura, basilar type migraine, familial and sporadic hemiplegic migraine, abdominal migraine, sinusitis origine migraine, acephalgic migraine, medication-, food- and/or alcohol migraine, migraine caused by changes in barometric pressure and menstrual migraine.

Basilar artery migraine, is caused by the disturbance of the basilar artery in the brainstem. Symptoms include severe headache, vertigo, double vision, slurred speech, and poor muscle coordination. This type occurs primarily among young people.

Carotidynia, also called lower-half headache or facial migraine, produces deep, dull, aching, and sometimes piercing pain in the jaw and the neck. There is usually tenderness to be felt as well as swelling over the carotid artery in the neck. Episodes can occur several times weekly and last from a few minutes to hours. This type occurs more commonly in case of older people.

Headache-free migraine is characterized by the presence of aura without headache. This occurs in case of patients with a history of migraine with aura.

Ophthalmoplegic migraine begins with a headache felt in the eye and is accompanied by vomiting. As the headache progresses, the eyelid droops (ptosis) and the nerves responsible for eye movements become paralyzed. Ptosis may persist for days or weeks.

Cholestasis migraine has prodrome syndromes: gastrointestinal symptoms, fatigue, malaise, arthralgias, myalgias, photophobia, nausea and vomiting.

Status migraine is a rare type involving intense pain that usually lasts longer than 72 hours, so that the patient may require hospitalization.

Diagnosis: by tests to rule out physical diseases, by evaluation of psychologic factors and personality, by blood analysis, MRI, CT, PET-SCAN, spinal tap, eye examination, x-ray examination of sinuses, kidney tests, migraine drugs effectivity examination, bile secretion, and blood pressure measuring.

Treatment: The conventional treatment focuses onto three areas i.e. trigger avoidance, symptomatic control and preventive drugs. Drugs are not able to eliminate the cause of the migrain.

RFR method: detects and eliminates the pathogens, which are the origine cause of the migraine. The RFR method is able to find the origine of the migrain and has not only an effect on the symptoms.

The most frequent resonances of migrain caused by viruses are: 276-293, 294-302, 307-321, 466-475 kHz

The most frequent resonances of migrain caused by bacteria are: 324-325, 358-362, 375-376, 378-387, 398-402, 415-421, 442-451, 560-568 kHz

The most frequent resonances of migrain caused by macroparasites are: 338-339, 368-369, 374, 383, 432, 481-482 kHz

Migraine sufferers seem to be at risk for thrombotic and hemorrhagic strokes as well as for transient ischemic attacks. RFR method decreases the occurance of ischemic attacks, of thrombotic and hemorrhagic strokes, as this method can eliminate Nanobacteria, Herpes viruses and other damaging microbiological factors of the blood vessels.

The most frequent resonances of migraine associated with gallbladder diseases are: 320-329, 333-339, 345-356, 365-370, 382-394, 408-417 kHz

Administer probiotica f.i. Bifidophilus and Acidophilus bacteria in order to regenerate the natural intestinal flora!

10.17. The Development of Autoimmune Brain Processes

The first factor in the development of an autoimmune brain process or brain disease may be a Borrelia attack. This process is caused by false and ineffective defense of the host against the chronic persisting microbas (see Chapter 6.20.3.). The process may be similar to that of some other intracellular persisting pathogens, f.i. chlamydial infections, mycoplasmal infections. In certain instances of Lyme borreliosis, free antibodies can neither be found in the serum nor in the liquor or in other fluids, as they are absorbed in the tissues bound in immunecomplexes. Some other co-pathogenic microorganisms may also influence the process. Moreover, some worm antigens may provoke the development of allergic processes in addition to the existing autoimmune disease.

Viruses can also often play a role in these pathological processes. A bout with the measles may be brief, the recovery occuring in about one week, but it may be prolonged, resulting in serious brain damage or death. In certain rare cases, chronic sclerosing neural damage, a serious complication of measles, may occur years later, resulting in a slow brain damage. The chronic sclerosing panencephalitis, a progressive and usually sclerotic disorder, is a rare late complication of measles that can appear several or many years later, producing mental deterioration, muscle problems and seizures. The role of a mycoplasmal co-infection in case of this disorder has to be examined (see Chapter 23.9.).

10.18. Chronic Brain Diseases and Mycoplasma

Mycoplasma species can be found in chronic brain diseases and nerve diseases f.i. Alzheimer's disease, Multiple Sclerosis as well as Parkinson's disease, with an occurrence rate of 40-60%. The occurrence rate of Chlamydia speciesis in these diseases is less. One of the causative factors of these chronic neurological diseases may be a mycoplasmal or/and a chlamydial infection. Although we do not know exactly what are the causes of autoimmune and of degenerative diseases, there is an increasing evidence that in case of many patients suffering from such chronic infections, particularly some certain bacteria, together with genetic predisposition and immune dysfunction play an important role in the pathogenesis. Certain *Mycoplasma species* and *Chlamydia species* can either activate or suppress the host's immune system, and they may use these activities to evade the host's immune response. For example, some Mycoplasma species can inhibit or stimulate the proliferation of certain lymphocyte subsets, induce B-cell differentiation and trigger the secretion of cytokines. Mycoplasmas are able to secret soluble factors stimulating the proliferation or inhibiting the growth and the differentiation of immune competent cells. The persistence of mycoplasmal infections has many similarities with the persistence of chlamydial infections. Both of these bacteria are obligatory intracellular parasites being dependent on intermediary metabolites and biosynthetic precursors of the host's cells, and they are supposed to cause some of their pathogenic effects during their intracellular persistence phase. Mycoplasma species release from the cells without cell lysis, can carry host cell surface antigens with them, eventually causing host responses against its own infected tissues. In such a way mycoplasmal infections are supposed to play an important role in the developing of autoimmune diseases, but this role is not well understood and cleared up yet

Mycoplasmas often offer resistance to antibiotic therapy. Antibiotics are usually not able to eliminate Mycoplasmas in the central nervous system.

Examinations used by diagnosing mycoplasmal infections: see Chapter 6.18.

Treatment: Doxycyclin, Ciprofloxacin, Azitromycin for a very long time (months).

RFR method: detects and may eliminate Mycoplasma or/and Chlamydia!

Mycoplasma fermentans has the most frequent incidence in such chronic infections.

The resonant frequencies of Mycoplasma fermentans are: 311-312, 328-329, 352-353, 360-361, 371, 403-404, 442, 448-450, 494 kHz
The other frequencies of Mycoplasma: see Chapter 6.18.
The resonant frequencies of Chlamydia are: 318-319, 430, 440-443, 470-471, 480-485, 562-568 kHz

10.19. Schizophrenia

Schizophrenia is a serious mental disorder characterized by the loss of contact with reality, hallucinations, delusions, abnormal thinking, confusion in doing one's work and a disrupted social functioning. Although the specific cause of schizophrenia is not known, the disorder clearly has a biologic basis. The reason which makes a person vulnerable to schizophrenia is not known, but it may include a genetic predisposition; f.i. problems that occurred before, during, or after birth; or a viral infection of the brain. The severity and the type of symptoms can vary significantly among people suffering from schizophrenia. Its symptoms can be sorted into three major groups: delusions and hallucinations; thought disorders and bizarre behavior; and deficient or negative symptoms.
The types of Schizophrenia: there are paranoid, disorganized, catatonic, undifferentiated and deficit types.
Diagnosis: can be established by symptoms, by differential diagnosis. The use of EEG, CT and MRI can be helpful as well.
Treatment: by administering Fluphenazine, Haloperidol, Perphenazine, Thioridazine, Clozapine, etc. and by psychotherapy.
RFR method: can only be effective if the patient has pathological resonances.
Test the patient for these frequencies of molds: 295, 308, 352-354, 376, 400, 464, 484, 504, 524, 562, 576 kHz and for the frequencies of Mycobacterium phlei: 409-411 kHz
If any of these frequencies are found, RFR method may be helpful.

10.20. Curious Neurological and Psychiatric Symptoms in Borreliosis

Patients infected by *Borrelia Burgdorferi sensu lato species* can show curious neurological and psychiatric symptoms, too. In some cases their symptoms are as if they were suffering from ALS, Alzheimer's disease, Lupus erythematosus, CFS, fibromyalgia syndrome, Bell's Palsy, Multiple Sclerosis, RA, morphea, autism, panic disorders, schizophrenia, etc.
Borrelia B.s.l. infection may induce neurodegeneration and produce the symptoms of ALS, induce demyelinization and show the symptoms of MS, can cause cerebritis in the brain and produce the symptoms of LE. Lyme borreliosis is often present as a co-infection among patients suffering chronic diseases mentioned above.
Children suffering from neurological symptoms of Lyme disease may have headache, blurry vision, double vision, confusion, irritability, sensory hyperacusis, fever, stiff neck, tiredness, sleeping disorders, attention-deficit/hyperactivity disorders (ADHD), executive dysfunctions, panic disorders with attacks that last often longer than 30 minutes, attention deficits, etc. In this chronic process they may be encephalopathic having lingering headaches, personality changes and memoryloss, too.
In case of a non-treated Lyme disease, besides other multiorganic symptoms (affecting mostly the joints, the nerves and the muscles all over the body) fatigue, multiple cognitive impairments, depression, sleep disorders, pain, migraine like headache are most often present. Anxiety, irritability and panic reactions are very usual.
Presenile dementia, cognitive and verbal disturbances can also occur.

All the symptoms mentioned above have a relapsing and remitting character, manifesting usually monthly or even less often, but if the disease progresses, the symptoms remit more often and can even persist.
The patient may be referred to a psychiatrist because of the unexplained medical symptoms requiring psychiatric assessment and therapy.
The brain SPECT scans can show a pattern of global non specific hypoperfusion in a heterogeneous distribution through the white matter. Abnormal MRI findings are often experienced in the early and late stage of Lyme disease. MRI findings of patients suffering from neurologic Lyme disease may demonstrate punctated white matter lesions on T2-weighted images, similar to those seen in case of other demyelinating and inflammatory disorders, such as Multiple Sclerosis, SLE, lues and other cerebral vasculitides. Patients presenting acute signs of aseptic meningitis will have pleocytosis and an elevated protein level in the spinal fluid. The neurocognitive testing usually show abnormalities.
Neuroborreliosis is an infection within the brain; but even those infections in the body, that do not pass the blood-brain barrier may affect the brain indirectly via immune effects (f.i. *mycoplasmas*, *HTLV* etc.), and thus worsen and widen and modify the symptoms.
An inappropriate diagnosis and treatment as to these infections for several years will result in the escalation of symptoms.
The differential diagnosis of the late stage borreliosis may include every medical or psychiatric conditions, even MS, LE, etc. Though co-occurring symptoms may be caused by diseases being simultaneuosly present, in case of borreliosis, a single disease process can have multiple manifestations. The bigger the number of co-occuring symptoms, the greater the likelihood that it is a systemic disease process with multiple manifestations. Multiple psychiatric syndromes, especially those with neurological and cognitive symptoms alone, without any signs of other affected organs, suggest a CNS pathological process, while significant psychiatric and somatic comorbidity suggest a systemic disease, f.i. borreliosis, especially in endemic areas all over the world. In case of the disseminated phase of lyme borreliosis these psychiatric and somatic symptoms are present, in a relapsing and remitting way often followed by a progressive deterioration.
Diagnosis: established by the carefully interrogated history of the patient, living ore having been in an endemic area, symptomatically, by serology: immunoblotting, ELISA, (repeatedly, if borreliosis is suspected), by PCR. By examining cerebrospinal fluid. (Solely a negative antibody analysis of the cerebrospinal fluid solely never excludes the possibility of a persisting neurologic infection caused by Borrelia B.s.l.) By SPECT or PET, neuropsychologic testings etc. The presence of global cerebral hypoperfusion deficits on SPECT together with characteristic neuropsychiatric features should dramatically raise the suspicion of an existing lyme encephalopathy among patients inhabiting endemic areas, regardless of the patient's memory concerning tick bites. Seronegative diseases can occur, cerebrospinal fluid testing can often be normal, thus lyme encephalopathy can often be an exclusive diagnosis.
Treatment: by administering psychotropics together with antimicrobials! A prolonged antibiotic therapy should be given. The choice of the type of the drug depends on the psychiatric symptoms. (Psychiatric drugs, often helpful among lyme borreliosis patients are f.i.: delta-sleep-promoting agents, modafinil, anticonvulsants, atypicals, lithium, serotonin-norepinephrine reuptake inhibitors, etc.)
RFR method: can eliminate all the pathogen components of the clinical symptoms. Should only be used after/together with psychiatric and antibiotic treatments. The therapy will last a very long time, borrelia can change its plasmids, remeasuring is advised, repeated treatments can often be needed.
The most frequent resonances are: 302, 378-387 kHz
As to the other frequencies see Chapter 6.20. and other special Chapters.

10.21. Infectious Emotional Crisis

In case of patients suffering from Lyme disease and having mycoplasmal infections too, neurological problems may develop presenting different neurological and psychiatric symptoms such as headache, nausea, vomiting, diarrhea, high fever, chills, muscle and joint aches, disorientation, depression, schizoid personality, suicid mentality, emotional crisis and anorexia. Since these patients besides having Lyme disease have a high prevalence of infection caused by Mycoplasma fermentans (and a lesser degree of prevalence of infection caused by other Mycoplasma species), they usually show evidence of multiple infections. In these states the diagnosis should be done as early as possible. Drowsiness, confusion, stupor, or, rarely, a comatous status and palsy of various degrees may occur, but as a rule, the permanent derangement of consciousness tends to be relatively mild. The stiffness of the neck and the spine, when bending forward attest meningeal irritation, which at first may be slight and unnoticed.

The Lyme disease is caused by spirochetes *Borrelia Burgdorferi sensu lato (B. afzelii, B. garinii, B. sensu stricto, B. valeisiana, B. lusitaniae*) usually transmitted by ticks or deerflies (see Chapter 6.20.3.). If the patient has a mycoplasmal infection, too, the disease develops fulminantly and usually spreads into the nervous system. In case of these combined infections the most frequent infective agent is the Mycoplasma fermentans. These combined infections are rapidly developing and become to be very severe due to the poor immune response suppressed by Mycoplasma species (see Chapter 6.18.1.).

Diagnosis: by PCR, ELISA, immunoblot methods by the examination of blood, urine and/or liquor.

Treatment: by administering effective antibiotics for a long time, by psychopharmacons and psychotherapy.

RFR method: Detect and eliminate at first the mycoplasma and then the Borrelia!

The most frequent resonant frequencies of B. afzelii are: 382-387.5 kHz

The most frequent resonant frequencies of B. burgdorferi are: 378-382 kHz

The most frequent resonant frequencies of B. garinii are: 380-382.5 kHz

The resonant frequencies of the vegetative (CWD) forms are: 301-302, 327, 341, 415, 420-422, 430, 512, 547-548, 556, 565 kHz

The other frequencies of the Borrelia species see in Chapter 6.20.

The resonant frequencies of antibiotic-resistant Borrelia species are: 382-390 kHz

The most frequent resonances of Mycoplasma fermentans are: 442-451, 491-495 kHz

The frequencies of other Mycoplasma species see in Chapter 6.18.

The RFR method may worsen the symptoms of the disease (Herxheimer reaction) for one or two weeks, the patient may need higher doses of psychopharmacons.

10.22. Multiple Sclerosis

In case of Multiple Sclerosis (MS) the nerves of the brain and the spinal cord lose patches of myelin. The term Multiple Sclerosis comes from the multiple areas of scarring that represent many patches of demyelination in the nervous system. A large important group of neurological disorders are termed demyelinating diseases as they share the common pathologic feature of foci of degeneration involving the myelin sheath of nerve fibers. These foci vary in size, shape, distribution and in the rate of development in these illnesses. The axons often suffer damage as well, but the destruction of myelin is considered to be the first of the alterations. There is no clear cause ascertained for this group of diseases as yet, but a wide variety of etiologic theories are proposed, including those, based on either infective, or metabolic, allergic, immunological, vascular causes, or on an earlier viral infection. The classification is based on the combination of clinical and pathological factors. Of course, it is possible, that the process of demyelination may have several different reasons and may be a common manifestation of different diseases. The

postinfectious encephalomyelitis syndrome is often referred to as parainfectious encephalomyelitis its onset being prior to, in association with, or following the rash of measles or the exanthemas of other viral infections.
The cause of Multiple Sclerosis is a multiple infection of the brain with *Borrelia Burgdorferi s.l.*, *nanobacteria* and/or other pathogens. An other theory supposes that a virus or some unknown antigen somehow triggers an autoimmune process, usually early in life. More over, also the *Mycoplasma fermentans* may play a role in the pathogenesis of this disease (see Chapter 23.9). In case of this disease the body, for some reason, produces antibodies against its own myelin and with these antibodies provoke an inflammation damaging the myelin sheath. The demyelination in the nerve pathways bringing signals to the muscles causes problems in the movement (motor symptoms), while the demyelination in those carrying sensations to the brain causes disturbances in sensation (sensory symptoms). This autoimmune process may play a role in the development of Multiple Sclerosis. The lesions on the brain may vary in diameter from less than 1 mm to several centimeters; affecting principally the white matter of the brain and the spinal cord but do not extend beyond the root entry zone of the brainstem and the spinal cord. They frequently encroach on the cerebral gray matter, but do not destroy the nerve cells (neurons). The lesions have a predilection for elongated structures where the myelin abuts the pial veins, hence the frequent involvement of the spinal cord and the optic nerves and the chiasm. The lesions are regularly to be seen in the paraventricular areas of the brain in relation to the veins in the walls of the lateral ventricles.
The histological appearance depends on the age of the lesion. The relatively recently developed lesions show a predominantly perivenous distribution of the demyelination with but a few axis cylinders, certain degenerations of oligodendroglia, neuroglial reaction, and perivascular infiltrations with mononuclear cells aswell. Later on, a large number of microglial phagocytes are infiltrating the lesion, and the astrocytes in and around the lesion will be increased in number and size. A long-standing lesion, on the other hand, will show a thickly-matted, relatively acellular, fibroglial tissue, with a few perivascular macrophages. In these lesions intact axis cylinders may still be discovered, while many may be destroyed, leading to a descending or an ascending degeneration of the long-fiber tracts.
Allmost all of the human neurodegenerative processes, such as the Alzheimer's disease and the MS, can be associated with chronic pathological calcification. No human tissue is resistant to nanobacteria. They easily cross the blood-brain barrier and cause brain calcification disorders and brain sand.
The symptoms of Multiple Sclerosis: About 50 percent of patients suffering from MS have optic neuritis as their initial symptom, the syndrome of which has a rapid onset, lasting over a period of several days. A partial or a total loss of vision concerning one eye may occur, sometimes associated with pain in the eye moving. The most frequent, classic features of this illness include an impaired vision, nystagmus, dysarthria, intention tremor, ataxia, impaired perception of position and impaired vibratory senses, bladder dysfunction, weakness of a limb, paraplegia and alteration in the emotional responses.
The most common *motor symptoms* are weakness, clumsiness, imbalance in walking, tremors, double vision, bowel problems, stiffness and tiredness.
Sensory symptoms, as well as numbness, tingling, dysesthesias, visual disturbances, sexual disorders, dizziness, and vertigo can also be present.
The acute Multiple Sclerosis runs an acute or sub-acute course leading to death in weeks or months. Alternatively, an acute course may develop rapidly, then remit partially or completely, to be followed by characteristic relapses. In some acute cases, the onset is marked by headache, vomiting, delirium and by succession of symptoms indicating a severe involvement of the brainstem or the brain, the optic nerves and the spinal cord.

Patients suffering from MS complain of the symptoms appearing in one leg though, actually both are involved. A patient complaining of weakness, ataxia, or lack of sensitivity in one of the lower extremities evidently suffer from the bilateral corticospinal tract disease manifested by Babinski's sign in both lower extremities. Symptoms of bladder dysfunction, including hesitancy, urgency, frequency and incontinence occur commonly with spinal cord involvements. In case of males, these symptoms are often associated with impotence, which the patient usually does not mention unless questioned.
Neuromyelitis optica, also referred to as **Devic's disease**, represents a combination of bilateral optic neuritis and transverse myelitis. This rare disorder resembles MS in several ways, but the target of the autoimmune process is a protein of the nervous system named aquaporin 4.
The Diagnosis can be based on the symptoms, such as possible motorous and sensory abnormalities; and on the examination of the cerebrospinal fluid. The MRI is the most sensitive diagnostic imaging technique, possibly revealing areas of the brain having lost myelin. EEG, CT can also be helpful.
Differential diagnosis: happens by distinguishing it from disseminated encephalomyelitis, meningovascular syphilis, encephalitis of some other origin and from Schilder's disease (named encephalitis periaxialis diffusa).
Treatment: a newest way of treatment using beta interferon and gamma globulin products reduces the frequency of relapses. ACTH and corticosteroids can also help. A variety of dosage regimens are employed. Prednisolon taken orally or Methylprednisolon given for a short period help to banish the acute symptoms.
The measuring with **RFR** can be used in order to find the pathological resonances.
One can see, that Multiple Sclerosis is a very complex multietiologic disease, its frequencies being: 288-294, 299-302, 311-329, 337-340, 348-353, 365, 372-373, 377-384, 389, 391, 396, 401-402, 420, 429-431, 439-453, 456, 458, 474-475, 480-482, 486, 488, 493, 518, 534-536, 540-544, 560-566 kHz
Species of *Borrelia Burgdorferi s.l., Nanobacteria, Chlamydia, Epstein-Barr Virus, Cytomegalovirus, VZV, Measles, Smallpox, Rubella viruses and Poliovirus* (got from an earlier infection or by vaccination), as well as several bacteria, such as *Shigella, Cryptococcus, Treponema pallidum* are listed participiants which may all have a role in the development of MS.
It is crucial to stop the myelin damage before it becomes irreversible.
RFR method: is used to eliminate the pathological agents and to stop the autoimmune cascade.

10.22.1. Schilder's Disease

Schilder's disease,, named also **Diffuse Myelinoclastic Sclerosis (DMS),** is a very rare neurodegenerative disease presenting clinically pseudotumoral demyelinating lesions. It is difficult to diagnose it. This disease is considered to be one of the borderline forms of Multiple Sclerosis. Some other diseases of this borderline group are Neuromyelitis Optica (NMO), Balo concentric sclerosis and Marburg Multiple Sclerosis.
Schilder's disease usually begins in childhood, affecting children between 5 and 14 years.
The cause of Schilder disease, i.e. an existing combined infection, is similar to that of Multiple Sclerosis. There is a latency period between the initial febrile illness and the subacute onset of Schilder's immune-autoimmune disease. Some of the cases may be examples of an acute disseminated encephalomyelitis. Others have a fulminant course without any clear distinction between the prodrome and the onset of the disease process, these are thought to be Schilder disease cases.
In case of DMS the combined infection occur caused by *Mycoplasma species, HTLV*, and other viruses, such as *Coxsackie virus* and *ECHO virus*; bacteria, such as *Borrelia species, Nanobacteria* and/or *Chlamydia species*. Certain mycoplasmal and borrelial antigens are

adsorbed to the myelin sheath and the said antigens will generate an immune-autoimmune process, causing demyelinative degeneration. A widespread demyelination of both cerebral hemispheres with varying degrees of axonal injury can be found when examining autopsy cases. The lesions, usually found to be somewhat asymmetrical, have sharp margins, and spare the immediate subcortical rim of white matter. Similar demyelinative changes are often found in the brainstem and cerebellum. Axonal injuries in form of Wallerian degeneration may occur throughout the nervous system, most especially in the spinal cord. The pathology of severe cases resembles that of Multiple Sclerosis much more than that of acute or chronic disseminated encephalomyelitis.
Some authors mention cases with characteristic bilateral lesions associated with additional multiple small plaques. The appearance of these plaques resembles the usual Multiple Sclerosis plaques more typically than that of typical acute disseminated encephalomyelitis lesions. These cases tend to develop in adolescence or adulthood rather than childhood, and their clinical course is more variable than those of the acute severe and more widely disseminated cases.
Symptoms are similar to those of Multiple Sclerosis and may include dementia, aphasia, seizures, personality changes, poor attention, tremors, balance instability, incontinence, muscle weakness, headache, vomiting, vision and speech impairments.
Diagnosis: by MRI: One or two roughly symmetrical large plaques, greater than 2 cm in diameter. There are no abnormalities of the peripheral nervous system and no other lesions present. By pathological analysis, consistent with subacute or chronic myelinoclastic diffuse sclerosis. A diagnosis based on MRI and PETscan studies without pathological confirmation increases the likelihood of etiological heterogeneity, including such entities as leukodystrophy, tumor, SSPE, various types of vasculitis, lymphangiomatosis, collagen vascular diseases, nutritional diseases, meningoencephalitis, and other entities in addition to Multiple Sclerosis and acute disseminated encephalomyelitis.
Its **Prognosis** depends upon the definition of the illness. High-dose corticosteroids are sometimes given, though there are no carefully designed trials made concerning their efficiency. Moreover, this treatment does not eliminate the infectious pathogen microorganisms.
Treatment: symptomatically, f.i. by administering corticosteroids, beta-interferon and immunosuppressive drugs. By supportive physiotherapy, occupational and nutritional therapy. By administering specific antibiotic therapies in order to eliminate *Borrelia* and *Mycoplasma* species. The neuroborreliosis treatment with antibiotics can cause Jarisch-Herxheimer reaction.
RFR method: detects and can eliminate all pathogen microorganisms.
The most frequent resonances are: 317-319, 324, 370-374, 378-387, 410, 427-429, 440, 442-451, 458, 474, 480-485, 530-536, 560-568, 584 kHz

10.23. Progressive Multifocal Leukoencephalopathy

The Progressive Multifocal Leukoencephalopathy (PML) is a rare, usually fatal illness characterized by multifocal progressive damages or inflammations of the white matter of the brain and the spinal cord caused by the *JC virus* (a kind of poliomavirus). The infection affects almost exclusively patients with seriously impaired function of T-lymphocytes and with severe **immune deficiencies, those** suffering f.i. from leukemia, lymphoma, carcinomatosis, AIDS or from autoimmune chronic diseases; or transplant patients being on **immunosuppressive medication**, or those receiving certain kinds of **chemotherapy or corticosteroid therapy.** Men are more frequently affected than women. People can be JC virus-infected with no apparent symptoms.
Polyoma viral infections are usually acquired early in childhood. The majority of these infections are subclinical and the virus may remain latently present in the kidney or other organs, too. Just like in case of herpes viruses, the JC virus remains latent until something

provokes its reactivation. Progressive multifocal leukoencephalopathy is developing usually in case of the activation of the polyomavirus latently present in the brain or other tissues since their infection got in childhood. This disease develops only if the immunoreactivity of the patient get decreased by *HTLVs* or by *Mycoplasma fermentans* and/or by other immunodepressive agents. The disease is consistently associated with the disorders of cell-mediated immunity together or without any deficits in the humoral antibody response. PML is a demyelinating disease caused maybe by autoimmune processes, in which the myelin sheath covering the axons of the nerve cells will get gradually destroyed, impairing the transmission of nerve impulses. PML affects the white matter mostly composed of axons from the outermost parts of the brain cortex.

The symptoms of Progressive Multifocal Leukoencephalopathy usually appear either gradually or suddenly years (from 2-10 years) after getting infected. If they once start, they usually worsen rapidly and vary depending upon which part of the brain is infected. The neurological signs and symptoms point on a diffuse, asymmetric involvement of the cerebral hemispheres. Weakness, hemiplegia, hemianopsia, aphasia or dysarthria and organic mental changes are frequent, a complete or incomplete transverse myelitis may develop as well. Headache and convulsive seizures do but rarely occur, though electroencephalographic abnormalities consisting of diffuse or focal abnormalities are often present. The content of the cerebrospinal fluid is in most cases normal. The progressive loss of intellectual ability and the development of dementia or of paralysis are characteristic for this illness. One to six months after the appearance of the symptoms the patients usually die.

Diagnosis: symptomatically, by noninvasive techniques, such as EEG, CT, MRI and PET scan, as well as by PCR-testing for JC viruses, by electronmicroscopy, by examining the biopsy tissues, etc.

Treatment: no treatment has as yet proved to be effective.

RFR method: can detect and eliminate the causative pathological microorganisms.

The most frequent resonances of JC virus are: 292-297, 318-319, 331-336, 341-346, 364-366, 372, 378-383, 397, 401-403, 418-419, 436, 457, 473-478, 530, 552, 569, 576-581 kHz

The resonant frequencies of Mycoplasma fermentans are: 442-451, 493-495 kHz

As to the resonant frequencies of HTLVs, see their special Chapter.

10.24. Parkinson's Disease (Paralysis Agitans, Extrapyramidal Syndrome of Abnormal Posture, Involuntary Movement)

The Parkinson's disease is a slowly progressing, degenerative disorder of the nervous system with several distinguishing characteristics, f.i. tremor when at rest, a sluggish initiation of movements and muscle rigidity.

The basal ganglia process the signals and transmit the messages to the deep-lying thalamus, which takes the processed information to the cerebral cortex. All of these signals are transmitted by a chemical neurotransmitters as electrical impulses along nerve pathways and between nerves. The main neurotransmitter of the basal ganglia is the dopamine. In case of Parkinson's disease, the nerve cells in the basal ganglia degenerate, causing a lower production of dopamine and restricted connections with other nerve cells and muscles. The cause of the degeneration of nerve cells and of the dopamine loss is not yet known. The only regularly observed changes are in the aggregates of melanin-containing nerve cells of the brainstem (substancia nigra, locus caeruleus) showing a loss of nerve cells, a reactive gliosis and distinctive eosinophilic intracytoplasmic inclusions (Lewy body). Parkinson's disease may be caused by a combined infection of the brain by

viral and fungal pathogens The RFR resonance frequencies of the viral component can be found between 570-580 kHz, proving thus, most likely, to be an unknown virus.
It is possible that chronic nocardial infections can play a role in the development of the Parkinson's disease. However, about half of the people with nocardiosis, mostly the elderly ones, have no any neurological disease befor. At the present nocardiosis is considered to be a complication resulting from the chronic brain infection in patients suffering from Parkinson's disease. In case of chronic nocardiosis, the patients develop brain abscesses, experience severe headaches and altered sensations, or weakness as well. Which of the parts of the body will weaken is depending on the locus of the brain abscess. The toxin produced by nocardia can cause brain damage.
Symptoms include tremors and rigidity. Emotional stress or fatigue may increase the smooth, rhythmic tremors. The initiating of a movement is particularly difficult for the patient, and the developing muscle stiffness will impair the movement as well.
Diagnosis of Parkinson's disease is done depending on the symptoms. An MRI examination is useful.
Differential diagnosis: by distinguishing it from cerebral vascular diseases, cerebral hypoxia, pyramidal tract deficit, neoplasmas, metallic poisoning, cerebral toxic poisoning caused by chemical substances, etc.
Treatment: by administering Levadopa, Carbidodopa, Bromocriptine, Pergolide, Selegiline, and anticholenergic drugs f.i. Benztropine, Trihexilphenidyl and Amantadine. These therapies can only weaken the symptoms but can not heal the patient.
RFR measuring can be used to find the pathological resonance(s).
The nocardiosis (see Chapter 6.14.5.) is frequently detected together with Parkinson's disease. This infection caused by the fungus-like bacterium Nocardia asteroides, usually starts in the lungs and can spread to the skin or the brain, causing a chronic disease process. Chronically ill people and those receiving drugs suppressing their immune system have an increased risk of getting ill with nocardiosis. An advanced age is also a risk factor of nocardiosis in the brain. These bacteria produce specific neurotoxins which damage the neuron cells in the thalamus, the hypothalamus and the hypocampus.
The species of *Borrelia Burgdorferi s.l., Chlamydia, Nocardia, Epstein-Barr Virus*, other *Herpes viruses* such as *Cytomegalovirus* and *Chicken pox* viruses, *Measles and Rubella* viruses, *Smallpox* and a hitherto unidentified virus are all possible pathogens concerning this brain process.
It is crucial to stop the dopamine cell degeneration before it becomes irreversible.
RFR method is used in order to eliminate the pathological agents and to stop the autoimmune cascade. See the end of this Chapter.
Frequently-found pathogenic microorganisms in brain diseases are the following:
Nocardia asteroides, its frequencies being: 350-357, 360-371, 454 kHz
Borrelia Burgdorferi s.l. and their plasmids, their frequencies being: 302-305, 312-322, 329, 353-357, 372-388, 401-409, 429, 442, 452-454, 508-511, 548, 556 kHz
Chlamydia, its frequencies being: 316-319, 374-386, 429, 444, 480-482 kHz
Shigella, its frequencies being: 313, 318, 369, 388-396, 403-410, 423-425, 499 kHz
Cytomegalovirus, its frequencies being: 305, 349, 406-412, 534 kHz
Epstein-Barr Virus, its frequencies being: 337-339, 342-347, 372-382, 397-398, 422, 450, 518 kHz
Herpes Simplex Virus-1, its frequencies being: 290-294, 344-346 kHz
Herpes Simplex Virus-2, its frequencies being: 352-365, 413, 425 kHz
Herpes Zoster Virus, its frequencies being: 410, 416-421, 467 kHz
Rubella virus (German Measles), its frequencies being: 372, 402, 450 kHz
Morbilli virus (Measles), its frequencies being: 381, 403-407, 492 kHz
The other frequencies of the Measles virus are: 327, 350, 364-373, 381-387, 390, 402-407, 450-456, 478, 492, 522-536, 564-567 kHz

The other frequencies of Herpes Simplex Virus-1 and 2 are: 366-377, 383-385, 413, 434 kHz
Molds, their frequencies being: 303, 318, 372, 380-381, 392, 401, 443, 450-453, 477, 482 kHz
Aspergillus, its frequencies being: 346, 356, 380-387, 394, 466, 504 kHz
Cryptococcus, its frequencies being: 296, 298, 304-305, 313-319, 402-405, 438, 446, 480-488, 524, 542 kHz
This list is not yet complete, as there exist other subspecies with differing frequencies.
It seems that the fungal components can be molds, f.i. in case of Alzheimer's disease the *Aspergillus* species, in case of Multiple Sclerosis the *Cryptococcus*, and in case of Parkinson's disease the *Nocardia*.
Borrelia infections and *Chlamydia* infections can initiate autoimmune processes in the brain.

10.25. Amyotrophic Lateral Sclerosis (ALS, Lou Gehrig's Disease)

The ALS (also named Lou Gehrig's disease), is a rapidly progressing, invariably fatal neurological disease attacking those nerve cells (neurons) responsible for the controll of the voluntary muscles. The disease results in the irreversible degeneration and the death of the motor neurons. The course of the illness is characterized by the progressive degeneration of the upper motor neurons (UMNs) as well as the lower motor neurons (LMNs). The UMN symptoms include hyperreflexia and spasticity, caused by the degeneration of the lateral corticospinal tracts in the spinal cord. The damage of the LMNs results in weakness, atrophy, and fasciculations. All these being the direct consequences of muscle denervation. In case of intense weakness of the respiratory muscles ALS can prove to be rapidly fatal. Aspiration pneumonia and medical complications of immobility goes along with morbidity.
The onset of the disease is usually focal and asymmetric, the bulbar motor neurons are often involved, resulting in bulbar weakness (progressive bulbar palsy). The anterior horn cells of the spinal cord can also be affected, resulting in the weakness of the limbs and in spinal muscular atrophy. The UMN involvement of the bulbar muscles results in pseudobulbar palsy causing spastic dysarthria, dysphagia and emotional incontinence. The UMN involvement of the spinal cord tracts causes a spastic weakness of the limbs, (i.e. primary lateral sclerosis). Later on, the spreading to other motor areas produces the classic combination of the upper and lower motor neuron dysfunction, named ALS. Patients suffering from familial ALS show a mutation in the genes producing the superoxide dismutase-1 (SOD1) enzyme. This enzyme acts as an antioxidant. Glutamate toxicity, mitochondrial dysfunction, and autoimmunity may all be cofactors of the pathogenesis of this disease.
The course of this fatal disease is progressive, the median time of its survival is 3-5 years. The illness usually occurs among patients aged between 40-60 years.
The ALS belongs to a group of disorders known as infectious motor neuron diseases, in which co-infections of *Mycoplasma fermentans, Borrelia Burgdorferi s.l.* and viruses (including *HHV6, ECHO viruses and Coxsackie viruses*) are present at the same time.
The earliest **symptoms** can include twitching, cramps, muscle stiffness, the weakness of the muscles of one arm or one leg, or that of the oropharyngx, where in latter case, slurred and nasal speech, difficulty in chewing or swallowing can be observed.
The damages of the LMNs can cause muscle atrophy, widespread, active fasciculations. The signs of the destruction of the UMNs (corticospinal tract) can include spasticity, hyperactive tendon reflexes and Babinski sign. Muscle cramps are common. Dysarthria, exaggeration of motor expressions and emotional lability (i.e. pseudobulbar affect) may

occur if the corticobulbar projections to the brainstem are affected. The patients may have inappropriately active tendon reflexes and weak, wasted, twitching muscles. (No loss of anal sphincter tone does occur, the ocular, the cardiac and the smooth muscles are not involved.) Hypoxia and cardiac arrhythmias are the most common causes of the death of patients suffering from ALS.
Some other infectious diseases, such as the Lyme disease and infections caused by HIV and HTLV can, in some cases, cause ALS-like symptoms. Some neurological disorders, such as the Multiple Sclerosis, the Post-polio syndrome, the Multifocal motor neuropathy and the Spinal muscular atrophy also can mimic certain parts of the disease.
The **Diagnosis:** is based on the neurological signs and symptoms, the abnormal reflexes, electrodiagnostic testings (lower motor neuron signs), MRI, PET scan, electromyography.
Differencial diagnosis: by differentiating it from other diseases f.i. from the Multifocal motor neuropathy with conduction block (by detecting anti-GM1 antibodies), by determining the level of the Vitamin B-12 and the folate of the serum, by HIV testing and by Lyme serology. Brainstem lesions including syrinx, mass, stroke, and demyelination forms or other degenerative diseases, cranial nerve palsies, cervical myelopathy, cord tumor, hereditary spastic paraparesis, transverse myelopathy, HIV-related myelopathy, radiculopathy, plexopathy, neuropathy, compressive myelopathy, Chronic inflammatory demyelinating polyneuropathy (CIDP), toxic or metabolic neuropathies, myopathies f.i. inclusion body myositis, polymyositis, myasthenia gravis, Guillain-Barré syndrome, etc.
Treatment: there is no specific ALS therapy. By administering neurotransmitter-inhibitors such as Riluzole (Rilutek), antiplasticity agents, f.i. Baclofen (Lioresal) and antibiotics for pneumonia or urosépsis. Inicially, the empiric use of a relatively broad-spectrum antibiotic may be effective against supposed pathogens, to be followed it by appropriate microbial cultures and specimens for laboratory evaluation. These medications may include f.i. cephalosporins, fluoroquinolones, vancomycin, penicillins and aminoglycosides.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances usually found in case of ALS are the following: 294, 304, 310-312, 317, 320, 329-331, 335, 340, 346, 349, 351-354, 364, 369-372, 375, 378-386, 392-393, 397-398, 403-404, 421, 431-433, 441-444, 447-452, 458, 465, 472-474, 480, 494-496, 502-505, 518-520, 532-535, 544, 556 kHz
The RFR method may increase the symptoms and cause depression for a short time.
The autoimmune responses, provoked by Mycoplasma fermentans and by Borrelia B.s.l. is thought to be the possible causes of motor neuron degeneration in case of ALS.
Physical therapies and special equipments can enhance the patient's independence and his feeling of safety throughout the course of this illness.

10.26. Prion Diseases (Mad Cow Disease, Creutzfeldt-Jakob Disease)

Prion diseases are a related group of rare, fatal brain diseases affecting animals and human beings as well. These transmissible spongiform encephalopathies (TSE) include the bovine spongiform encephalopathy (BSE, or „mad cow" disease) concering cattle; the Creutzfeldt-Jakob disease (CJD) regarding people, scrapie among sheep and goats; and the chronic wasting disease (CWD) of deer and elk. The name prion diseases is associated with, a misfolded cellular prion protein, while their other name points at the spongelike nature of the damaged brain tissue.
Unfortunately, there are several human diseases that fall into this category of spongiform encephalopathies, including kuru, Creutzfeldt-Jakob disease, and the Gerstmann-Sträussler-Scheinker syndrome. All these diseases resulting eventually in a spongiform encephalopathy, are possibly transmitted by a suspected infectious agent named prion which means proteinaceous infectious particle. The diseases are characterized by certain

misshapen protein molecules that appear in the brain tissue. The normal forms of these prion protein molecules reside on the surface of many types of cells, including the brain cells, their proper functions are not yet known by scientists. On the other hand, scientists believe that the abnormal prion proteins, clumping together and accumulating in the brain tissue, are the probable cause of the brain damage occuring in TSE diseases. The neuropathology of CJD was classically described as the combination of spongiform changes, neuronal loss and astrogliosis. If a spongiform change is present in the human brain tissue, a large number of small holes or vacuoles can be seen in histological samples of the affected tissue. The frequency and the morphology of these holes are characteristic of this disease and not present in normal brain tissues. The disease known as kuru (another human spongiform encephalopathy) has long been recognized and was first diagnosed among certain tribes in New Guinea practicing cannibalistic rituals – the women often smeared the brain tissue of the tribe's member who had died onto their own body in order to receive the life „power" of the victim – women of this tribe, who participated in these rituals acquired the disease later on. With the decrease of cannibalism, decreased kuru as well.

Kuru is a progressive and fatal neurological disorder which occurs exclusively among the natives of the Highland of New Guinea. The symptoms include abnormal involuntary movements resembling myoclonus, athetosis, or chorea, and convergent strabismus. Dementia develops in the late phase of the diseases. Death sets in within 4 to 24 months, usually owing to decubitus, ulcers and bronchopneumonia.

The human mad cow disease, i.e. the Creutzfeldt-Jakob disease is a subacute spongiform encephalopathy, which is a progressive, inevitably fatal infection, producing muscle spasms and the progressive loss of mental functions. The slowly developing brain damage increases and the loss of intellectual ability (i.e. dementia) becomes apparent.

The symptoms include apathy, irritability, forgetfulness and confusion. Some patients tire easily, and though being sleepy, are unable to fall asleep, or suffer from other sleep disorder. Then the symptoms accelerate, usually much more rapidly than in case of Alzheimer's disease, until the person get profoundly demented. Trembling, clumsiness, and peliculiar body movements also may develop. Vision may become blurry or dim. About 90 percent of patients die within 1 year. In the early stages of disease, patients may have a failing memory, behavioral changes, lack of coordination and visual disturbances as well. As the illness progresses, the mental deterioration becomes more pronounced and involuntary movements, blindness, weakness of the extremities and coma may occur.

According to an earlier perception, prion diseases are being caused by slow viruses. Diseases caused by slow viruses are characterized by a long asymptomatic period of even months and years, lasting from being infected upto the appearance of clinical illness. Some slow viruses provoke a conventional inflammatory response in the time being clinically dormant; others are able to reside in the cells for many a period without causing any detectable cytopathic changes. In case of slow viral infections, the role of immunity is largely unknown. In case of certain slow viral infections there are elevated levels of circulating antibodies present, while in other cases, there can no detectable immune response be found. Viruses have been recovered in the nervous system of patients having a subacute sclerosing panencephalitis, a progressive multifocal leukoencephalopathy and a progressive rubella-like encephalitis as well.

Prevention: by the eliminating of infected human and animal tissues as well.

Diagnosis: by special laboratory tests and by brain biopsy.

Differential diagnosis: by distinguishing it from other Prion diseases and from Alzheimer's disease.

Treatment: symptomatically. CJD can't be cured, and its progress can't be slowed.

RFR method: detects and may eliminate the viruses!

The most frequent resonances are: 363-373, 381-387, 390, 402-407, 448-458, 492 kHz

10.27. Cerebral Palsy

Cerebral palsy is characterized by poor muscle control, spasticity, paralysis as well as other neurological deficiencies resulting from brain injuries occuring during pregnancy, birth, after birth, or before the child is 3 years of age. The areas of the motor controll center of the brain, which control the muscle movements are particularly vulnerable to injury in case of premature and very young infants. Many different types of injury can cause cerebral palsy, but its cause is most often unknown. With RFR measuring one can often find viral infections among these patients, which infections could be connected with the cerebral palsy state, but one can not prove it for sure. Such viral infections are often caused by *herpes viruses* (see Chapter 5.2.4.), *cytomegalovirus* (see Chapter 5.2.4.4.), *measles* (see Chapter 5.1.2.4.) and by some other known viruses as well, moreover I detected frequencies of some as yet non-identified pathogens.

Cerebral palsy, being a group of non progressing motor system disorders of various degree, can be distinguished from other, more or less similar, neurological diseases by their progressing unsteadiness in standing and walking as well as by the impaired coordination of some other motor functions. The pathology of these illnesses is characterized by the degeneration of the cells of cerebellum and/or its related fiber systems. Some forms of progressive ataxia, associated with pathologic changes predominantly in the cerebellum, are hereditary though there are some rare sporadic forms, too f.i. the illnesses caused by subacute spinocerebellar degeneration and by olivopontocerebellar degeneration.

The symptoms regarding the patients differ, depending on the degree of the brain damage varying from barely noticeable clumsiness to severe spasticity causing contorted arms and legs or even confining the child to use a wheelchair. The cerebral palsy is classified as: spastic, choreoathetoid, ataxic and mixed.

Diagnosis: by laboratory tests, electrical studies of the muscles, by muscle biopsy, by CT and MRI.

Differential diagnosis: alcoholic cerebral ataxia, Wernicke-Korsakoff syndrome, Pierre Marie hereditary ataxia, other motor system diseases, Verding-Hoffman Syndrome, Oppenheim syndrome, neural muscular atrophy, restricted dyskinesis, Refsum's disease and Behr's syndrome.

Treatment: symptomatically, there being no specific or effective treatment for this illness.

RFR method: detects and may eliminate the viruses.

The most frequent resonances are: 290-295, 305-307, 316-321, 332, 372, 382-384, 397, 401-402, 410-412, 442-451, 454-457, 460-463, 475-480, 504, 555-558 kHz

10.28. Meningitis

Meningitis is an inflammation of the piarachnoid, the fluids which fills the space enclosed by it as well as the ventricles of the brain. Since the subarachnoid space is continously around the brain, the spinal cord as well as the optic nerves, an infective agent gaining entry to every part of it may extend immediately to all part of it, even to its most remote recesses. In such a way a meningitis is always cerebrospinal.

There are more ways of causing a pyogenic infection regarding the cranial contents: either by a hematogeneous spread or by an extension from the surface structures (f.i. from the ears, the paranasal sinuses, the osteomyelitic foci in the skull, or connatal sinus tracts), as well as from penetrating cranial injuries.

The causative agents of meningitis are mostly bacteria or viruses and infrequently fungi or other factors, too.

10.28.1. The Most Frequent Bacteria Causing Bacterial Meningitis

Ninety percent of the cases of bacterial meningitis are caused by any of the species of *Haemophilus influenzae* (see Chapter 6.7.2.2. and 10.28.3.), *Neisseria meningitidis* (see Chapter 6.3.2.), *Streptococcus pneumoniae* (see Chapter 6.16.2.1.1.), *Staphylococcus aureus* (see Chapter 6.15.3.1.), Escherichia coli in case of newborns, or other *Enterobacteriaceae* (see Chapter 6.5.2.), such as *Klebsiella* (see Chapter 6.5.4.), *Proteus* (see Chapter 6.5.5.) and *Pseudomonas* (see Chapter 6.8.1.). Rare meningeal pathogens are certain species of the *Salmonella genus* (see Chapter 6.5.7.), the *Shigella genus* (see Chapter 6.5.9.) and of the *Clostridium genus* (see Chapter 6.17.) as well.

The in Chapter 6. afore-mentioned symptoms, such as fever, headache, vomiting, seizures, impairment of consciousness, stiff neck and back compose the meningitis syndrome, and are common to bacterial meningitis irrespectively of its etiology. If the initial symptoms are pain in the neck or the abdomen, as well as a confusional psychosis, an irritable or a delirious reaction, the diagnosing is much more difficult! These symptoms can progress to stupor, coma and finally even to death.

Meningococcal meningitis should always be suspected in epidemics of meningitis, if the evolution is extremely rapid; and the onset is attended by morbilliform, petechial, or purpuric skin eruptions, or by larger ecchymoses, and a lividity of the skin of the lower parts of the body; and if there occurs a circulatory collapse.

The Waterhouse-Friderichsen syndrome, an overwhelming, rapidly progressing infection caused by Neisseria meningitidis, is characterized by severe diarrhea, vomiting, seizures, internal bleeding, low blood pressure, shock and even by death.

Diagnosis: by using cerebrospinal fluid examinations and bacterial cultures, by testing the lactic-dehydrogenase isoenzyme levels. By using CT, MRI and other laboratory examinations.

Differential diagnosis: by distinguishing it from viral, tuberculous, leptospiral and fungal meningitis, from viral meningoencephalitis and from Mollaret's meningitis as well.

Treatment: the bacterial meningitis is a medical emergency. The rapid destruction of bacteria in the meninges and the cerebrospinal fluid is essential to survive the infection. For this reason, bactericidal drugs should be administered as soon as possible. The substitution of fluids lost by fever, sweating, vomiting and poor drinking is most necessary. A combination of intravenous antibiotics with intravenous corticosteroids and shock therapy is often needed.

RFR method is to be used simultaneously with the continuously administered antibiotic therapy.

10.28.2. The Viral Meningitis Syndrome

Viral or aseptic meningitis is an inflammation of the meninges caused by a virus. Encephalitis is an inflammation of the brain caused by a virus. Encephalomyelitis also caused by a virus is an inflammation of both the brain and the spinal cord. Several different viruses can infect the brain and the spinal cord, including f.i. also the herpes viruses and the mumps virus, too. Some certain kinds of the viral meningitis occur in epidemics and are spread by insects. Other viruses do not directly infect the brain and the spinal cord, but rather cause immune reactions which indirectly lead to the inflammations of these areas.

The majority of the cases of viral meningitis are caused by either: the *Polioviruses* (see Chapter 5.1.5.2.1.), or the *Coxsackie viruses* (see Chapter 5.1.5.2.2.), *ECHO viruses* (see Chapter 5.1.5.2.3.), *mumps* (see Chapter 5.1.2.3.), *measles* (see Chapter 5.1.2.4.), *rubella* (see Chapter 5.1.7.2.), *chickenpox* (see Chapter 5.2.4.2.1.), *Herpes viruses* (see Chapter 5.2.4.1.), *Arboviruses* (see Chapter 5.1.7.), *Myxovirus influenzae* (see Chapter 5.1.1.1.),

Epstein-Barr Virus (see Chapter 5.2.4.3.), or rarely *Hepatitis* (see Chapter 5.2.5.) or *Arenaviruses* (see Chapter 5.1.7.5).
Viral brain infections can cause three different group of symptoms. Some infections are mild, causing fever and a general feeling of illness, often without any special symptoms. Viral meningitis usually produces fever, headache, vomiting, weakness and a stiff neck. Encephalitis disrupts the normal brain function causing personality changes, seizures, weakness of one or more parts of the body, confusion, sleepiness that can develop to coma and the symptoms of meningitis, too.
Diagnosis: by CT, MRI, and by the identification of viruses.
Differential diagnosis: by distinguishing it from bacterial meningitis and other types of non-viral meningitis.
Treatment: by administering antiviral drugs and symptomatically.
RFR method: detects and eliminates the viruses.
As to the resonance frequencies of the certain viruses see Chapter 5.

10.28.3. Haemophilus Meningitis

Haemophilus influenzae meningitis is an infection of the membranes covering the brain and the spinal cord (meninges) caused by H. influenzae bacteria. These small, oxidase-positive, pleomorphic, Gram-negative aerobic or facultatively anaerobic coccobacilli can be sorted into 2 strains, the encapsulated and the unencapsulated ones. The encapsulated strains are surrounded by a polysaccharide capsule which plays an important role in the determination of the virulence of the microorganism. The outer membranes consisting of lipo-oligosaccharides (LOS) also contribute to the degree of virulence. The capsular antigens of the encapsulated strains are divided into 6 serotypes designated as A, B, C, D, E and F. The unencapsulated strains lack polysaccharide capsule and are designated as untypeable strains. *Haemophilus Influenzae B (HIB) is the most virulent encapsulated strain* accounting for more than 95% of all cases of H. influenzae meningitis in the prevaccination era. As the result of the historical migration of people, the HIB strains have achieved a worldwide distribution. Their clones express a repeating polymer of polyribosyl-ribitol-phosphate (PRP) in their capsules being a particularly important virulence factor.
Haemophilus nfluenzae meningitis (HIBM) may follow an upper respiratory way infection, and may develop slowly, or quickly. The infection usually spreads from the respiratory tract to the bloodstream, and then to the meninges, causing there an inflammation which develops to a serious illness and leads even to death.
The infection of a colonized person occur either in an invasive or in a noninvasive way.
Epiglottitis is an example of the noninvasive infections occuring in the upper airways of susceptible individuals, which are boys aged 3-6 years. They have the highest risk to get this form of disease having a particular susceptibility. Somewhat younger children, mostly boys have the susceptibility for HIB meningitis. The reason for these susceptibilities and risks is not known yet.
In case of invasive infections the HIB bacteria enter the bloodstream from the nasopharyngeal colony, becoming locally invasive. The mechanism of this invasiveness is not cleared up yet, but most likely involves bacterial and host's factors resulting in incapacity to contain HIB bacteria.
The infection of distant parts of the body can occur only by having a particularly in higher degree of bacteremia, sufficient to overcome the bacterial defense systems of the host. The containment of colonized bacteria is possibly easier for the hosts than the clearance of bacteria within the circulating blood. The elimination of the HIB bacteria from the circulation can only occur in case of a well functioning spleen, and a well functioning humoral and cellular immune system. Preceding viral infections are favourable for the invasiveness of HIB (i.e. to get from the colonized site into the blood stream or from the

blood stream into the target tissues) either because of their disrupting of barriers or due to their capacity to weaken the host's immuneresponse. Upper respiratory way infections, or otitis media presumably caused by viruses, often precede HIB meningitis. Once a higher degree of bacteremia is achieved, one or more places of the body may become infected. The bacterial invasion of the nervous system getting through the venous drainage from the locus of the nasopharyngeal colonization to the vulnerable nearby central nervous system loci (f.i. the cribriform plate, the thin sinus walls) or, more likely, by using the blood flow to get to the places with reduced blood brain barrier function (f.i. the choroid plexus).
The passage into the blood circulation as well as the immunologically privileged CNS is made easier not only by the capsular epitopes, which do not arouse an effective host's immune response, but also by those epitopes which take part in the bacterial attachment to the given endothelial.
The polysaccharide capsule of the HIB bacteria is not only responsible for the virulence and the invasiveness, but provides also a resistance against opsonization and against the complement-mediated bactericidal activities, and inhibits the neutrophil phagocytosis as well.
Unencapsulated Haemophilus species most often cause noninvasive infections, the locus of which is usually contiguous with the upper respiratory tract. These unencapsulated species cause mostly sinusitis and otitis media, and less often noninvasive infections of the lower respiratory tracts among children; though they can cause also an in a community acquired pneumonia as well as a superinfection of chronic bronchitis among adults. These noninvasive infections can probably also be preceded by viral respiratory illnesses. Bacteremia due to unencapsulated Haemophilus species is rather rare.
Special care should be given to infants who did not receive HIB immunization or who are known to have a pertinent immunodeficiency, but, remember that in HIB-immunized children there may develop HIB meningitis due to an unrecognized immunodeficiency, vaccine failure, or if they are infected with an untypeable or with any other non-B Haemophilus strain.
Most children younger than 18 months suffering from fever and seizures but who show normal findings when examined (i.e. show no sign of meningismus), certainly do not have meningitis.
At the time of the setting of HIB meningitis epidemics due to particularly virulent strains there were fulminant ceses among older infants and toddlers. In fulminant cases, a more intensive medical attention is needed, because of the possibility of medical emergencies such as coma or status epilepticus.
Infants younger than 2 months do but very seldom develop HIBM, justifying in part the current vaccination schedule for children. In rare instances when these very young infants do develop HIBM, the manifestations tend to be fulminant, even if thee is no contemporary evidence for an epidemic due to a particularly virulent HIB strain. Presentations in these cases suggest sepsis because the infants tend to be moribund with high fever. Meningismus may or may not be found. Pneumonia with a pneumatocele formation, pericarditis, or osteomyelitis may further complicate the diagnosis and the management of these severely ill infants.
Meningitis in a child older than 5 years is much more likely to be caused owing to meningococcus or pneumococcus than to HIB, though there may be some cases of HIBM even among these older children, too. The risk of HIB of these older children or of adolescents is greatest among those, who have immunoglobulin production abnormatities or immunoglobulin function abnormalities, sickle cell disease or other causes of actual or functional splenectomy, nephrosis and some other forms of chronic renal disease, cystic fibrosis and some other forms of chronic pulmonary disease, history of malignancy requiring chemotherapy or radiotherapy (as well as other diseases requiring the use of immunosuppressive agents) and cranial defects associated with abnormal communications

of the external environment with the subarachnoid space. It is unclear whether diabetes mellitus or alcoholism, factors, which may predispose to get HIBM of adults, may predispose adolescents to get HIBM, too.
HIB meningitis does but rarely occur in case of adults. An adult patient's data concerning abnormal immunoglobulin production or function, actual or functional asplenism, nephrosis, diabetes mellitus, chronic alcoholism, or cerebrospinal fluid fistula favor the possibility, that the meningitis is caused by HIB.
Symptoms of the HIBM include irritability, poor feeding in infants, fever (the temperature in young infants may be below normal), severe headache (of older children and adults), nausea and vomiting, stiff neck or pain in neck when flexed, pain in back if the neck is flexed forward and the chin brought toward chest (older children), unusual body positions and sensitivity to light.
Meningeal signs that may be found in children include nuchal rigidity to passive flexions and the signs of Kernig or Brudzinski. Although resistance to passive neck flexion is found in most cases of childhood meningitis, Kernig and Brudzinski signs are found in approximately half of the cases.
Risk factors for HIB meningitis include age younger than 5 years, a compromised immune state (f.i. in case of a mycoplasmal or HTLV infection), immunologic illnesses (f.i. agammaglobulinemia, IgG2 subclass deficiency) illnesses or treatments resulting in immunocompromised state of the patient (f.i. neoplasm, AIDS, malnutrition, chemotherapy, radiotherapy etc.), lack of HIB immunization with conjugate vaccines, HIB colonization at a vulnerable age.
Diagnosis: by serology (antibodies in blood) showing recent exposure to H. influenzae, by blood culture, by the identification of HIB from liquor etc.
Treatment: The treatment of meningitis must be started as soon as the HIBM diagnosis is suspected, by administering intravenous (IV) antibiotics, f.i. Ceftriaxone or Cefotaxime, corticosteroids given to reduce hearing loss, a common complication of meningitis concerning children as well as mannitol to reduce subarachnoid space pressure, etc.
Prevention: by vaccination.
RFR method: detects and may eliminate Haemophilus influenzae and the agents of coinfections.
The most frequent resonances of Haemophilus influenzae are: 336-337 kHz
Use RFR method together with antibiotic therapy. The RFR method can immediately penetrate the brain.

10.28.4. Sleeping Sickness

The human African trypanosomiasis (HAT), or sleeping sickness, is an illness endemic in sub-Saharan Africa. It is caused by the flagellate protozoan, *Trypanosoma brucei*, which exists in two morphologically identical subspecies: i.e. *Trypanosoma brucei rhodesiense* (East African or Rhodesian African trypanosomiasis) and *Trypanosoma brucei gambiense* (West African or Gambian African trypanosomiasis). Both of these parasites are transmitted to human hosts by the bites of infected tsetse flies (Glossina species), found only in Africa (see also Chapter 8.6.1.). The pathological alterations of the central nervous system include perivascular infiltrations of the parasites into the interstitium of the brain and into the spinal cord leading to meningoencephalitis with oedema, bleeding and granulomatous lesions. The chronic CNS phase (i.e. the 2. stage or late stage) of the disease may develop over 10 years, its symptoms being persistent headache, daytime somnolence followed by nighttime insomnia, behavioral changes, mood swing changes, in some cases depression, loss of appetite and loss of weight. Seizures occur more often in case of children.)
Prevention: with DNA vaccines for there is no effective immunotherapy of acute and chronic Chagas' disease yet. Fighting the vector (Triatoma) by using sprays and paints

containing insecticides, synthetic pyrethroids, and by the improvement of the conditions of housing and sanitary in rural areas.
Treatment with drugs (f.i. Suramin, Melarsoprol etc.) for early- and late-stage diseases, can result in the solution of symptoms and the clearance of parasitemia. Lumbar punctures is necessary every 3 months for the first year in case of patients having recovered from East African trypanosomiasis and every 6 months as long as for 2 years in case of patients having recovered from West African trypanosomiasis.
RFR method: detects and may eliminate the trypanosoma!
Use RFR method and administer antibiotics at the same time!
The most frequent resonances of Trypanosoma species are:
T. cruzi: 459-466 kHz
T. brucei brucei: 422-432 kHz
T. brucei gambiense: 323-325, 357-359, 369-371, 392-399, 412, 522 kHz
T. brucei rhodesiense: 423-429 kHz
T. abrassari: 305, 332-336, 531 kHz
T. equiperdum: 434-452 kHz
T. lewisi: 424-426 kHz
The frequencies of some trypanosoma species unidentified yet, are: 300-340, 356-372, 392-420, 520-560 kHz

10.28.5. Naegleria Fowleri Meningoencephalitis

Naegleria fowleri is a ubiquitous free-living ameba, the etiologic agent of primary amebic meningoencephalitis (PAM), which is a rare and usually fatal disease. This free-living thermotolerant amoeba belongs to the phylum of Percolozoa distributed worldwide, mainly in warm (from 25-35 degrees Celsius i.e. 77-95 degrees Fahrenheit), aquatic environments, less commonly in soil and sewage.
It exists in forms of trophozoite and cyst and in a transient flagellate form as well. PAM usually occurs after swimming or diving in warm water contaminated with N fowleri. The N fowleri trophozoites enter the nose, invade the olfactory mucosa, penetrate the submucosal nervous plexus, cross the cribriform plate and get into the subarachnoid space. The protein and glucose content of the cerebrospinal fluid supports the growth of amebae, which, multiplying rapidly invade the parenchyma of the brain causing thus PAM. The invasive trophozoites are highly phagocytic ingesting red blood cells and brain tissues causing thus hemorrhagic necrosis of the region involved. N fowleri secretes lysosomal hydrolases, phospholipases. heat-stable hemolytic proteins, heat-labile cytolysin, phospholipase A and a cysteine protease. All these products can kill the cells being in contact with the trophozoites.
PAM is usually a diffuse hemorrhagic meningoencephalitis associated with purulent meningitis. The cortical gray matter is the most severely involved area. Because of the severe edema of the brain, the pressure of the cerebrospinal fluid will be elevated, an uncal or a cerebellar herniation can occur as well as cranial nerve palsies involving the III., the IV., and the V. nerves.
Symptoms: This illness is characterized by changes in olfactory perceptions (taste and smell), vomiting, nausea, fever, headache, later on by cerebellar ataxia, reduced deep tendon reflexes and by a rapid onset of coma and death within two weeks. Patients often have papilledema, nystagmus and in the final stage just prior to death, decerebrate posturing, too.
Above all this, this ameba can cause neutrophilic myocarditis; though amebic trophozoites are not present in the myocardium.
The medical care of patients with PAM is complicated because of the rarity of the disease, the difficulty in early diagnosing, and because of the fact, that N fowleri is rapidly lethal. Once the clinical symptoms begin, the patient has but a very short time for an effective

therapy. The very high mortality rate of PAM suggests that most PAM patients are already in an irresponsible stage of the illness while searching for a medical care.
Diagnosis: PAM can be diagnosed by the observation of N. fowleri trophozoites f.i. by Giemsa stained cerebrospinal fluid showing the trophozoites with large karyosome and contractile vacuoles, by direct wet-mount microscopy showing the trophozoites with lobopodia extension and retraction. Indirect immunofluorescence patterns are available in some laboratories.
Polymerase chain reaction (PCR) testing is available in some research institutes using a number of different primers.
Serologic testing has no role in the diagnosis of acute PAM as the short time from the onset upto death is not long enough to produce measurable antibodies.
Treatment: by administering amphotericin B together with rifampin, sulfonamides, chloramphenicol, doxycyclin and miconazole may be useful even if they are synergistic with amphotericin B. Aggressive antibody treatment can be effective. Supportive therapies can be needed. The destruction of N. fowleri may cause toxic symptoms.
RFR method: detects and may eliminate N fowleri. Use RFR method as soon as possible together with the traditional medical treatment!
The most frequent resonances of Naegleria fowleri are: 352-371 kHz
The RFR method with other frequencies eventually found can help to prevent secondary bacterial infections.

10.28.6. Tropical Eosinophilic Meningitis

The nematode (i.e. roundworm) *Angiostrongylus cantonensis*, a rat lungworm, is the cause of human tropical eosinophilic meningitis (TEM).
(An other Angiostrongylus species i.e. the *Angiostrongylus costaricensis* infects normally rats, but is also the causative agent of human abdominal or intestinal angiostrongyliasis, predominantly infecting children living in countries from the USA to Argentina, including some Caribbean Islands. The life cycle of *Angiostrongylus costaricensis* is similar to that of *Angiostrongylus cantonensis*, but the adult forms of this worm reside in the arterioles of the ileocecal area of the definitive host releasing there eggs into the intestinal tissues. The eggs and larvae causing intense local inflammatory reactions, then degenerate, so that, they can not be found in the stool.)
Most cases of TEM have been reported from Southeast Asia and the Pacific Basin, though the infection is spreading to many other areas of the world, including Africa, the Caribbean, Sri Lanka and China as well.
The disease can be acquired by ingestion of by worm contaminated or infected animals (f.i. crabs, freshwater shrimps, raw snails or fish). Infecting human beings, the juvenile worms migrate to the brain, or rarely to the lungs, and ultimately die there. This nematode produces extensive tissue damages by moving through the brain and provokes a marked inflammatory reaction when dead. The pathological lesions are characterized by lymphocytic and eosinophilic infiltrations of the meninges; by hemorrhagic worm tracts through the brainstem and the spinal cord; and by granuloma formation around the dead parasites. Necrosis of the vessel walls, aneurismal dilatation of the arteries and perivascular hemorrhages may also be present.
Symptoms: TEM is sometimes characterized by mild fever, headache, and stiffness of the neck. The disease is generally benign and self-limited. An acute severe headache is the most significant symptom, in half of the cases abnormal neurologic findings can be found, too. Visual impairment, abnormal fundus, involvement of the cranial nerves, impairment of the sensorium and weakness of the extremities without localization can be noted mostly among severely ill patients.
Diagnosis: symptomatically, if living in epidemic area.

Differential diagnosis: by distinguishing it from toxocariasis, paragonimiasis, hydatid disease, schistosomiasis japonicum, trichinosis, cysticercosis, gnathostomiasis, etc.
Treatment: there is no effective treatment of the brain process known as yet. Prasiquantel or similar drugs may help.
Prevention: by avoiding to eat infected and not properly cooked food f.i. nails, praws, crabs, etc. Raw vegetables should be carefully inspected to exclude the presence of planarians and mollusks before meals.
RFR method: can detect and eliminate the parasite.
The most frequent resonances are: 337-340, 357, 369-374, 377, 381-389, 410, 432, 482, 565-570 kHz
The RFR method may cause severe pain owing to the moving of the worm in the brain.

10.28.7. Chronic Meningitis

Chronic meningitis is a brain infection causing inflammation in the meninges lasting for a month or longer. The most common causative agents are viruses (f.i. the *cytomegalovirus, the herpes viruses, the HIV virus*), bacteria (f.i. the causative bacteria of *tuberculosis, syphilis* and *Lyme disease*) and the fungus *cryptococcus* as well.
The symptoms of chronic meningitis are similar to those of bacterial meningitis, but the illness develops more slowly, usually rather over weeks than days. The fever is usually less severe than in case of bacterial meningitis. Headache, confusion, backache and even nerve abnormalities are common.
Diagnosis: can be done on the basis of symptoms; using CT, MRI, examinations of the cerebrospinal fluid and the blood, etc.
Differential diagnosis: by distinguising it from brain abscesses and brain tumors.
Treatment: by administering intravenous antimicrobial drugs (depending on the causative microorganism) f.i. amphotericin B, flucytosine, fluconazole, antibiotics, Acyclovir, and in some cases corticosteroids, too.
RFR method: can be effective.
As to the resonance frequencies of the Cryptococcus, see Chapter 7.1.3.
As to the resonance frequencies of the Cytomegalovirus, see Chapter 5.2.4.4.
As to the resonance frequencies of the Herpes viruses, see Chapter 5.2.4.
As to the resonance frequencies of the HIV, see Chapter 5.1.10.2.
As to the resonance frequencies of the M. tuberculosis, see Chapter 6.14.4.1.
As to the resonance frequencies of the Syphilis, see Chapter 6.19.2.
As to the resonance frequencies of the Lyme disease (Borrelia B.s.l.), see Chapter 6.20.3.

10.28.8. Other Types of Meningitis

The diseases sorted in this group include *psittacosis* (see Chapter 6.13.2.1.), *Q-fever* (see Chapter 6.6.2.) some mostly tropical arboviral infections such as the *O'nyong-nyong fever*, the *Mayaro viral disease*, the *Semliki Forest viral diseases*, the *Rift Valley fever*, the *Venezuelan equine encephalitis* (see Chapter 5.1.7.1.) and some rare illnesses mostly of unknown origin f.i. the *Behçet's disease* (see Chapter 23.24.3.), the *Vogt-Koyanagi-Harada Syndrome* (see Chapter 23.28.), and the *Mollaret's disease*. The neurological manifestations occuring in case of these aseptic meningitis forms are mild, they cause fever and a general feeling of illness. Specific symptoms, such as headache, vomiting, weakness, lethargy and a stiff neck can also be present.
Diagnosis: by the examination of the cerebrospinal fluid, by the measuring of antibodies against viruses, by MRI, by CT.
Differential diagnosis: by distinguishing it from other forms of meningitis, and other brain processes f.i. stroke, hematoma, aneurysm, or tumors.
Treatment: by administering antiviral agents and analgetics.

RFR: could detect and eliminate the viruses, but the exact frequencies of these groups of viruses have not been found yet.

10.28.9. Central Nervous System Candidiasis

Candida was a relatively uncommon CNS pathogen until the 1960s when the use of chemotherapeutic agents, glucocorticoids, intensive antibiotic therapy and intravenous drugs rendered increasing numbers of patients susceptible to opportunistic infections. Despite the high prevalence of mucosal candidiasis and that of the immunodeficiency of patients suffering from infections caused by *HIV/AIDS*, other *HTLVs* and other immunodepressant microorganisms f.i. *Mycoplasma fermentans*, Candida rarely causes invasive candidiasis of any form, nor CNS candidiasis.

Candida can infect both the meninges and the parenchymal brain tissue. Practically the majority of cases of CNS candidiasis are associated with disseminated or invasive candidiasis. A variety of histopathologic and clinical patterns have been described.

Candida meningitis is the most frequent clinical manifestation of invasive candidiasis-related CNS-candidiasis. It affects mostly neonates. As discussed later on, it has a chronic and indolent course among adults, while in case of neonates it usually is an acute process. Candida meningitis is one of the most common manifestations of neonatal invasive candidiasis. This type of meningitis typically subacutely evolves fever, headache, meningismus and diminished consciousness over several days to weeks. Focal neurologic signs, cranial nerve deficits, papilledema and seizures are infrequent. The chronic form of Candida meningitis mimics tuberculous or cryptococcal meningitis. Patients infected with Candida glabrata species are known to become progressively obtunded within weeks to months. In newborns and especially in premature babies the respiratory distress is a common manifestation of Candida meningitis caused by different species and is clinically indistinguishable from bacterial meningitis or sepsis.

Candida brain micro and macro abscesses have also been reported in literature. Most have been secondary to a primary source of candidiasis, including previous untreated episodes of candidemia. Candidal abscesses are less common than is meningitis. Its variable clinical picture consists of fever, consciousness, seizures and certain focal manifestations depending on the size and locus of the abscess.

Vascular complications include the invasion of the arteries at the base of the brain. Cases with evidence of vasculitis, thrombosis, infarction, intraluminal proliferation of Candida spp. and small vessel invasion were all described concerning this illness. Clinical presentations include basilar artery thrombosis and subarachnoid hemorrhages resulting from rupture of mycotic aneurysm or arteritis with vascular invasion. Candida pachymeningitis results in cranial nerve palsies. Patients with a spinal disease complain of back pain and show various neurologic signs and symptoms.

Diagnosis: by CT and MRI, which can be able to detect parenchymal abscesses or even a meningeal inflammation, by microscopic examination of the cerebrospinal fluid which can detect the Candida filamentum.

Differential diagnosis: by distinguishing it from cryptococcosis, histoplasmosis, tuberculosis, etc.

Treatment: by using first line antifungal agents for the treatment of CNS candidiasis, such as Amphotericin B, Fluconazole and 5-fluorocytosine.

RFR method : can detect Candida species.

The most frequent frequencies of C. albicans are: 380-390, 443-453, 572-586 kHz

The most frequent frequencies of C. tropicalis are: 345, 359-362 kHz

As to the other frequencies of C. albicans and Candida tropicalis, see their special Chapter.

10.29. Encephalitis Syndrome

The encephalitis syndrome, an acute, febrile disease is usually caused by *viral* infections. The illness shows an evidence of meningeal involvement, added to which, there are various combinations of the following symptoms and signs present: convulsions, confusion, stupor or coma; aphasia or mutism; hemiparesis with the asymmetry of tendom reflexes and Babinski sign; involuntary movements, ataxia, somnolence and myoclonic jerks; nystagmus, ocular palsies and weakness of the facial muscles. The residual signs of encephalitis can include mental deterioration, amnesic defect, personality change, Parkinson's syndrome and hemiparesis. The symptoms of encephalitis are caused by the brain's defense mechanisms activated to over come the infection. In some cases, these illnesses can quickly lead to death, f.i. among people suffering from viral tropic encephalitis death occurs even in 5 to 30 percent of the cases.
Though numerous tropical and non-tropical bacteria, fungi and parasites can cause an encephalitis syndrome, but the term encephalitis is nethertheless only used concering encephalitis syndromes of viral source. The number of viral infections and post viral allergic reactions is large, and one might suppose that clinical problems would be infinitely complex.
Diagnosis: according to he clinical picture, by examination of specific antibodies, virus isolation, MRI, etc.
Treatment: symptomatic and supportive. By admiistering effective antimicrobic drugs in case of bacterial or fungal infections.
RFR method: detects and may eliminate the pathogen agents!
The most frequent resonances of the viral encephalitis syndrome are: 289, 336-339, 372, 379-384, 396-397, 402-405, 410, 430-439, 449-451, 472-475, 552-555 kHz

10.30. Acute and Chronic Transverse Myelitis

Transverse myelitis is a neurological disorder, in case of which the transmission of the nerve impulses up and down elong the neurons in the spinal cord is totally blocked at one or more points. The spinal cord appears to be vulnerable in case of three types of inflammatory processes. There are some specific viral infections which tend to attack principally the gray matter. The *Herpes Zoster Virus* and *other Herpes viruses* (see Chapter 5.2.4.) and tree viruses of *poliomyelitis* (see Chapter 5.1.5.2.1.) are the most frequent examples. Secondly, all the subacute and chronic primary meningeal infections may damage the spinal roots and the outer surfaces of the spinal cord. *Syphilis* (see Chapter 6.19.2.) and *Borreliosis* (see Chapter 6.20.3.) offers the best known numerous examples of this category of inflammatory diseases. *Tuberculous* (see Chapter 6.14.4.1.) meningitis and fungous (see Chapter 7.) meningitis provide some other examples. Thirdly, there is a group of primary inflammations of the white matter, i.e. the leucomyelitis f.i. the postvaccinal myelitis after *smallpox, rabies and chickenpox*, the demyelinative myelitis in Multiple Sclerosis, and the necrotizing myelitis.
In case of patients, suffering from Multiple Sclerosis and certain bacterial infections, or who inject heroin as well as amphetamine may develop an acute transverse myelitis. In case of an acute and in case of a chronic myelitis there may be an allergic and an autoimmune reaction or process present.
Symptoms: Acute transverse myelitis usually begins with a sudden pain in the back, followed by numbness and muscle weakness, starting in the feet and moving upward. These effects may get worse within several days and then, if getting even more severe, can cause paralysis, loss of sensation and loss of bowel and bladder control. He higher the block in the spinal cord develops, the more severe the effects will become.
Diagnosis: of transverse myelitis is suggested by dramatic neurological symptoms. It is very important to examine the spinal fluid. Use CT and MRI examinations, too.
Differential diagnosis: by determining viral, bacterial, fungal infections and allergic or autoimmune processes.

Treatment: by administering antimicrobic drugs and corticosteroids, stopping allergic and autoimmune processes.
RFR method: detects and eliminates the pathogen microorganism such as the virus, the bacterium and the fungus. RFR method is very important in processes caused by viral infections. The microorganisms disintegrated owing to RFR method or antibiotics may cause allergic and autoimmune processes.
I think, that the RFR method can make a favourable turn in the treatment of transverse myelitis, if the infection is of viral origin.

10.31. Epilepsy

Epilepsy is a disorder characterized by recurring seizures. This convulsive disorder is the expression of a sudden, excessive, disordered discharge of neurons either in a structurally normal or in a diseased cortex. The discharge can cause an almost instantaneous disturbance of sensation, the loss of consciousness, convulsive movements (EEG) shows abnormal electrical activities and if the magnetic resonance imaging (MRI) reveals scarring in small areas of the brain. In some cases, these defects are microscopic scars resulting from brain injury at birth, or later. Some specific types of seizure disorders, f.i. the juvenile myoclonic epilepsy are inherited. These epileptic seizures may be triggered by repeated sounds, flashing light, or even by being touched on certain parts of the body. Even minor stimuli can trigger a seizure in people with epilepsy. Reflex epilepsy suggests that epilepsy is a natural state, a physiologic event resulting from excitation and from the subsequent inhibition of a damaged part of the cerebrum. Eventually, the physiologic event initiating the seizure is a high-voltage discharge of some certain assemblages of the cortical neurons. The seizures can be initiated in an entirely normal cerebral cortex too, without there being any visible macroscopic lesions, f.i. if the cortex is activated by a drug or injured by hypoxia, or by some microbic substances. But it is the visible focal lesion that has been the most thoroughly investigated. Some of the electrical properties of the cortical focus suggest that its neurons are deafferented. Deafferented neurons are hypersensitive; they remain chronically in a state of partial depolarization. Their cytoplasmic membranes have an increased permeability renders them susceptible to activation by hypertermia, hypoxia, hypoglycemia, hyponatremia by the metabolites of microorganism, as well as by repeated sensory stimulation and during certain phases of sleep.
The post-seizure state, named also postconvulsive paralysis of cerebral function, has also EEG alterations, correlated with random way generalized slow waves. With the recovery of the normal mentation the EEG returns to normal.
Simple partial seizures begin with electrical discharges in a small area of the brain, the discharges remaining confined to that area. The person experiences abnormal sensations, movements, or psychic aberrations, depending on the part of the brain affected.
In case of **jacksonian** seizures, symptoms begin in one of the isolated parts of the body, such as the hand or foot.
Complex partial seizures begin with a period of 1-2 minutes, during which the person losing its touch with the surroundings. The person may stagger, move the arms and legs in a strange and purposeless way, utter meaningless sounds, fail to understand what others say and resist any help. The confusion lasts for several more minutes, followed by full recovery.
Convulsive seizures, is a grand mal with tonic- clonic seizures.
Petit mal does not produce any convulsions, nor any dramatic symptoms what so ever.
In **status epilepticus**, which is the most serious seizure disorder, the seizure doesn't stop. Status epilepticus is a medical emergency, the person having convulsions with intense muscle contractions, being unable to breathe properly, having widespread electrical discharges in the brain.
Diagnosis: by complex EEG, MRI, and symptomatically.

Differential diagnosis: by distinguishing it from brain tumor, drug intoxication, psychosis and dementia.
Treatment: by administering f.i. Carbamazepine, Ethosuximide, Gabapentin, Lamotrigine, Phenobarbital, Phenytoin, Primidone, Valproate, Celontin, Diamox, Tegretol, Mebaral and Dilantin.
These drugs may have side effects f.i. such as sedation, rash, swollen gums, hair loss, anemia and libido loss.
RFR method: is only effective in those special cases, in which a microorganism plays a role in the disease.
The most frequent resonancies are: 307, 324, 332, 358, 372, 402, 410, 428, 496-500 kHz

10.32. Poliomyelitis

Poliomyelitis is a highly contagious, sometimes fatal, viral infection which can produce permanent weakness, paralysis and other symptoms. *Poliovirus*, an enterovirus, is spread by swallowing material such as water contaminated by infected feces. The infection spread from the intestine throughout the body, but the brain and the spinal cord are the most severely affected organs.
The causative agent of poliomyelitis is an RNA virus of the picorna group. Three antigenically distinct types are defined: Brunhilde, Lansing, and Leon. Cross neutralization is verificated in case of highly immunized experimental animals, but if a person gets infected by one of its types, he will not be protected against the invasion of another of its types. Poliovirus is worldwide present, but epidemics had been limited to a relatively small number of areas. That the infections are much more prevalent, than supposed by the number of clinically recognized cases, is proved by the widespread distribution of neutralizing antibodies in the population all over the world.
The incubation time of this infection varies from 3 to 40 days. The illness has three forms i.e. the minor illness, the nonparalytic poliomyelitis, and the paralytic poliomyelitis.
Symptoms: regarding the *minor form of poliomyelitis* are nonspecific and do not show any clinical or laboratory evidence of the viral invasion of the central nervous system. There is only fever and upper respiratory tract manifestations present, such as a feeling of pharyngeal discomfort, the reddening and swelling of the lymphoid tissue of the throat. Gastrointestinal disturbances with nausea, vomiting, diarrhea or constipation; a grippe-like disease can occur, as well. The virus can be found and identified in the pharynx.
In case of nonparalytic poliomyelitis there are signs of meningeal irritation and abnormalities in the spinal fluid. Stiffness of the neck and the back, a positive Kernig's sign and a severe meningeal irritation are characteristic.
In case of paralytic poliomyelitis: meningeal irritation, findings of abnormal spinal fluid and the involvement of the motor nerve cells in the spinal cord and the brain are characteristic, as well as the involvement of the cranial nerve nuclei, resulting in paresis or paralysis of various muscles. Lesions of the anterior horn cells may also be present. The Auerbach's and the Meissner's plexus and the sympathetic ganglions are usually involved in fatal cases. The disease begins with fever and with certain signs of the minor form of the illness. All symptoms disappear within 6-8 days, though the fever will return together with the development of a meningeal irritation and paralysis. Children usually have upper respiratory tract syndromes as well.
In the early stage of spinal paralytic poliomyelitis there are severe cramping pains in the muscles innervated by the affected neurons, as well as hyperesthesia of the overlying skin. Paresis of one leg develops most often in case of children less than five years old. Paralysis of the muscles of respiration is most common among those over sixteen years of age. In case of men there develop quadriplegia and respiratory paralysis, while the loss of bladder

function occurs more frequently among women. The disease affecting the lumbar portion of the spinal cord causes weakness of the legs, affecting the inferior portions it weakens the muscles of the abdomen and the back. Pain, tenderness, spasm and twitching herald the oncoming paralysis, the reflexes are abolished in the time of the development of flaccid paralysis.

Encephalitic symptoms can occur either isolated, or together with bulbar or spinal poliomyelitis. The incidence of polioencephalitis is variable. In fatal cases, the confusion is marked, progressing to lethargy and death. In case of focal polioencephalitis, there may be a clinical evidence of brain damage, though the lesions may be without any sign. Depending on the fact, which parts of the brain and the spinal cord are affected, in certain cases the disease does not progress any further, while in other cases weakness and paralysis will develop in certain muscles. The person may have difficulty in swallowing and may choke on saliva, food or fluids.

Diagnosis: symptomatically, and by identifying the poliovirus in stool samples and by detecting high level antibodies to the virus in the blood.

Prevention: polio vaccination is included in the routine childhood immunizations in developed countries. Two types of vaccines are available: an inactivated poliovirus vaccine (Salk vaccine) given by injection and a live poliovirus vaccine (Sabin vaccine) taken orally. The live oral vaccine provides a better immunity it is therefore usually preferred. In very rare cases, the live vaccine can cause polio, especially in people who have an impaired immune system.

Treatment: symptomatically. Polio can't be cured, neither do antiviral drugs affect the course of the disease.

RFR method: detects and eliminates the virus.

The resonant frequencies of the poliovirus are: 289, 336-338, 372-379, 382-385, 397, 403, 419, 438, 450, 473, 488, 493, 552, 576-579 kHz

10.33. Herpes Simplex Virus Infections in the Nervous System

HSV, belonging to the family of Herpesviridae is a double-stranded DNA virus characterized by the following unique biological properties:

Neurovirulence, the capacity to invade and replicate in the nervous system including the brain, brainstem, spinal cord, cranial and peripheral nerves, as well as ganglions.

Latency (the establishment and maintenance of latent infection in nerve cell ganglia): In case of infection caused by HSV1, the trigeminal ganglia are involved most often, while in case of infection caused by HSV2 the sacral nerve root ganglia (S2-S5) are involved, but the Herpes virus may infiltrate other cranial and peripherical nerves of the ganglia or the brain and the spinal cord as well.

Reactivation: The reactivation and replication of latent HSV can be induced by a variety of stimuli (eg., fever, trauma, emotional stress, sunlight, menstruation), resulting in overt or covert recurrent infections and in peripheral shedding of HSV. In case of immunocompetent patients with equal chances of acquiring HSV1 and HSV2 both orally and genitally, HSV1 reactivates more frequently in the oral rather than the genital region. Similarly, HSV2 reactivates 8-10 times more frequently in the genital region compared to orolabial HSV2. Reactivation is more frequent and severe in case of immunocompromised patients.

Dissemination of infection occurs in people with impaired T-cell immunity such as organ transplant recipients and individuals suffering from a HIV-related disease.

Seroprevalence: Antibodies to HSV1 increase with age starting in childhood and correlate with the socioeconomic state. At the age of 30 years, 50% of individuals being in a high socioeconomic state and 80% of those being in a lower socioeconomic state are

seropositive. Antibodies to HSV2 begin to emerge at puberty, correlating with the degree of sexual activity. The lifetime seroprevalence can be 60-85%, while 5% of the infected persons have no antibodies.

Symptoms: Herpes simplex infections are asymptomatic in as many as 80% of patients, symptomatic infections may be characterized by significant morbidity and recurrence as well. Moreover, infections can cause life-threatening complications, particularly in case of immunocompromised hosts.

About 30-70% of older patients infected by herpes viruses, affecting the central nervous system and the peripherial nerves, have *neuritis* (intercostal neuralgia, lumbago, etc.), and in addition, if they have a herpes viral infection combined with infections caused by other microorganisms (for example Borrelia Burgdorferi s.l.) there may *Alzheimer's disease* as well as *Multiple Sclerosis* develop.

A strong association of HSV1 was found with *Bell's palsy.*

Acute and chronic aseptic meningitis. is an acute, usually benign lymphocytic meningitis. 36% of women and 13% of men suffering from primary genital HSV2 have meningeal symptoms. This illness does mostly occur in case of infections caused by HSV2. Meningeal symptoms usually start within 3-12 days after the onset of genital lesions; they reach a maximum for 2-4 days and then recede in 2-4 days. HSV2 and even also HSV1, were identified by PCR techniques in the cerebrospinal fluid of the patients suffering from benign recurrent aseptic meningitis, which fact suggests that HSVs may be the cause of a group of the formerly idiopathic syndrome named Mollaret meningitis.

Ganglionitis and myelitis: Genital and anorectal HSV infections may be complicated by urinary retention, sacral neuralgia and sacral anesthesia, caused by ganglionitis and radiculitis belonging to these areas. The symptoms usually resolve in 1-2 weeks. Transverse myelitis does occur but rarely.

Herpes simplex encephalitis: This is an acute necrotizing viral encephalitis, nearly always caused by HSV1, in the time after the neonatal period. This accounts for 10-20% of all cases of encephalitis and this cause, on the other hand, most often the cases of sporadic acute necrotizing encephalitis. Herpes simplex encephalitis occurs as a primary infection in about 50% of the cases and in case of recurrent infections and reinfections caused by different species of HSV1 as well. The clinical features include the following: nonspecific findings of encephalitis, including headache, signs of meningeal irritations, altered mental state and generalized seizures. Changes, referable to focal necrosis of the orbitofrontal and the temporal cortex, as well as the limbic system will develop, including anosmia, memory loss, olfactory and gustatory hallucinations and focal seizures, too. A rapid development of hemiparesis and coma may come about. The clinical manifestations may in some cases get worse, mimicking an acute psychosis or delirium tremens. In case of untreated patients the mortality rate is high (cc 70%). There often remain neurological sequelae even in case of treated patients.

Diagnosis: by the rapid detection of HSV DNA in clinical specimens using the most sensitive noninvasive *PCR techniques*. PCR can detect asymptomatic viral sheddings as well. In case of HSV encephalitis, the PCR examination of the cerebrospinal fluid provides also a rapid, most sensitive, noninvasive diagnostic method, as sensitive as the brain biopsy.

MRI is a sensitive imaging procedure in case of herpes simplex encephalitis demonstrating a focal localization in the temporal area associated with edema and the enhancements of contrast materials.

Examining the *cerebrospinal fluid samples* there is a moderate pleocytosis with mononuclear and polymorphonuclear cells present, a mildly elevated protein level as well as a normal glucose level. *Tissue cultures* for HSV are often positive within 48 hours of inoculation showing characteristic cytopathic effect, ballooning of cells and cell death. The

immunofluorescent staining of the tissue culture cells can quickly identify the HSV and distinguish type 1 from type 2.
The *antibody testing* can demonstrate primary seroconversions, particularly in case of infections of HSV1 in childhood. The sero-cross-reactivity between HSV1 and HSV2 can be excluded by using glycoprotein G antibody assay. The increase in antibody titer does not occur during the recurrences of HSV (in contradistinction to those occuring during the recurrences of HVZ).
Treatment: there is no effective drug as yet to definitively eliminate Herpes simplex viruses in the nervous system. Nucleoside analogs (f.i. acyclovir, valacyclovir, famcyclovir, etc. can inhibit the HSV polymerase 30-50 times more effective than the human alpha-DNA polymerase.
RFR method: can detect the specific resonances of HSV and eliminate the virus even in the CNS! (See also Chapter 5.2.4.1.)
The general ranges of Herpes Simplex Virus-1 are: 290-294, 344-346 kHz
Its other frequencies are: 302, 306-314, 335, 346-350, 380-383, 394-402, 413, 438, 474-478, 488-490 kHz
The general range of Herpes Simplex Virus-2 (genital) are: 352-365, 413, 425 kHz
Its other frequencies are: 307-309, 318, 338-341, 366-367, 372-375, 383, 396, 400-402, 410-412, 450, 420-422, 454, 475-476, 480-484, 533, 544 kHz
The species of the HSV1 group are not the same in Europe as that in the US or in Africa. The different ethnic groups have different herpes virus subpopulations. If the treatment is not definitive, the latent virus may reactivate, and the clinical symptoms reappear. The treatment of the brain process can cause cerebral edema requiring additional medical treatments. It is most important to eliminate also the other, co-factor pathogens present in case of Alzheimer's disease and Multiple Sclerosis as well. If the acoustic nerve is attacked by HSV, tinnitus and deafness may develop. During the treatment the tinnitus may increase, if the elimination of the virus is not complete. The deafness can not be cured by this method, but the development to deafness can be inhibited. HSVs may be latent in the host for a long time without causing any characteristic signs, though it can attack the cardiac sympathetic nerves as well, causing problems (f.i. hypertension, hypotension, tachycardia, bradycardia) at any time and during the killing process as well.

10.34. Tropical Spastic Paraparesis

Tropical spastic paraparesis (TSP) is a slowly progressing viral infection of the spinal cord that causes weakness in the legs. This infection is caused by the *Human T-cell Lymphotropic Virus (HTLV-1).* About this retrovirus, see Chapter 5.1.10.1.1. TPS may be spread by sexual contact or by contaminated needless. It can also be transmitted from mother to child either across the placenta or in the breast milk.
This seldom found disease caused by a HTLV infection is endemic among the Native-American population, as well as the Guaymi in Panama, the Native-Americans from Florida, New Mexico, and the Caribbean countries.
The symptoms may begin years after being infected. Within the process of responding to these infections caused by *HTLV-1 and HTLV-2).* the immune system may injure the nerve tissues, causing symptoms. Weakness and muscle stiffness in both legs begin gradually and get worse slowly. Certain sensations felt in the feet or/and the arms may cease. The immune response is an autoimmune process, in which a myelin degenerative disease can develop.
Diagnosis: the determing this HTLV infection should not rely solely on the results got by the PCR analysis. Patients proved to be positive concerning the presence of proviral DNA by polimerase chain reaction should be tested using additional assays, evaluated clinically, and retested using a new specimen before establishing the diagnosis.

Differential diagnosis: Multiple Sclerosis, chronic tranverse myelitis, prion disease, progressive multifocal leukoencephalopathy and a chronic amebic meningoencephalitis.
Treatment: Though there is no definite cure yet known, a marked improvement can be experienced among people treated with corticosteroids, such as cortisone, suppressing the autoimmune response. Plasmapheresis can produce a temporary improvement as well.
RFR method: detects and may eliminate the HTLV! This treatment has to go on for a very long time.
RFR method and corticosteroid therapy should be used together, as the virus elimination will increase the inflammation in the brain.
The resonances of HTLV-1 are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz
The resonances of HTLV-2 are: 314, 320-324, 370-376, 493-501 kHz
Opportunistic infections caused f.i. by fungi or bacteria, are very frequently present concerning the TSP. Detect the opportunistic microorganisms and eliminate them using RFR, but the infections have to be treated in the conventional medical way, as well.

10.35. Encephalomyocarditis Viruses Infection

The *encephalomyocarditis viruses* (EMC viruses), f.i. *Columbia-SK virus*, *Mengo virus*, are a group of small RNA viruses, immunologically indistinguishable from each other. Rodents, particular certain species of wild rats, constitute the major reservoir for EMC viruses. Certain strains were also isolated from primates, swine and rodents from many parts of the world. The strain of Mengo virus was isolated from mosquitoes, i.e. Taeniorhynchus fuscopennatus in Uganda. Human infections vary from mild febrile diseases to severe encephalomyelitis. This viral meningitis can be characterized by chills, fever, headache, stiff neck and pleocytosis of the cerebrospinal fluid. There were sporadic EMC virus isolates found in adults and children suffering from illnesses diagnosed as a paralytic poliomyelitis, and as a Guillain-Barré syndrome, as well as a severe meningoencephalitis. Myocarditis has not been observed regarding these EMC viral infections.
Diagnosis: by isolating the virus from from the blood and the cerebrospinal fluid. This EMC viral disease has specific antibodies, too.
Treatment: symptomatically and supportively.
RFR method: detects and may eliminate this virus!
The most frequent resonances are: 276-292, 300-305, 336-338, 379, 384, 397, 421-441, 473, 552-554 kHz

10.36. Sciatic Neuralgia, Lumbago, Ischias

The damages of the vertebrae and the discs between the vertebrae can put pressure on the nerve roots. The pressure can cause pain, which often worsens when moving the back, coughing, sneezing, or straining. If the nerve roots of the lower back are under pressure, the pain will be felt either only in the lower back, or along the sciatic nerves to the buttocks, thighs, calves and feet as well. Sciatica is the name of symptoms including these pains of the leg as well as weaknesses or numbnesses traveling from the low back through the sciatic nerve along the leg. The sciatic neuritis causes pain in the lumbar region and behind the leg from the buttock to the ankle. The pain is aching or burning and will aggravate by moving or straining. The sciatic nerve is tender to palpation or stretching. There may be a slight weakness of the hamstrings and the muscles below the knee. The ankle jerk reflex may be absent. The sensory impairments are usually slight. It is necessary to distinguish the symptoms of sciatic neuritis from those of sciatic compression. In case of compression, caused f.i. by tumors, the symptoms are more progressive f.i. the onset is more gradual, the muscle wasting more conspicuous, the nerve less tender to palpation, and the sensory loss greater. The inflammation of the sciatic nerves can be caused by *herpes*

viruses, by *other neurotrop viruses or by bacteria*. The patients suffering from this sciatic neuritis of infective origin can be treated effectively with RFR method.
Diagnosis: by using physical examinations, x-ray, CT, MRI, myelography and dermal electrical resistance measuring
Treatment: by administering analgesics, muscle relaxants.
RFR method:
1. detects the virus or the bacteria and eliminates them
2. can be used just like using electroacupuncture on a local dermal segment.
The most frequent resonances are: 344-345, 472-473 KHz
As to the frequencies of Herpes viruses, see Chapter 5.2.4.
Electroacupuncture method in case of lumbago happens using RFR method **on 90-120 Hz** sweeping with waveform square.

10.37. Intercostal Neuralgia

Intercostal neuropathic pains are caused by *Herpes zoster* infections affecting the intercostal nerves causing then postherpetic neuralgial pains. These chronic, burning type, neuropathic pains remain within the area infected with the virus and last for a long while. (See Chapter 5.2.)
Diagnosis: symptomatically
Treatment: solely by administering analgesics, so that there is no drug to eliminate the virus. However, antiviral drugs, f.i. acyclovir or famcyclovir can help.
RFR method: detects and eliminate thes Herpes Zoster Viruses.
The most frequent resonances are: 372, 396-399, 403-410, 440-444, 448-456 kHz, as the other frequencies see Chapter 5.2.4.2.2.

10.38. Facial Palsy Syndrome (Bell's Palsy)

In case of this syndrome, there are abnormal alterations to be found in the functioning of the facial nerve, leading to sudden weakness or to the paralysis of the muscles of one or two sides of the face. The facial nerve is the seventh cranial motor nerve stimulating the facial muscles. Some viral infections, f.i. *Herpes zoster*, s well as Lyme disease, *(Borrelia sensu lato)* or certain mechanical nerve damages may produce this syndrome.
In case of the complete interruption of the facial nerve at the stylomastoid foramen will paralyse every muscle of the facial expression. Food remains between the teeth and the lips, saliva may dribble from the corner of the mouth. The patient complains of heaviness or numbness of the affected par of the face, but no sensory loss is demonstrable and the taste remains intact. If the peripherial facial paralysis exists for some time and the motor function returns but incompletely, a kind of spasm may appear. The palpebral fissure becomes narrowed and the nasolabial fold will be deepened. Attempts to move one group of the facial muscles result in contraction of all. Facial spasms develop and persist indefinitely, being initiated by every facial movement. This condition, named facial spasms, may also appear in case of adults, who have never had a Bell's palsy.
The most common disease affecting the facial nerve is the Bell's palsy, presumably due to an inflammatory reaction in or around the nerve near the stylomastoid foramen. The onset is acute, the paralysis may be evolved within a few hours, though a pain behind the ear may have been felt for a day or two. Occasionally the taste sensation can be lost, and, rather rarely, hyperacusis is present. In some cases there can a mild pleocytosis in the cerebrospinal fluid be found.
Acoustic neuromas frequently involve the facial nerve. The paralyzed side becomes flat and expressionless. Most people experience numbness or a heavy feeling in the muscles of the face, the sensation actually remains normal. If the upper part of the face is involved, to close the eye on the affected side might be difficult. Bilateral facial paralysis (i.e. facial

diplegia) occurs in case of an acute idiopathic polyneuritis, in one form of sarcoidosis i.e. the Heerfort's syndrome as well as concerning the Melkerson's syndrome. A stroke can cause weakness of the facial nerve. Other causes of the facial palsy may include brain or other tumors that compress the nerve; the destruction of the facial nerve due to a viral infection, such as herpes viral infections (f.i. the Ramsay Hunt syndrome caused by HZV); infections in the middle ear or the mastoid sinuses. Lyme disease does often cause facial paresis, even on both sides. Fractures of the bone at the base of the skull and several other, even more rare disorders can be the cause of this syndrome.

Diagnosis: by the blood test concering the Lyme disease, Herpes virus examination, by x-ray, CT, MRI. There do not exist any labor test for Bell's palsy syndrome.

Treatment: Bell's palsy syndrome does not have any specific treatment. In the earlier phase of the inflammation the administering of corticosteroids, other antiinflammatory and/or analgesic drugs may be needed.

The most frequent resonances in case of Bell's palsy syndrome of viral or bacterial origin are: 290-294, 344-345, 353-362, 376-386, 415-421, 442-451 kHz

RFR method: is solely in the earlier stage effective. Detects and eliminates the herpes viruses and the borrelia species! A complete recovery can be achieved in 1 or 2 months if the paralysis is partial. The pain will disappear in case of viral or bacterial infections treated by RFR method.

10.39. Diabetic Neuropathies

Diabetic neuropathies (DNs) are neuropathic disorders associated with diabetes mellitus. These disorders are thought to be resulting from diabetic macro- and microvascular injuries involving the small blood vessels which supply the nerves of vasa nervorum. The most common injuries which may be associated with diabetic neuropathy are third nerve palsy, mononeuropathy, mononeuropathy multiplex, diabetic amyotrophy, painful polyneuropathy, autonomic neuropathy and thoracoabdominal neuropathy. DNs are characterized by the progressive loss of the nerve fibers which phenomenon can be assessed noninvasively by certain nerve function tests, including nerve conduction studies and electromyography, quantitative sensory testing and autonomic function tests. Excluding other causes, the presence of symptoms and/or signs of hypoxic dysfunction of a peripheral nerve in people with diabetes can be defined as diabetic peripheral neuropathy. DNs can be sorted into several syndromes concerning the distinct pattern of the peripheral nerve involvement. Patients often have multiple and overlapping syndromes.

Peripheral neuropathies have been described in patients with primary and secondary types of diabetes of diverse causes, suggesting a common etiologic mechanism based on chronic hyperglycemia, hypoxia and local anoxia. PN is the term for a nerve damage of the peripheral nervous system caused either by diseases of the nerve itself or by side-effects of systemic illnesses. Peripheral neuropathies vary in their symptoms and origin, and can affect the nerve and the neuromuscular junction.

Vascular and neural diseases are closely related and intertwined.

Indirect neurological hypoxic damage due to vascular damage: blood vessels depend on normal nerve functions, while nerves depend on their adequate blood flow. Vasoconstriction is the first pathological change in the microvasculature. *Nanobacteria* release endothelin 1-3, which are very strong vasoconstrictor substances. As the disease progresses, neuronal dysfunction correlates closely with the development of vascular abnormalities, such as capillary basement membrane thickening and endothelial hyperplasia, which contribute to a diminished oxygen tension and hypoxia. Neuronal ischemia is a well-established characteristic sign of diabetic neuropathy in case of *Coxsackie viral* infections. Vasodilator agents (f.i. ACE inhibitors, α1-antagonists) can lead to a substantial improvement in the neuronal blood flow, with corresponding improvements in the velocity of nerve conduction. A microvascular dysfunction (so thus

the progression of neural dysfunction as well) occurs early in case of diabetes mellitus, and leads to structural, functional and clinical changes observed in diabetic neuropathy. In diabetes mellitus type 1, a distal polyneuropathy becomes typically symptomatic after many years of prolonged chronic hyperglycemia. Conversely, in type 2 it may without treatment develop already after but a few years. Patients with diabetes mellitus type 2 may sometimes have neuropathy already at the time of being diagnosed.

Cause of direct neurological damages: *Coxsackie viral, Herpes viral and Mycoplasmal infections* can lead to immune-autoimmune demyelination processes, neural damages, combined with hypoxial damages.

Peripheral neuropathies are present in many a disorder, such as:

Genetic diseases: Friedreich's ataxia, Charcot-Marie-Tooth syndrome.

Metabolic/Endocrine diseases: diabetes mellitus, chronic renal failure, porphyria, amyloidosis, liver failure, hypothyroidism.

Toxic causes: alcoholism, drugs, organic metals, heavy metals, excess intake of Vitamin B6 (pyridoxine) etc.

Inflammatory autoimmune diseases: Guillain-Barré syndrome, SLE, Sjögren's syndrome.

Vitamin deficiency: Vitamin B12, Vitamin A, Vitamin E, thiamin.

Muscle problems (myopathies), sensory and motor disturbances may be present in many peripheral nervous system diseases, so that it is difficult to establish an accurate diagnosis.

Generalized peripheral neuropathies are symmetrical and are usually caused by various systemic illnesses and disease processes affecting the peripheral nervous system in its entirety. They can further be subdivided into several categories:

Distal axonopathies are the result of some metabolic and toxic derangement of neurons. They may be caused by metabolic diseases (such as diabetes), renal failure, deficiency syndromes (such as malnutrition and alcoholism) and by toxins and drugs.

Myelinopathies develop due to a primary attack on myelin causing an acute failure of impulse conduction. They can also be found f.i. in Acute inflammatory demyelinating polyneuropathy (AIDP), Guillain-Barré syndrome, Chronic inflammatory demyelinating polyneuropathy (CIDP), Genetic metabolic disorders and Leukodystrophy.

Neuronopathies are the result of the destruction of the neurons of the peripheral nervous system. They may be caused by motor neurone diseases, sensory neuronopathies caused f.i. by *Herpes Zoster Virus*, toxins and autonomic dysfunctions.

Sensorimotor polyneuropathy: Long nerve fibers are affected to a greater degree than shorter ones, as the nerve conduction velocity will in proportion to the nerve's length become slower. In this syndrome, a decreased sensation and the loss of reflexes occurs first in the toes bilaterally, extending then upwards. It is usually described as glove-stocking distribution of numbness, sensory loss, dysesthesia and night time pain. The pain can be felt like a burning, pricking sensation, achy and dull. Pins and needles sensation is common. The loss of proprioception, i.e. the loss of the sensation of where the limb is placed in the space, can be early experienced. These patients do not feel when they step on a foreign body (like a splinter), or if they have a callus caused by an ill-fitting shoe. Consequently, they are at risk of developing ulcers and infections on feet and legs, which can even lead to amputation. Similarly, they can get multiple knee, ankle and foot fractures and develop a Charcot joint. The loss of motor function results in dorsiflexion, contractures of the toes, loss of the interosseous muscle function leading to contraction of the digits, i.e. the so called hammer toes. These muscle contractures occur not only in the foot but also in the hand where the loss of the musculature makes the hand appear to be gaunt and skeletal. The loss of muscular function is progressive.

Autonomic neuropathy: the autonomic nervous system is composed of nerves serving the heart, the gastrointestinal system and the genitourinary system. Autonomic neuropathy can affect any one of these organ systems. Orthostatic hypotension, i.e. an uncomfortable

sensation of fainting when the patient stands up is the most commonly recognized autonomic dysfunction in case of diabetic patients. Diabetic autonomic neuropathy is the result of the failure of the heart and the arteries in their function to appropriately adjust heart rate and vascular tone in order to keep the blood fully and continually flowing to the brain. This symptom is usually accompanied by the loss of the sinus respiratory variation, that is, the usual change in the heart rate experienced when breathing normally. Cardiac autonomic neuropathy is defined by these two symptoms.

Manifestations of neuropathy in the GI tract are delayed gastric emptying, gastroparesis, nausea, bloating and diarrhea. The absorption of medicaments in case of diabetes mellitus can greatly be affected by a delayed gastric emptying. This fact can worsen the hypo/hyperglycemia of patients orally taking antidiabetic drugs. A sluggish movement of the small intestine can lead to bacterial overgrowth, worsen by the presence of hyperglycemia, all leading to bloating, gas and diarrhea.

Urinary symptoms include frequent urinating, urgency, incontinence and retention. Due to the retention of urine, urinary tract infections are frequent. Urinary retention can provoke bladder diverticula, bladder stones and reflux nephropathy.

Cranial neuropathy: Oculomotor (3rd) neuropathy is the most common cranial neuropathy. The oculomotor nerve controls all muscles moving the eye with the exception of the lateral rectus and the superior oblique muscles. It also serves to constrict the pupil and to open the eyelid. The onset of a diabetic third nerve palsy is usually abrupt, beginning with a frontal or periorbital pain and can be followed by diplopia. All oculomotor muscles innervated by the third nerve may be affected, excepting those which control the size of the pupil, as the pupillary function within CNIII is found on the periphery of the nerve, and, being closer to the vascular supply, will make them less susceptible to ischemic damages. NerveVI, the abducens nerve, innervating the lateral rectus muscle of the eye (i.e. moving the eye laterally), is also often affected, but the involvement of cranial nerve IV, i.e. of the trochlear nerve, (innervating the superior oblique muscle, moving the eye downwards) is unusual.

Mononeuropathies of the thoracic or lumbar spinal nerves can occur and lead to painful syndromes mimicing myocardial infarction and cholecystitis.

Diabetic radiculoplexus neuropathy may occur in cervical and lumbosacral distribution and is in literature referred to by various designations including diabetic amyotrophy, Bruns-Garland syndrome, diabetic plexopathy, etc.

Its most frequent initial symptom is a severe, sudden, unilateral pain in the hip/lower back and the shoulder/neck. Weakness does develop days to weeks later. Atrophy of the limb musculature may occur. Allodynia, paresthesia and sensory loss are common. These symptoms usually begin unilaterally and may later be experienced on the opposite side as well.

Reflexes of the affected limb may be depressed and absent.

This disorder often occurs among patients over 50 years with poorly controlled diabetes. It is more common among men than women.

Its course is generally monophasic with improvement over many months; however, some residual deficits often remain. As a complication, due to the loss of sensation of the diabetic foot, there is an increased risk of injuring it. Small chronic infections can progress to ulceration requiring amputation.

As regards diabetic angiopathy, see the special Chapter of Diabetes mellitus.

The cause of DNs is a genetic predisposition combined with infections caused by *Mycoplasma fermentans, HTLV, Coxsackie B4 virus*, other *Coxsackie viruses*, *ECHO viruses*, *Herpes viruses* and *Nanobacteria*.

Diagnosis: symptomatically, by capillar resistance examinations.

Treatment: by tight glucose control and symptomatically, by reducing the pain caused by hypoxia and other symptoms (f.i. by administering tricyclic antidepressants, serotonin

reuptake inhibitors and antiepileptic drugs in order to reduce the pain. Glucose control is not able to solve the problem of diabetic angiopathy and neuropathy, as these develop due to the effects of viral, mycoplasmal and nanobacterial infections.

RFR method detects and may eliminate the special pathogens of diabetes:

Resonances present in case of Diabetes Mellitus type1 are: 307-308 *(Coxsackie B4)*; **361-365** *(Coxsackie B4)*; **370-374** *(HTLV-1 group)*; **442-451** *(Mycoplasma fermentans)*; **493-495** *(Mycoplasma fermentans)* **kHz**

Resonances present in case of Diabetes Mellitus type2 (receptor) are: 370-374 *(HTLV-1 group)*; **420-426** (caused probably by *Coxsackie virus B4*); **442-451** *(Mycoplasma fermentans)*; **493-495** *(Mycoplasma fermentans)* and **534-544** (caused probably by *Coxsackie virus B4*) **kHz**

Resonances present in case of Mixed diabetes are those of Diabetes type1 and 2.

The first step to be taken is to eliminate Mycoplasma species.

Other most frequent resonancies are: 324-325, 375-381, 560-568 *(Nanobacteria)*; **286-302** *(Coxsackie viruses A9, B3* and *4, ECHO viruses 2-14, 18-19, 22-24)*; and **291-293** *(HSV1)* **kHz**

10.40. Polyneuropathy

Polyneuropathy is a neurological disorder occurings when many peripheral nerves malfunction throughout the body simultaneously. It may be acute, appearing without warning, or chronic to develop gradually over a longer period of time. Many polyneuropathies show both motoric and sensory dysfunctions while some display autonomic dysfunctions. These disorders are often symmetric, frequently involving distal extremities. Polyneuropathy has many a different cause. An infection can cause polyneuropathy sometimes due to *toxins produced by infective agents* (in case of diphtheria, borreliosis, mycoplasmosis, HIV, hepatitis infections, Colorado tick fever, leprosy, syphilis). Certain *toxins* (like alcohol and other chemicalies) and *autoimmune reactions* (in case of f.i. Guillain-Barre syndrome, polyarteriitis nodosa, rheumatoid arthritis, Sjögren syndrome and SLE) can also cause polyneuropathy. Cancer can cause polyneuropathy by directly invading or compressing the nerves or by producing toxic substances.

Borreliosis is very often associated with polyneuropathy. Approximately 5-10% of untreated patient with Lyme disease show signs of cranial neuropathies, while up to 60% of patients with early neuroborreliosis will develop cranial neuritis usually starting 3 weeks after having been infected. Seventh nerve palsy is the most common by far. Bilateral facial palsy can be seen in 35% of the patients and is a unique characteristic that is useful for distinguishing it from idiopathic Bell palsy and other disorders. The are typically even other associated neurological symptoms present as well, depending on the nerve affected visual or auditory disturbances, facial paresthesia and/or vertigo. Multiple cranial neuropathies can occur, too.

(Aseptic, viral meningitis is relatively common in among 15% of untreated patients bitten by the Ixodes tick, as well as in among 30% of Lyme disease patients. Symptoms usually occur 2-10 weeks following the infection. Headache, pain and stiffness of the neck and photophobia typically indicate a meningeal irritation. Meningitis may be accompanied by cranial or peripheral radiculoneuropathy).

The symptoms can progress gradually or in a relapsing-remitting pattern, with partial improvement following the attacks. Although the clinical sings of inflammatory radiculoneuropathy in case of borreliosis is often indistinguishable from that of spinal-root compression, the involvement of the thorax of multiple dermatomes and the lack of a quickly developing injury can help to establish the diagnosis.

As for the peripheral neuropathy, patients usually report intermittent paresthesias. Its most frequently experienced sign is a decreased vibratory sensation of the distal lower extremities. A "stocking-glove" distribution of epicritic sensory deficits is also common.
The evaluation and classification of polyneuropathies has to begin with the patient's history and physical examination in order to document the pattern of the disease process (whether they affect arms, legs, or are distal, proximal, symmetric), at what time they started, how long they lasted, whether they fluctuate, and what kind of deficits and pain are being involved. In case of often occurring pain it is to be determined where and how long the pain has been felt. Patients suffering from late axonal neuropathy can tell about intermittent distal limb paresthesias months to years after having been infected. It differs from the neuropathy of early Lyme disease the symptoms being less severe. Acrodermatitis chronica atrophicans-associated neuropathy is common in Europe and can be characterized by neuropathic pain, paresthesia and muscle cramps.
In some cases, the damages of the nerves controlling the blood vessels, intestines and other organs result in abnormal blood pressure, digestion problems and in the loss of other basic body processes. Peripheral neuropathy can involve the damage of a single nerve or a nerve-group (mononeuropathy) or can affect multiple nerves (polyneuropathy).
A combined infection caused by *Borrelia Burgdorferi sensu lato* and *Mycoplasma* species can cause spreading immune vascular damages resulting thus in an *autoimmune polyneuropathy.*
Diabetes can also be the cause of mononeuropathy or multiple mononeuropathies that lead typically to the damage of vision and the weakness of thigh muscles. The most common form of diabetic neuropathy is the distal polyneuropathy sensation in hands and feet.
Diagnosis: symptomatically and by electromyography, muscle and nerve biopsy, serum creatine kinase testing, antibody testing, and by specific tests applied for specific disorders associated with polyneuropathies.
Differential diagnosis of its causes: Vitamin deficiency, cancer, toxins, infections (Guillain-Barré Syndrome), liver disease, diabetes mellitus, amyloidosis, certain hereditary and idiopathic polyneuropathies (Charcot-Marie-Tooth disease, Dejerine Sottas syndrome, Refsum's disease, Morvan's syndrome, Guillain-Barré syndrome) motor neuron disorders, motor neuropathies, kidney failure, porphyria (some types), spinal muscular atrophy, catecholamine disorders and alcohol.
Treatment: depending on its cause. By administering antibiotics, analgetics, anticonvulsants, tricyclic antidepressants, etc.
RFR method: detects and eliminates the infective agents. Combine the RFR method with antibiotic therapy. In case of autoimmune polyneuropathies the immune cascade should be cut off by administering corticosteroids.
As to the resonant frequencies: see the special chapters of microorganisms.

10.41. Tardive Dyskinesia Forms

Dyskinesia is a type of movement disorders which can be sorted into bradykinesia forms and hyperkinesia forms. Bradykinesias are characterized by abnormal slowness (f.i. rigidity), difficulty in initiating and terminating actions, and by the masked facial expression of patients with Parkinson disease. Hyperkinesias are purposeless movements, including akathisia, chorea, dystonia, myoclonus, stereotypy, tic, tremor and abnormal pyramidal reactions, such as paralysis, paresis, hyperreflexia and spasticity.
Tardive Dyskinesia Forms (TDs are involuntary movements of the tongue, lips, face, trunk and extremities occuring in patients treated with long-term dopaminergic antagonist medicaments. TDs differ from acute movement disorders occuring usually in the same group of patients. Acute movement disorders occuring as manifestations of the effects of neuroleptics and other dopamine antagonists include akathisia, acute dystonia and other hyperkinetic dyskinesias. The acute effects of dopamine antagonists include also

parkinsonian syndromes manifested in bradykinesia, rigidity and pill rolling tremor. Acute movement disorders resulting from being exposed to dopamine antagonists are commonly termed as extrapyramidal syndromes (EPSs). TDs develop most commonly among patients suffering from schizophrenia, schizoaffective disorders, bipolar disorders treated with antipsychotic drugs for long periods, though TDs can occasionally occur among other patients as well.
The pyramidal system, controlling voluntary movements, has precise anatomic pathways from the cortex to the muscles. The voluntary movements occuring via the pyramidal system are visible. By contrast, the extrapyramidal motor activities result in not noticeable automatic movements and in static, postural movement activities. The extrapyramidal system includes theorized connections within the basal ganglia, the striatopallidonigral system and other structures of the CNS contributing to the regulation of movements, including the related brainstem nuclei and the cerebellum.
The corticospinal lesions above the pyramidal decussation result typically in the paralysis of volitional movements of the contralateral half of the body and in a fixed posture with the flexion of the upper extremity and in the extension of the lower extremity. The unilateral lesions of the upper pons and the midbrain often result in the extension of the ipsilateral arm and leg.
Genetical predisposition:
Abnormalities of the dopamine receptor D2 (DRD2), the dopamine receptor D3 (DRD3), the dopamine transporter (DAT) and the manganese superoxide dismutase (MnSOD) genes are supposed to play a role in the development of TDs.
Diagnosing the acute and chronic dyskinesias is difficult without knowing the patient's past history. A precise documentation of the patient's complete movement and medication history can facilitate the accurate delineation of movement disorders. Thus, a full neurologic and pharmacologic history can provide the basis to distinguish an idiopathic Tourette disorder from acute medication-induced tardive tics. The former illness is characterized by rigidity, dystonia, choreoathetosis, spasticity, foot deformity and intellectual deterioration, is associated with excessive iron deposition in the basal ganglia that can be observed making MRI and PETscan.
Treatment: The primary prevention of TD can occur by using the lowest effective dose of neuroleptic medicaments for the shortest period of time. After diagnosing TD, the reducing or discontinuing the therapy with the causative agent is advisable, if possible. The risk of a permanent movement disorder must be weighed against the risks of exacerbating psychosis. In addition, TD may initially worsen after the discontinuation of the neuroleptic therapy.
Atypical neuroleptics, (f.i. clozapine) variably bound to dopaminergic, serotonergic, alpha-adrenergic, histaminic and muscarinic receptors can control psychosis and reduce the risk of TD as well.
RFR method: detects and can eliminate all pathogen microorganisms.
The most frequent resonances found in case of this disorder are: 346, 346-350, 353-359, 416, 442-451, 532, 578 kHz
The role of these microorganisms concerning the development of TD is unknown as yet, so that it is necessary to make further examinations.

10.42. Foix-Alajouanine Syndrome

Foix-Alajouanine syndrome (FAS) is the term of a vascular myelopathy, when an arteriovenous malformation of the spinal cord, predominantly affecting the lower thoracic and/or lumbosacral levels, is present. Cervical cord involvement is rare. Findings include necrosis of the affected cord regions. Grey matter (as compared to white matter) structures are more severely involved. Masses of enlarged, tortuous and thick-walled subarachnoid veins are observed overlying the surface of the cord. Smaller blood vessels with thickened

fibrotic walls are also present in the affected spinal cord segments. The enlarged, abnormal veins are associated with dural arteriovenous (AV) shunts or fistulas, usually intradurally though rarely extradurally as well. These AV shunts are associated with the reflux of arterial blood into the venous drainage of the cord. This fact results in an increased venous pressure in the affected regions of the spinal cord, leading often to ischemic injuries.

Symptoms begin after a brief exertion as a heavy feeling in the legs that generally improves after resting. Early observable bowel, bladder and sexual function problems are common. Over months symptoms gradually worsen, the patient may have difficulty standing for a long time. Patients suffer from increasing unilateral and/or bilateral weakness, dysesthesia and numbness or tingling in the lower extremities, which may be symmetric or asymmetric. Complaints of nonradiating lower back pain in the lumbosacral or coccygeal regions are common. A pathological examination reveals disseminated nerve cell death in the spinal cord and abnormally dilated and tortuous vessels situated on the surface of the spinal cord. The rectal sphincter tone is frequently diminished.

The onset of the illness in middle aged people suggests that the syndrome is acquired, in contrast to other arteriovenous malformations, which are assumed to be congenital abnormalities. The specific location in the spinal cord can not be easily explained. FAS is caused by a combined chronic infection with *Herpes viruses* or other neurotrop viruses such as *Coxsackie viruses*, with *Nanobacterium sanguineum* (causing blood vessel damages) and with immunesuppressive microorganisms such as *mycoplasma, EBV, CMV* and/or *HTLV* and, most frequently, *HIV.*

Diagnosis: by CT, MRI, myelographic studies, MR angiogram, spinal catheter angiography, neurophysiologic studies, electromyography, nerve conduction studies, etc.

Differential diagnosis: by distinguishing it from amyotrophic lateral sclerosis, ankylosing spondilitis, multiple sclerosis, polyradiculopathy, spinal cord infarction, spinal epidural abscess, syringomyelia, vitamin B-12 associated neurological disease, primary or metastatic neoplastic disease and spinal artery thrombosis.

Treatment: by surgery, by administering corticosteroids

RFR method: detects all microorganisms present, there being typically many different kinds of microorganisms to be found in case of FAS.

The most frequent resonances of this syndrome are: 287-294, 344-345, 338-339, 353-361, 378-380, 402-411, 424-426, 442-451, 479-481, 486,-489, 493-495, 530-536 kHz

11. DISORDERS ASSOCIATED WITH INFECTIONS OF THE RESPIRATORY TRACT

These disorders of the respiratory tract include a number of various viral, bacterial, mycoplasmal, chlamydial and fungal infections. Pneumonia and bronchitis begin usually by inhaling the causative microorganisms via the airways into the lungs, though, the infection is sometimes carried by the bloodstream to the lungs, or, in other cases the pathogens migrate to the lungs directly from the place of a nearby infection. In such cases systemic infections and also lung abscesses may develop. Lung abscess, a pus-filled cavity in the lung surrounded by inflamed tissue, is usually caused by a certain type of severe infections and in case of a weakened immune response.

11.1. The Common Cold

The common cold is caused by viral infections of the lining of the noise, the sinuses, the throat, and the large airways. Many different viruses can cause common colds. Picorna viruses, f.i. *rhinoviruses*, cause its colds mostly in spring, summer and autumn. *Influenza viruses and Respiratory Syncytial Viruses*, appearing regularly in the late autumn and winter, cause a variety of illnesses besides colds, too, but these resemble the rhinoviral common cold. *Human Corona Viruses* are difficult to detect, and the concerning epidemiological data are very rare. The strains HCoV 229E and HCoV OC43 are generally known as viruses responsible for the *common cold syndrome.*

The symptoms of the common cold are the following: profuse watery and later on sometimes even mucopurulent nasal discharge, a sore throat, a moderate cough and mild constitutional symptoms. The infection ceases usually after a short duration (3-5 days).

Diagnosis is based on isolation of the infective viruses in organ cultures, in human embryo kidney cultures, or by detecting an increase in the amount of specific antibodies. The neutralizing antibody test is more sensitive and precise than the complement fixation test, the risen antibody-titer persists for a longer time. Both tests, however, provide an adequate diagnostic information. There is evidence that *corona viruses* cause bronchiolitis and pneumonia among children and adults. Fever usually does not develop, but, sometimes a slight fever accompanies the onset of symptoms.

Complications may prolong the symptoms. A bacterial reinfection of the ears, the sinuses, the windpipe as well as the tracheobronchial region may follow the cold and require treatment with antibiotics.

Treatment: symptomatically; in the case of bacterial reinfection by administering antibiotics.

RFR method: detects and eliminates the viruses.

As to the frequencies see Chapter 5.

11.2. Scarlet Fever

Scarlet fever is a hemolytic streptococcal infection. The incubation period is from 3-5 days. The symptoms include feverishness, chilliness, headache, malaise, exanthema, loss of appetite and a sore throat. In case of children nausea and vomiting occur especially often. Some patients might complain of diarrhea. Chilliness is a constant symptom, but true rigors are rare.

The soreness of the throat is aggravated when swallowing and even the turning of the head is accompanied by pain.

About half of the patients have mild symptoms at the lower part of the respiratory tract, including cough and hoarseness. The cough is not productive and is rarely associated with chest pain. Loss of voice due to laryngitis does never occur. Earache is common and may last from a few hours to several days. Occasionally, epistaxis is observed, too.
In some cases of high fever the diffuse blush of the skin is more pronounced. It spreads rapidly over the abdomen to the upper and lower extremities. The face appears flushed, a pallor around the mouth is prominent. Itching may be mildly felt.
Diffuse redness of the mucous membranes of the posterior pharynx, the tonsils, and the soft palate are invariably experienced.
The uvula as well as the tonsils and the pharynx are frequently edematous. In case of sinusitis and rhinitis there can a thick, mucopulurent nasal discharge, tinged with blood be observed. Marked adenopathy is frequently followed by suppuration.
Lymphoid hyperplasia and edema lend the posterior pharynx a cobblestone appearance. The cervical and the submandibular lymph nodes get enlarged to a great degree.
Diagnosis: symptomatically, and by bacterial culturing.
Differential diagnosis: by distinguishing it from nonbacterial exudative tonsillitis and pharyngitis, infectious mononucleosis, herpes simplex pharyngitis, influenza viral infection, rubella, rubeola, diphtheria and Vincent's angina.
Treatment: by administering antibiotics, analgesics and antipyretics.
RFR method: use together with the antibiotic treatment!
The most frequent resonances are: 340, 353-355, 360-375, 402-405, 445-455 kHz
This list isn't yet complete, as the resonance frequencies of streptococcus quickly change.

11.3. Moraxella Catarrhalis Infections

Moraxella catarrhalis, formerly Micrococcus catarrhalis, Neisseria catarrhalis, and Branhamella catarrhalis is a gram-negative, arobic, oxidase-positive diplococcus (see also Chapter 6.8.3.), facultatively pathogen concerning the upper respiratory tract.
Though the commensal state of Moraxella catarrhalis in the nasopharynx is still accepted, the bacterium can be a common cause of otitis media, sinusitis and, occasionally, the cause of laryngitis, bronchitis or pneumonia in case of children and adults suffering from a chronic lung disease. It can also cause bacteremia and meningitis among immunocompromised patients. Bacteremia can be complicated with local infections such as osteomyelitis or septic arthritis as well, and it can be also associated with nosocomial infections. Its transmission is believed to be due to direct contact with contaminated secretions by droplets.
The endotoxin of M catarrhalis, a lipopolysaccharide similar to those found in Neisseria species, may play a role in the process of these diseases. Some strains of M catarrhalis have pili or fimbriae, which helps to adhere to the respiratory epithelium. A produced protein of certain strains assures complement resistance.
Antibodies to M catarrhalis appear to be age-dependent, the titer of specific immunoglobulin G (IgG) increases gradually among children.
M catarrhalis is the third most common cause of otitis media and sinusitis as concerns children (next to Streptococcus pneumoniae and Haemophilus influenzae). Otitis media: **Symptoms of otitis media** caused by M. catarrhalis include otalgia, fever and loss of hearing. loss. M catarrhalis can sometimes be isolated in the middle ear exudates of children suffering from otitis media.
Symptoms of sinusitis caused by M. catarrhalis concerning young children includes headache, fever, cough at night, persistant nasal discharge lasting for more than 2 weeks and pain in the maxillary or frontal area. This sinusitis goes often with infections caused either by Streptococcus pneumoniae or by Haemophilus influenzae.
The exacerbations of adults who suffer from chronic pulmonary diseases f.i. COPD, pneumoconiosis, asthma bronchiale, malignancies, etc. and show symptoms characteristic

of bronchitis or pneumonia, might be caused by a M. catarrhalis infection. The lower respiratory tract infections of smokers are often caused by M catarrhalis
As to children, the lower respiratory tract infections caused by M. catarrhalis are usually associated with the history of recently got RSV or CMV infection, as well as with congenital dysplasias, malformations or other immunosuppressed conditions.
Nosocomial infections occur mostly in pulmonary units or pediatric intensive care units. Patients suffering from immunodeficiency of different etiology or from chronic respiratory diseases are predisposed to M. catarrhalis *bacteremia.*
Diagnosis: by bacterium identification and symptomatically.
Treatment: By administering amoxicillin-clavulanate, second-generation and third-generation cephalosporins, trimethoprim-sulfamethoxazole. Alternatively, azithromycin, clarithromycin, or dirithromycin can also be effective. Nearly all Moraxella catarrhalis strains are beta-lactamase producers. The dosage depends on the severity of the infection and the susceptibility of the bacteria.
RFR method: detects and may eliminate Moraxella!
The most frequent resonance frequencies of Moraxella catarrhalis are: 294-299, 350-352, 392-400, 512-520 kHz
The most frequent resonance frequencies of Neisseria catarrhalis are: 315-319, 500-503 kHz
The most frequent resonance frequencies of Branhamella catarrhalis are: 392-398 kHz

11.4. Croup

Croup, a characteristic respiratory syndrome concerning infants and children under 6 years, is usually caused by contagious viral infections of the upper and the lower airways provoking difficulties in the breathing, especially by inhalation. A number of different viruses can be the causative agents of this illness. In the autumn, *parainfluenza viruses* are the most likely source of the disease. Less commonly, croup can also be caused by *measles virus* or other viruses, f.i. *Respiratory Syncytial Virus* or *influenza viruses, adenoviruses,* ECHO viruses, moreover, *mycoplasma* as well. In case of croup caused by an influenza virus the symptoms may be particularly severe occuring among children between ages 3 months to 5 years. The disease is usually spread by breathing in airborne droplets containing the virus or by having contact with infected objects or areas.
Symptoms: usually begin with cold-like symptoms. Then, the laryngotracheobronchial airways getting narrowish caused by the progressive swelling (edema) of their mucosal lining, the breathing in becomes difficult. This difficult inhalation together with a barking cough and hoarseness are the commonsigns of the illness occuring mostly at night. This impaired breathing may awaken the child from its sleep. Spasmodic croup may be triggered by allergies, but starts usually with a viral infection. Increasing or continued difficulty in breathing to progressive stridor, rapid heart rate, fatigue, changes in mental state, bluish skin discoloration (such as cyanosis), and/or dehydration indicate that the child should be hospitalized, Oxygen should be given if the blood level of oxygen is low.
Treatment: Symptomatically, by administering corticosteroids as well. By administering bronchodilatatory drugs, f.i. Epinephrine to be inhaled as a mist through a nebulizer.
Antibiotics are to be used only in rare cases, when a bacterial infection is also present.
RFR method: detects and eliminates the virus and bacteria.
The frequency resonances of the Croup are virus-dependent, see these resonances in Chapter 5.

11.5. Bronchitis

Bronchitis, the inflammation of the bronchi, is usually caused by infections, but it can also be present in case of chronically ill people suffering from a heart disease and/or a lung disease, as well as among elderly people or by smokers. Bronchitis does occur most often in winter, caused by viruses, bacteria, as well as by the bacteria-like species of Mycoplasma and Chlamydia. Smokers and people having chronic lung or airway diseases hindering them in the clearing of the inhaled particles from the bronchi, may have repeated attacks.
Chronic sinusitis, bronchiectasis, lower airway allergies and even tonsillitis may give rise to recurring infections of the bronchi. Irritative bronchitis may be caused by various kind of dust components, chemicals, organic solvents as well as by smoking.
In case of chronic gastroenteritis enteral bacteria can often cause bronchial complications.
Symptoms: Infectious bronchitis often begins with the signs of a common cold, f.i. runny nose, tiredness, chills, a dry coughing, yellow and green sputum, pains of the back and the muscles, a slight fever and a sore throat, too.
Treatment: by administering NSAID, antibiotics, f.i. docycycline, ampicillin, amoxacillin, trimethoprim-sulfamethoxazole, macrolids etc. Of course, antibiotics does not help in case of viral infections.
RFR method: detects and eliminates the infective agents f.i.:
The most often found viruses are as follows:
The resonances of Respiratory Syncytial Virus are: 284, 342, 364, 377-385 kHz
The resonances of Adenovirus are: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz
The resonances of Influenza A and B are: 284, 307-324 kHz (see also Chapter 5.1.)
The resonances of Cytomegalovirus are: 304-307, 348-351, 405-410, 512, 534, 548 kHz
The resonances of Herpes Simplex Virus are: 290-294, 344-346, 352-365, 413, 425 kHz (see also Chapter 5.2.4.1.)
The resonances of Cold viruses are: 395-396 kHz (see also Chapter 5.1.2.)
The most often found bacteria are as follows:
The resonances of Hemophilus influenzae are: 335-338, 374-375, 452, 482-494, 564 kHz
The resonances of Enterobacter aerogenes are: 351, 373-375, 418, 477, 487, 564 kHz
The resonances of Klebsiella pneumoniae are: 372, 381, 390-392, 397-406, 414-423, 429 kHz
The resonances of Staphylococcus aureus and other related species are: 281, 324-329, 331-332, 345, 372, 376-384, 397, 402, 434, 445, 462, 482, 491, 537, 557, 562, 567 kHz
The resonances of Streptococcus pneumoniae are: 274-277, 288, 310-321, 330, 337, 340, 345, 351-353, 366-370, 382-387, 391, 397-410, 416, 426-434, 442, 450, 468-470, 478, 498, 508, 516, 542, 548 kHz
The resonances of Pseudomonas aeruginosa are: 323-325, 330-340, 351-358, 372, 380, 388-397, 401, 414, 438, 447-448, 496, 579 kHz
The resonances of Mycoplasma are: 320-324, 337-352, 397, 499 kHz
The resonances of Chlamydia are: 283, 317-319, 375-386, 429, 440-444, 480-486 kHz

11.5.1. Swyer-James Syndrome

Swyer-James syndrome is a form of postinfectious obliterative bronchiolitis affecting children, in which case the involved lung or a part of the affected lung does not grow normally and is slightly smaller than the non affected lung. This type of hypoplasia occurs as a result of diminished vascularity and of the arrest of the progressive growth and alveolarization of the affected part of the lung. Swyer-James syndrome develops as a complication of simultaneously attacking viral, mycoplasmal and bacterial infections, which are followed by a chronic lung disease, characterized by bronchiolar abnormalities, air trapping and abnormal lung dynamics when inhaling and by forced expiration. In this

illness the affected pulmonary parenchymal pattern is similar to that of obliterative bronchiolitis. Swyer-James syndrome patients develop a small lung in the involved part of the lung as their lung could not grow normally and the patients show a compensatory overexpansion of the contralateral lung. The peripheral bronchioli become "pruned" secondary to an obliterative bronchiolitis. In case of this syndrome a mosaic pattern of hyperlucency can be observed in the affected areas of the lung, small vessels and vascular occlusions as well are present in these abnormal areas.
Typically, children with Swyer-James syndrome suffered from severe pneumonia earlier in their life. Pathogens causing these severe infections include Respiratory Syncytial Virus, different Influenza viruses, antibiotics sensitive or resistant Mycoplasma pneumoniae, M. pulmonis (in Canada), M. fermentans, Candida albicans and several Staphylococci, MRSA, Streptococci and/or Diplococci. A mycoplasma infection causes usually a general immunodepression. In case of repeated and/or prolonged antibiotic therapies a pulmonary candidiasis can develop.
Diagnosis: by x-ray and by other imaging techniques finding the characteristic signs of Swyer-James syndrome appearing a few months to a few years after the causative infection.
Treatment: by administering antibiotics. (Many a bacterium and mycoplasma population can be antibiotics resistant.)
RFR method detects and eliminates viruses, antibiotics resistant bacteria and mycoplasma.
The most frequent resonances are: 305-307, 321-324, 337-350, 364, 376-381, 394-399, 442-451, 478-479, 493-495, 568, 576-586 kHz

11.6. Pneumonia

Pneumonia is the infection of the lungs, involving the small air alveoli and the tissues around them. One certain microorganism, or a combination of infective agents is the most often cause of this disease. An increasing number of the bacteria causing pneumonia are developing to be resistant to antibiotics.
Antibiotic resistance is a serious problem concerning the therapy which can be solved by using RFR method.
The symptoms are as follows: a productive cough, sputum, chest pain, fever, chills and shortness of breath. Pneumonia usually produces distinctive changes in the way the sounds are transmitted, which can be heard with stethoscope.
The diagnosis of pneumonia will be confirmed by auscultation, by chest x-ray examination, which can help to determine, the locus of the infection f.i. interstitial, bronchopneumonia, diffuse or focal process.
The RFR method detects and eliminates the microorganism causing pneumonia and can eliminate the antibiotic resistant bacteria species, too.

11.6.1. Klebsiella Pneumonia

Klebsiella pneumonia is a rare, severe lung infection affecting mostly diabetic patients and alcoholists. Community-acquired Klebsiella (Friedländer) pneumonia is an illness of debilitated middle-aged and older men with alcoholism.
Symptoms: The person may cough up dark brown, yellow-green or dark red sputum. There develop abscesses in the lungs and collections of pus in pleura, named empyema. Other serious diseases, including sinusitis, enteritis, prostatitis renal abscesses may also be present. The locus of the most frequent pulmonary lesion is usually in the right upper lobe progressing often rapidly, and, if untreated, the infection may spread from lobe to lobe. Cyanosis and dyspnea develop rapidly, jaundice, vomiting, diarrhea may also be present. The physical findings consist primarily of signs of consolidation unless a pleural effusion or a necrotizing pneumonitis with rapid cavitations has intervened. Lung abscesses and

empyema are much more frequent than in pneumococcal pneumonia and are related to the destructive capabilities of this microorganism. If this infection progress, in an indolent fashion, can laed to a chronic necrotizing pneumonitis resembling tuberculosis.
Diagnosis: by physical examination, x-ray, bacterium culture.
Treatment: if treated early enough, Klebsiella pneumonia can be cured with intravenous antibiotics, usually by administering cephalosporins or quinolones.
RFR method: detects and eliminates the bacterium! This RFR method is usefull and effective also in case of antibiotic resistance.
Its resonant frequencies are: 381, 391-392, 395-405, 414-430 kHz

11.6.2. Legionnaire's Disease Pneumonia

Legionnaire's disease, caused by the bacteria *Legionella pneumophila* and other species of Legionella. This bacteria live in water, their outbreaks occur if the bacteria spread through the air conditioning systems. This disease causes relatively minor symptoms but can be even life threatening.
Symptoms include fatigue, fever, headache, muscle ache, a dry cough followed by sputum.
Serously infected people get sort of breath and generally diarrhea.
Diagnosis: by physical examinations, x-ray, and bacterium culture.
Treatment: most people treated with erythromycin usually get better, but their recovery might take a long time.
Its resonant frequencies are: 350-355, 369-372, 402, 449-459 kHz

11.6.3. Haemophilus Influenzae Pneumonia

The genus Haemophilus consists of nonmotile, gram-negative rods i.e. coccobacilli requiring specific growth colonial factors X and V for their multiplication. *H. influenzae, H. pertusis, H. ducreyi, H. aphrophilus,* the *Koch-Weeks bacillus (H. Aegyptius)*, and *Moraxella lacunata* are important as regards the causing of human diseases. Two other species can be found in the pharyngx of healthy individuals, and, may rarely cause pharyngitis, endocarditis or pneumonia. They invade mostly the respiratory tract, the bacillus responsible for the bulk of infections is *H. influenzae*. Haemophilus influenzae is a bacterium, and not a virus, which causes the flu. Its b type strains are the most virulent, causing serious diseases, including meningitis, epiglottitis, laryngotracheitis, sinusitis, otitis media, conjunctivitis, osteomyelitis, bronchitis, meningitis and pneumonia, usually among children.
Symptoms: of the infection include sneezing and a runny nose followed by typical pneumonia symptoms, such as fever, cough producing sputum, shortness of breath, and general malaise.
Prophylaxis: by giving Haemophilus influenzae type B vaccines.
Diagnosis: by physical examination, x-ray, bacterium culture.
Treatment: by administering antibiotics f.i. ceftriaxone, erythromycin, doxycyclin, Chloramphenicol, etc.
RFR method: detects and may eliminate Haemophilus influenzae!
Its resonant frequencies are: 282, 335-338, 374-382, 452, 482-494, 564 kHz

11.6.4. Pneumonia Caused by Pseudomonas Species

Pseudomonas aeruginosa, Pseudomonas fluorescens, Pseudomonas putida, as well as other, oppurtunistic pathogen Gram negative bacteria, sorted formerly to this Pseudomonadacea family, f.i. *Stenotrophomonas maltophilia, Burkholderia cepacia,*

Comamonas testosteroni can cause Gram negative infections often acquired in hospitals or a nursing home, mostly among patients in immunocompromised state.
Symptoms: These infections may rapidly destroy the lung tissues, quickly tending to be serious. Fever, coughing, and shortness of breath are common. The coughed-up sputum may be thick, red-coloured, with consistency of currant jelly. The pulmonary infection is often associated with microabscesses. Other serious diseases, including bronchitis, sinusitis, gastroenteral and urogenital inflammations, f.i. pyelonephritis, and skin infections can also be caused by these pathogens.
Diagnosis: by physical examination, x-ray, bacterium cultures in case of patients with damaged immune state.
Treatment: by administering aminoglycosid antibiotics f.i. tobramycin, neomycin.
Its resonant frequencies are: 323-326, 330-336, 351-362, 372, 380, 389-397, 401, 414, 438, 446-447, 496-512, 579 kHz

11.6.5. Chlamydial Pneumonia and Psittacosis

The symptoms of chlamydial pneumonia are similar to those of mycoplasmal pneumonia. The family Chlamydiaceae has three human pathogenic species, *Chlamydia trachomatis*, *Chlamydophila psittaci* and *Chlamydophila pneumoniae*, with its AR39, CWL029 and J138 strains. The members belonging to this family are obligate, intracellular microorganisms; they are not viruses, being more closely related to bacteria, as they have cell walls with chemical and metabolic properties similar to bacteria and are affected by broad spectrum antibiotics.
Psittacosis, also known as parrot disease, parrot fever or a kind of ornithosis, is a rare form of pneumonia caused by *Chlamydophila psittaci*, which microorganism infects birds, f.i. parrots, parakeets, lovebirds, pigeons, finches chickens, and turkeys. This infection is usually transmitted by the bite of an infected bird though sometimes, it can happen by cough droplets from person to person. This mostly is a disease of people working in pet shops or on farms.
Diagnosis: by identifying the typical cytoplasmic inclusion bodies in Giemsa-stained or fluorescein antibody-stained smears, by x-ray.
Treatment: by administering doxycyclin for a long time.
The resonant frequencies of the Chlamydiaceae are: 283, 317-319, 375-386, 429, 444, 480-486 kHz
The resonant frequencies of psittacosis are: 298, 311, 335-338, 439, 476 kHz

11.6.6. Anthrax Pneumonia

Anthrax is a local and systematic disease caused by the bacterium *Bacillus anthracis*, infecting the skin, the lungs, and the gastrointestinal tract. Anthrax is a highly contagious and potentially fatal disease. It usually spreads to people from animals, especially from cows, goats and sheep. The dormant spores of the bacteria can live in the soil and in animal products for decades. Though the infection attacks people usually through their skin, but can also happen by eating contaminated meat or by inhaling spores or bacteria. The lymph nodes of the affected area may get swollen, the person may feel ill, sometimes experiencing muscle aches, headache, fever, nausea and vomiting. The spores multiply in the lymph nodes near the lung.
The **symptoms** are vague and like to those of influenza. Brain infection, f.i. meningoencephalitis may also come about. The infection can be fatal by spreading into the bloodstream.
Diagnosis: by bacterium culturing and x-ray.
Treatment: by administering doxycyclin
Its resonant frequencies are: 324, 349-350, 356-372, 360-366, 390-400, 422-510 kHz
The resonant frequencies of the spores of B. anthracis are: 386-392 kHz

11.6.7. Staphylococcal Pneumonia

This type of pneumonia tends to develop in tcase of very young, and very old persons, as well as among people being already debilitated by other illnesses. In case of older children and adults the primary staphylococcal pneumonia may be secondary to influenza infection or measles infection. In case of healthy adults, staphylococcal pneumonia is generally preceded by an influenza-like respiratory infection.

Symptoms: the Staphylococcus infection can cause typical pneumonia symptoms, the chills and the fever are more persistent in this infection than in Pneumococcal pneumonia. The onset of the illness is abrupt, with chills, high fever, progressive dyspnea, cyanosis, cough, and pleural pain. An early peripheral vascular collapse often occurs, the patients seems to be sicker than his physical findings would suggest. In the early phase the sputum is not characteristic, but may be bloody or frankly purulent. Staphylococcus may cause even abscesses in the lungs, moreover, serious diseases including enteritis, meningitis, arthritis, endocarditis etc.

Diagnosis: by x-ray, physical examination, bacterium culture.

Differential diagnosis: just like as in case of pneumonia caused by other agents.

Treatment: infections caused by penicillinase resistant staphylococci need special treatment, but some staphylococci are becoming resistant to these special drugs, too. Vancomycin can be of great value in case of multidrug resistance.

The RFR method is effective.

The resonant frequencies of Staphylococcus aureus are: 376-384 KHz

As to the other frequencies of Staphylococci see its special Chapter.

Staphylococcus aureus has a lot of plasmid with known genetic codes.

Their resonant frequencies are the following: 324-331, 345, 372-382, 397, 402, 434, 445, 462, 482, 491, 537, 557-567 kHz

11.6.8. Pneumococcal Pneumonia

Streptococcus pneumoniae (i.e. the pneumococcus) is the most common bacterial cause of pneumonia. The diseases produced by different pneomococcal serotypes show little variations in severity or in clinical manifestations. The prognosis of the disease in case of type 3 pneumococcal pneumonia is usually ominous, probably because type 3 infections occur frequently in case of aged people as well as in case of patients suffering from other debilitating disease, too, f.i. diabetes mellitus and congestive heart failure. The lesions are usually segmental or lobar in distribution concerning adults and bronchopneumonia, characterized by patchy involvements, in case of children and elderly patients. Pneumococcus has 80-85 known types. If infected with one of these types, there develops only a partial immunity to reinfection with the same type, but no immunity to the other types. Pneumococcus pneumonia usually occurs after a viral infection attacking and damaging the respiratory tract (f.i. adenovirus 392-397 kHz), and also infects this area. *Streptococcus pneumoniae* can cause several other infections, too f.i. otitis media, tonsillitis, ulcus serpens corneae, bronchitis, etc.

Symptoms: are those, characteristic to pneumonia. Thee illness can progress and complications in the lungs can occur, f.i. atelectasis, abscess, pleural effusion, empyema. Other extrapulmonal complications include metastatic brain abscess, pericarditis, endocarditis, arthritis, paralytic ileus, liver damage and jaundice as well.

Diagnosis: by x-ray, blood culture, by using Neufeld-Quelling reaction (i.e. capsular precipitin reactions) technique.

Differential diagnosis: by distinguishing it from other pneumoniae caused by Staphylococcus, Hemolytic Streptococcus, Klebsiella, (f.i. Friedlander's pneumonia), Francisella tularensis, Haemophilus influenzae, Mycoplasma, Chlamydia, viruses,

Psittacosis, Mycobacterium tuberculosis, as well as pneumoniae caused by mycotic infection such as histoplasmosis, coccidioidomycosis.
Treatment: the Pneumococcal pneumonia may be treated with several antibiotics, including penicillin. In case of allergy to penicillin by administering erythromycin etc.
RFR method: together with antibiotic treatment. Pneumococcus should be treated with RFR method continuously, as long as the bacteria is alive.
The resonant frequencies of Pneumococcus are: 310-321, 330, 337, 345, 351-353, 358-379, 382-387, 391, 397-414, 418, 426-433, 434, 442-446, 450, 468-470, 478, 498, 508, 516, 542, 548-551, 576 kHz
Detect and eliminate the pneumococcus!

11.6.9. Mycoplasmal Pneumonia

Mycoplasma pneumoniae is generally the cause of pneumonia, epidemics occur especially in confined communities, f.i. students, military personnel and families.
Symptoms: The disease often starts with fatigue, sore throat, followed by a dry cough with fever, pharyngitis and by an often multilobular pulmonary infiltration, concerning which, the roentgenographic signs are more extensive than indicated by physical examination. This microorganism can also cause upper respiratory way infections with no pneumonia and asymptomatic infections aswell. Attacks of severe coughing may produce sputum. Occasionally anemia, joint pain and even neurological problems can develop. Some patients feel weak and tired even after several weeks. The usually mild mycoplasmal pneumonia can get severe, though most people can recover without treatment. This infection can causes many a disease including urethritis, colpitis, other urogenital inflammation, osteomyelitis and neurological diseases as well. The genital tract of infants frequently becomes colonized at birth with M. hominis, M. genitalium, M. bovine group-7, and with T-strain Mycoplasmas, coming presumably from the birth canal; and about 15% of the infants have nasopharingeal colonization with these organisms, too. At about one year of age, the rate of the carriage of these microorganisms decreases, and then increases again at the time of puberty.
Diagnosis: by laboratory findings such as leukocytosis, increased sedimentation, x-ray. Complement fixation tests, fluorescent antibody tests, indirect hemagglutination method and growth inhibition tests all offer a highly specific diagnostic information.
Differential diagnosis: by distinguishing it from pneumonia of every type, from influenza, Q-fever, psittacosis and from tularemia infection.
Treatment: by administering doxycyclin, erythromycin etc. Advisably for a long time.
Its resonant frequencies are: 306-308, 320-324, 337-344, 346-352, 362, 397, 499 kHz
Its general ranges are: 321-324, 337-344, 346-350 kHz

11.6.10. Viral Pneumonia

Viral pneumonia infections can be caused by a variety of viruses including myxoviruses, Respiratory Syncytial Virus, adenoviruses, rhinoviruses, Coxsackie viruses, ECHO viruses, reoviruses, measles and HVZ-viruses. Secondary bacterial pneumonia may complicate viral pneumonia, especially in case of influenza and measles.
Viruses frequently cause lung infections concerning infants and children, most frequently the Respiratory Syncytial Virus, the Adenoviruses 370-387, 391-397 kHz, the influenza A and B viruses 284, 307-324 kHz, and the parainfluenza viruses are the causative agents (see Chapter 5.1. and 5.2.).
Measles virus cause pneumonia, mostly in case of malnourished children.
Among people of any age with an impaired immunity, there can develop severe pneumonia caused by Cytomegalovirus (see also Chapter 5.2.4.4.) and by Herpes Simplex Virus (see also Chapter 5.2.4.1.).
Diagnosis: by x-ray, by physical examination.

Treatment: symptomatically, in case of a secondary bacterial infection by administering antibiotics.
RFR method: detects and may eliminate the virus!
The general range of RSV is: 377-385 kHz
The frequency resonances of other Respiratory Syncytial Viruses are: 340-343, 362-365, 566-569 kHz
The frequency resonances of Adenoviruses are: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz
The frequency resonances of Influenza A and B viruses are: 284, 307-324 kHz
The frequency resonances of Cytomegalovirus are: 304-307, 348-351, 405-410 kHz
The frequency resonances of Herpes Simplex Virus are: 290-294, 344-346 kHz

11.6.11. Fungal Pneumonia in General

Fungi are apt to cause pneumonia: *Histoplasma capsulatum* causes histoplasmosis, *Coccidioides immitis* causes coccidioidomycosis and *Blastomyces dermatitidis* causes blastomycosis.
Other fungal infections occur primarily among people with severely suppressed immune systems. These infections include cryptococcosis, caused by *Cryptococcus neoformans*; aspergillosis, caused by *Aspergillus*; candidiasis, caused by *Candida albicans*; nocardiosis, caused by *Nocardia asteroides*, and mucormycosis caused by fungi of the class of *Phycomycetes*.
The infection may cause acute pneumonia, or it may develop into chronic pneumonia causing symptoms persisting for months. The disseminated form of the disease is likely to occur in case of people suffering from AIDS or other immune system disorders.
Treatment: by administering antifungal drugs, such as itraconazole, fluconazole, or amphotericin B.
The resonant frequencies of Histoplasma capsulatum are: 298-308, 315-319, 374, 380-385, 424, 432-435 kHz
The resonant frequencies of Coccidioidomycosis (Coccidiodes immitis and others) are: 321, 334-338, 347, 362-366, 370-374, 382-389, 391-396, 436, 456, 498, 512, 540-549, 544, 574 kHz
The resonant frequencies of Blastomycosis (Blastomyces dermatitidis) are: 304-307, 316-319, 371-373, 428-434 kHz
The resonant frequencies of Cryptococcosis (Cryptococcus neoformans) are: 296, 304-305, 313-319, 374, 402-405, 438, 454, 486-490, 534 kHz
The resonant frequencies of Aspergillosis are:
Those of Aspergillus flavus: 434-438, 464-468, 504 kHz
Those of Aspergillus glaucus: 387-389, 534-539 kHz
Those of Aspergillus niger: 350-359, 393-397 kHz
Those of Aspergillus terreus: 344-348, 380-387 kHz
The most frequent resonances found in case of Aspergillosis are: 346, 356, 380-387, 390-395, 466, 504, 536 kHz
The resonant frequencies of Candidiasis (Candida albicans) are: 297, 332, 345, 352, 359, 372, 380-390, 396, 403, 410, 443-453, 520, 554-558, 568, 572-586, 590 kHz
The resonant frequencies of Nocardiosis, i.e. Nocardia asteroides are: 350-357, 360-371, 454, 464-484 kHz
The resonant frequencies of Mucormycosis are: 313-318, 338, 347, 351, 364-368, 373-376, 384-389, 392-398, 401, 446-455, 482-490, 511, 540-546 kHz
The resonant frequencies of Mucormycosis in Diabetes mellitus are: 318, 473 kHz
The resonant frequencies of Mucor plumbeus are: 295-297, 369-370, 400-402, 448-450 kHz

The resonant frequencies of Mucor racemosus are: 317, 365, 373-375, 384-389, 396, 408, 445-449, 454, 484-486, 497 kHz
The resonant frequencies of Mucor mucedo are: 292-293, 313-315, 376, 498 kHz

11.6.12. Pneumocystis Pneumonia

Pneumonia caused by *Pneumocystis carinii* occurs among patients with impaired antibody responses and/or cellular immune responses, or among people suffering from severe protein-calorie malnutrition. A progressive pulmonary insufficiency is the cardinal clinical manifestation of the illness. A chromatoid body of an oval or crescent merozoite form, as small as 1-2 micrometers, can be histologically observed. On account of cyst ruptures mature trophozoites, measuring from 2 to 4 micrometers are released into the alveoli. This microorganism is of low virulence, proliferates slowly and may require the presence of another microbial agent for its multiplication.
Pneumocystis carinii is a common organism that may reside harmlessly in normal lungs, usually causing a disease solely if the defense of the body is weakened due to cancer, cancer treatment, immunosuppressive treatment, or AIDS. This infection is often the first indication that a person with HIV infection has developed into AIDS. The fully developed clinical picture includes severe dyspnea and tachypnea. The patient shows extreme air hunger, is anxious and cyanotic. The person may suffer from dry cough. Fever is absent or slight. Roentgenograms show hazy alveolar infiltrations spreading from the hilus and eventually affecting most parts of the lung. Some patients may develop peripheral, somewhat nodular infiltrations, which can be confused with other infections or even with malignant processes usually requiring biopsy to confirm the diagnosis. Focal emphysema may be present, but pleural effusions are rare. Complications include pneumothorax caused by a ruptured emphysematous blob.
Diagnosis: by examining the Giemsa stained smears by phase- and DIC-microscopy, or by fluorescent antibody, eosinophilia, x-ray, biopsy.
Differential diagnosis: by distnguishing it from a cytomegaloviral infection, pulmonary aspergillosis, other mycotic infections, pulmonary fibrosis.
Treatment: by administering trimethoprim-sulfamethoxazole, dapsone, clindamycin primaquine.
RFR method: detects and may eliminate the pathogen!
The resonant frequencies of Pneumocystis carinii are: 348, 379, 404-410, 416 kHz

11.6.13. H1N1 Influenza

The flu pandemic occuring in 2009. is a global outbreak of the new strain of influenza A virus subtype H1N1, termed by the World Health Organization (WHO) Pandemic H1N1/09 virus, first identified in April 2009. The disease has also been termed 2009 H1N1 Flu by the U.S. Centers for Disease Control and Prevention (CDC), and is generally called swine flu. H1N1 influenza can spread by coughing, sneezing, or by touching contaminated surfaces, nose and mouth. This type of influenza is also an acute infection of the respiratory tract: affecting the nose, throat and the lungs. The virus can be attached to and then penetrate into the respiratory epithelial cells of the trachea and bronchi. The replication of the viruses results in the destruction of the host cells. Viremia does not occur. The virus shed into the respiratory secretions remains there for 5-10 days. Symptoms, lasting even for a week, are the same as those of the seasonal flu, such as high fever, myalgia, rhinorrhea, sneezing, sore throat, pharingitis, cough, rhinitis, conjunctivitis, headache, cervical lympadenopathy, muscle and joint pains. Nausea, diarrhea and vomiting were also been reported. Into a higher risk group belong persons suffering from various immune system damages caused f.i. by *Mycoplasma*, *HTLV* and *Coronavirus* (SARS) infection, or by immunsuppressive medications and systemic chronic diseases, as f.i.

asthma bronchiale, diabetes mellitus, heart diseases, etc. Children with neurodevelopmental disorders also belong to a higher risk group and so do pregnant women as well. For patients belonging to these higher risk groups the disease can prove to be fatal.
Prevention: by vaccination and by respecting hygiene. The usual influenza vaccine administered at the beginning of an influenza season is not effective for this viral strain.
Diagnosis: by viral strain identification.
Treatment: by administering oseltamivir (Tamiflu) or zanamivir (Relenza). The initiation of giving these antiviral agents within 48 hours from the onset of the symptoms is imperative for the effectiveness of the treatment.
RFR method: can detect and eliminate the virus.
Its most frequent resonances are: 276-286, 309-311, 560 kHz
The most frequent resonance the new mutant (2010): 250-268 kHz

11.7. Tuberculosis

Tuberculosis, (see also Chapter 6.14.4.1.) a contagious, potentially fatal infection is caused by the airborne *Mycobacterium tuberculosis, Mycobacterium africanum* and *Mycobacterium microti,* as well as by *Mycobacterium bovis,* transmitted by unpasteurized milk. Though the mycobacteriums are common bacteria, they generally cause infections only among people with impaired immune system. Bacteria causing tuberculosis infect primarily the lungs but may also attack the lymph nodes, the bones, the skin, and other tissues, too. Many mycobacterium species are highly resistant to most antibiotics, which are used to treat tuberculosis. These infections are caused by inhaling contaminated indoor air, which only occurs, if a person with active tuberculosis coughs out bacteria remaining in the air for several hours. Fetus may acquire tuberculosis from its mother before birth or during birth by breathing in, or swallowing infected amniotic fluids. Infants may get tuberculosis after birth by breathing in air containing infected droplets. Mycobacterium avium infections occur via infected birds: parrots, finches and other birds.
The immune system of a person infected, usually destroys the bacteria, or seals them off at the locus of infection. The activation of dormant bacteria can occur if the person's immune system becomes impaired, for example, by AIDS, by getting corticosteroids, chemotherapy, or by being at an advanced age, in which cases the infection can be life threatening.
Active tuberculosis usually begins in the lung as a pulmonary tuberculosis.
An infected person may at first simply feel unwell or have a cough, which can be put against his smoking or a recently suffered flu. The cough may produce a small amount of green or yellow sputum cought out in the morning. The sputum may be streaked with blood, large amounts of blood are rarely experienced. Night sweat and a low grade fever are also common. Shortness of breath may be a signal of pneumothorax or of fluid getting into the pleural space.
Miliary tuberculosis, a potentially life-threatening type of tuberculosis can develop if a large amount of bacteria spreads by the bloodstream all through the body. The symptoms of miliary tuberculosis are vague and not characteristic; incl of weight, fever, chills, weakness, anemia and other blood abnormalities, a general feeling of discomfort and difficulty in breathing.
There do exist certain other species of the mycobacterium genus, which can cause infections in the lung. The members of this Nontuberculous Mycobacteria Complex (NTM) are f.i. *Mycobacterium avium,* and *Mycobacterium fortuitum.*
Diagnosis: by physical examination, chest x-ray, tuberculin skin test, laboratory analysis of the sputum, etc. People suffering from severe tuberculosis and having a damaged immune system may have false negative test results.

Treatment: by administering antibiotics even the most advanced cases of tuberculosis can be cured. There are some antibiotics that can be used, each of which can kill a million but one of bacteria, thus, at least two drugs with different mechanisms of action should always be given, which then together, are able to kill virtually all bacteria. The most often used antibiotics are isoniazid, rifampicin, pyrazinamide, streptomycin and athambutol.
RFR method: is to be used after treatment with antibiotics, or together with it. The method is advised to begin only after an exact measuring of resonances, as the bacteria can vary and change their characteristic frequencies.
The general range of Mycobacterium tuberculosis is: 429-436 kHz
Its other frequencies are: 332-333, 345, 360-366, 368-370, 372, 374, 378-383, 392-394, 397-401, 409-410, 432, 522 kHz
The general resonant frequencies of Mycobacterium bovis are: 382-387, 428-432 kHz
The resonant frequencies of Mycobacterium avium are: 310, 340, 347-350, 357, 369, 374-379, 383, 394-398, 417, 447-450, 544 kHz
This list is not yet complete, as there are other subspecies having different wave lengths.

11.8. Severe Acute Respiratory Syndrome (SARS)

Severe acute respiratory syndrome (SARS) is a viral respiratory illness caused by a coronavirus, named SARS-associated coronavirus (SARS-CoV). SARS was first reported in China, February 2003. SARS-CoV is a newly known virus, easily transmitted from person to person by respiratory droplets affecting the mucous membraines, when an infected person coughs or sneezes, or, if a person after touching a contaminated object touches his/her own mucous membranes. It is possible, that its spreading can happen in many an other way, too. The illness affects more than two dozen countries in North America, South America, Europe and Asia.
Symptoms: In general, SARS begins with high fever, headache and a feeling of discomfort. Mild respiratory symptoms and less often diarrhea may be experienced. Dry cough, pneumonia will be developing in less than a week.
Prevention: By avoiding contamination, by frequent washing of the hands even with alkohol based hand sanitizers. There is no available vaccine against the SARS-CoV.
Treatment: symptomatically, as no specific therapy is known yet. Infected peple are to be controlled with mouth an throat swabs and blood testing three times a week for 2 weeks, once a week for the next months until there can no virus be detected for 3 following weeks. In problematic cases repeated nose washing, bronchoscopy, bronchial lavage etc. might be still necessary.
Differential diagnosis: by distinguishing it from pneumonia cused by any other agents.
Diagnosis: by PCR (of blood, stool, respiratory secretions or body tissues), though the negative results do not exclude the possibility of SARS-CoV infection.
RFR method: detects and may eliminate the coronavirus!
The most frequent resonances are: 310-320, 350, 357, 381-387, 389, 395-398, 445, 464-467, 470-481 kHz

11.9. Parasite Cystic Diseases of the Lung

Certain protozoans, or worms enter the human body by ingesting, or via the skin or the lungs. Parasites infecting the intestines can infect other organs by spreading their larvae.
In case of *Toxocariasis* the invasion of the organs happens by roundworm larvae of *Toxocara canis* or *Toxocara cati*. The larvae penetrate the intestinal wall and are spread by the blood into the brain, the eye, the liver, the lung and the heart. The larvae cause damages while migrating to tissues as well as eosinophilic inflammations moreover, even eosinophilic abscesses or granulomas in the liver and the lungs. The disease is named visceral larva migrans, as the larvae move slowly.

In case of Echinococcosis, occuring among persons living in infected areas liver or lung cysts can develop. These cysts vary in size and can be present without causing any symptoms, but in certain other cases can cause chest pain, chronic cough, expectoration, dyspnea and even pneumothorax. Eosinophilic pneumonitis, pleural effusion, parasitic lung embolism and hemoptysis can also occur. Cysts may rupture into the peritoneal or the pleural cavity, or even into the blood vessels, causing extraordinary manifestations and more severe complications as well.
Diagnosis: symptomatically, by detecting specific antibodies, by chest x-ray.
Treatment: by administering mebendazole, albendazole. Surgical removal is needed only in certain cases.
RFR method: can detect the parasites.
The resonant frequencies of toxocariasis are: 390-398, 432-438 kHz
The resonant frequencies of echinococcosis are: 440-447, 450-470 kHz

11.10. Paragonimiasis of the Lungs

Paragonimiasis is a chronic infection of the lungs caused by the trematodes of the genus *Paragonimus*. Clinically, the disease is characterized by cough and hemoptysis (see also Chapter 9.3.4.). An eosinophilic granuloma is formed about the adult worm, leading eventually to the formation of a fibrous cyst. Pulmonary lesions develop frequently communicating with a bronchiole, resulting in a secondary bacterial infection. The picture shows a kind of chronic bronchitis and bronchiectasis and the production of brownish sputum and hemoptysis. Fibrosis and calcification occur, presenting a picture of tuberculosis.
Diagnosis: by peripheral blood eosinophilia, by finding the operculated ova in the sputum, stool, pleural fluid, or tissues, complement fixation tests.
Treatment: by administering Bithionol, Praziquantel, Yomesan, and other anthelminthic drugs.
RFR method: detects the parasite, and after administering antiparasitic drugs, it is to be used in order to eliminate them.
Its resonant frequencies are: 347-351, 430-447, 557-560 kHz

11.11. Eosinophilic Pneumonia, Hypersensitivity Pneumonitis

Allergic alveolitis, extrinsic allergic pneumonia, allergic interstitial pneumonitis, and hypersensitivity pneumonitis are all terms referring to the same clinical entity, reflecting an immunological reaction at various parts of the lung.
Hypersensitivity pneumonitis (f.i. extrinsic allergic alveolitis, allergic interstitial pneumonitis, organic dust pneumoconiosis, dust mite allergic pneumonitis) is an inflammation in and around the tiny air sacs of the lung (alveoli) caused by an allergic and *hypersensitive reaction to inhaled dusts, microorganisms or, less commonly, chemicals and biological materials.* Many dust types can cause allergic reactions in the lungs. Organic dusts containing microorganisms, proteins and chemicals as well, may cause hypersensitivity pneumonitis. Respiratory illnesses induced by a recognized exposure to the spores of thermophilic actinomycetes, particularly *Saccharopolyspora rectivirgula* (formerly *Micropolyspora faeni*) and *Thermoactinomyces vulgaris*, are named *farmer's lung*, *mushroom picker's lung*, and *bagossosis*, according to the occupation of the individual. Farmer's lung, caused by repeated inhaling of heat-loving bacteria (Thermophilus) present in moldy hay, is a well-known example of hypersensivity pneumonitis. In this case the hypersensitivity will develop to natural mediator substances.

A combination of type III and type IV allergic reactions will damage the lung tissues in case of hypersensitivity pneumonitis. Lymphocyte sensitization and antibody formation lead to lung inflammation and to the building up of white blood cells in the walls of the alveoli. Functioning lung tissues, replaced or destroyed, lead to symptomatic diseases.
Aspergillus fumigatus, caused pneumonia occurs among atopic or asthmatic individuals, and is a unique clinical syndrome termed *allergic bronchopulmonary aspergillosis.*
Eosinophilic pneumonia, also named pulmonary infiltrates with eosinophilia syndrome, is a special form of hypersensitive allergic pneumonia, in case of which, eosinophils, a special type of white blood cells, appear in an increased number in the lungs and also in the bloodstream. Eosinophils take part in the immune defense of the lung. The number of eosinophils increases during many inflammatory and allergic reactions, including the case of asthma bronchiale, which frequently occurs in certain types of eosinophilic pneumonia.
Asthma bronchiale affects genetically predisposed people and is characterized by a chronic allergic and immunologic inflammatory process of the lower respiratory tract, in case of which, the bronchioli occasionally may get narrowed and get filled with an excessive amount of mucus, due to their hyperreactivity to certain stimuli. The interaction caused by environmental and genetic factors results in airway inflammation, limits the airflow and leads to functional and structural changes of the airways, f.i. to bronchospasm, mucosal edema and mucus plugs. This airway inflammation is caused by various interacting cells, cellular elements and cytokines and can lead to recurrent or persistent bronchospasm, which can get released spontaneously or as a result of an effective therapy.
Allergic asthma is often associated with a personal and/or familial history of allergic diseases such as rhinitis, urticaria and atopic dermatitis, with positive skin reactions to intradermal injections of airborne antigen extracts, and is associated with an increased level of total and specific serum IgE and with positive responses to provocation by specific antigens by using inhalation tests. Allergic asthma is often seasonal, occurring mostly among children and young adults. A variety of compounds used in industry can cause asthma bronchiale concerning susceptible individuals. The most common triggers, in case of children, are *viruses causing a common cold*, *RSVs*, *mycobacteria, measles, Hepatitis A virus* and *other viruses*. There may develop an asthma process initiated by *Mycoplasma fermentans, Mycoplasma pneumoniae* and *HTLV* infections, in case of which the airway inflammation is caused by interaction between various cells, cellular elements and cytokines. In case of susceptible individuals, this airway inflammation may cause recurrent or persistent bronchospasms. Asthmatic airway inflammation is associated with airway hyperreactivity or bronchial hyperresponsiveness (BHR), which is defined as the inherent tendency of the airways to narrow, in response to a variety of stimuli such as allergens, haptens and irritants.
Lymphocytes play an important role in the pathogenesis of asthma. According to certain theories the airway inflammation present in asthma is characterized by the loss of balance between two populations of Th lymphocytes, i.e. Th1 and Th2. Th1 cells produce IL-2 and IFNs, which are critical cellular defense factors of the response to infections. In contrast, Th2 generates a family of cytokines (IL-4, -5, -6, -9, and -13) that can mediate allergic inflammations. The imbalance of the Th1/Th2 relationship can initiate allergic and immune-autoimmune processes.
The symptoms of asthma, ranging from mild to life threatening, can usually be controlled with a combination of drugs and environmental changes. Asthma disease is sometimes associated also with chronic respiratory impairments. The limitation of the airflow may be but partially reversible, owing to the developing alterations in the airways such as hypertrophy and hyperplasia of the smooth muscles, subepithelial fibrosis occuring in case of chronic untreated patients.
According to genetic association studies more than 100 genes are associated with asthma and many of these genes are related to the immune system or to modulating inflammations.

Some genetic variants may only cause asthma if combined with specific environmental exposure.
Inhaled natural allergens include common household wastes, such as those of house dust mites and cockroaches, grass pollens, mould spores, the epithelial cells of pets, other organic compounds, including perfumes and perfumed products. Others, f.i. soap, dishwashing liquid, laundry detergent, fabric softener, paper tissues, paper towels, toilet paper, shampoo, hairspray, hair gel, cosmetics, facial cream, sun cream, deodorant, cologne, shaving cream, aftershave lotion, air freshener and candles and oil-based paints can also be provokers of asthma.
Medicaments causing asthma may be allergens or haptens, including aspirin, β-adrenergic antagonists, penicillin and others, food allergens may be milk, peanuts, eggs, etc. Other environmental materials, such as fossil fuel, ozone, smog, nitrogen dioxide, and sulfur dioxide may also be allergic sources. Various industrial compounds and other chemical components inducing asthma are sulfites, monochloramines (NH_2Cl), dichloramines ($NHCl_2$), trichloramines (NCl_3), and various others.
An other process causing asthma is associated with an infection caused by tiny worm larvae of *Ascaris lumbricoides*. Dogs, cats, pigs, horses and human beings all can get Ascaris infections. The larvae of this worm migrate through the wall of the small intestine and are carried by the lymphatic vessels and the bloodstream to the lungs. There, they pass into the alveoli and than ascend in the respiratory tract, and will be swallowed by the patient. The antigens of Ascaris, (and certain other worm antigens) can provoke an allergic cascade reaction among persons susceptible to it. Ascaris antigens and certain other worm antigens can cause hypersensitivity to histamine and to other biological mediators. The antigens of ascaris and other worms may condition other enviromental antigens, inducing thus new allergic cascade reaction over again. Most of the ascaris infections are coinfected also with *Bacteroides fragilis* which is usually the carrier of a species of *Coxsackie virus*.
In case of persons becoming hypersensitive owing to worm antigens, other new environmental antigens will cause asthmatic reactions. Asthma sufferers may become allergic to many an air pollutant, f.i. pollens, animal danders, smoke, etc. The production of histamine in the lungs and histamine sensitiveness are increased under these circumstances. Some other important mediators also play a role in the evolution of the immediate type hypersensitivity, f.i. the slow-reacting substances of anaphylaxis (SRS-A), platelet-activating factors (PAF), eosinophil chemotactic factors of anaphylaxis (ECF-A), basophil kallikrein of anaphylaxis (BK-A), neutrophil chemotactic factors of anaphylaxis (NCF-A) and secondarily, the prostaglandins as well.
Asthma disease can have many a different cause and many different clinical forms, such as from seasonal allergy to other immune-autoimmune processes.
The symptoms *of asthma bronchiale* are paroxysms of dyspnea, cough and wheezing. It is an episodic disease, where acute exacerbations are interspersed with symptom free periods. Most attacks are short-lived, lasting from minutes to hours and after their resolution the patient seems to be clinically completely recovered. However, there can be a phase, in which the patient experiences a certain degree of airway obstruction daily. This phase can be mild, with or without superimposed severe episodes, or be much more serious, with severe obstructions of the airways persisting for days or weeks, which latter condition is known as status asthmaticus. Allergic asthma is often associated with a personal and/or familial history of allergic diseases such as rhinitis, urticaria and eczema; with positive wheal and flare skin reactions to intradermal injections of airborne antigen extracts; and is associated with an increased level of total and specific serum IgE and with positive responses to provocation with specific antigens by using inhalation tests. Allergic asthma is often seasonal and occurs mostly among children and young adults. A variety of compounds used in industry can cause asthma bronchiale concerning susceptible individuals.

Tiny ascaris larvae (i.e. ascaris antigens) can also cause asthma bronchiale, but less commonly. Ascariasis is caused by an intestinal roundworm *Ascaris lumbricoides* (see also Chapter 9.1.1.). Dogs, cats, pigs, horses and human beings all can get Ascaris infestation. The larvae of this worm migrate through the wall of the small intestine and are carried by the lymphatic vessels and the bloodstream to the lungs. There they pass into the alveoli and than ascend in the respiratory tract, and will be swallowed by the patient. The antigens of Ascaris, (and certain other worm antigens) can provoke an allergic cascade reaction among susceptible persons. Ascaris antigens and certain other parasite antigens cause hypersensitivity to histamine. Mycoplasma pneumoniae and M. fermentans have a key role concerning the development of asthma bronchiale. Bordetella pertussis, HTLV, RSV and certain Coxsackie viruses (resonancing by 360-366 kHz) and perhaps Bacteroides fragilis as well can influence the development of this illness. A Mycoplasma pneumoniae coinfection is in most cases of ascariasis to be found, which bacterium often carries Coxsackie viruses with itself (see also Chapter 9.1.1.). In case of a hypersensitive person these antigens can cause asthmatic reactions. Asthma sufferers become allergic to many air pollutants, f.i. pollens, animal danders, smoke, etc. The production of histamine in the lungs as well as the histamine sensitization is increased in these cases. (The vast interconnectedness of histamine to allergies has been scientifically well studied. Other important mediators also play a role in the evolution of the immediate type hypersensitivity, f.i. the slow-reacting substances of anaphylaxis (SRS-A), platelet-activating factors (PAF), eosinophil chemotactic factor of anaphylaxis (ECF-A), basophil kallikrein of anaphylaxis (BK-A), neutrophil chemotactic factor of anaphylaxis (NCF-A) and secondary the prostaglandins as well.

Diagnosis: by physical examinations, eosinophilia, x-ray, by immunological and hypersensitivity tests.

Treatment: by administering selective beta-2 adrenergic agonists to relieve sudden attacks of *asthma bronchiale*. Inhalative corticosteroids suppress the allergic inflammation and the hypersensitivity and can prevent new attacks as well, but might have side effects. Inhibitors of mast cell degranulation, f.i. disodium chromoglycate, anticholinergic drugs, leucotriene modifiers, theophylline etc. have a less important role in the treatment. Desensitization, immunotherapy may be effective. The treatment of Ascariasis can be done by administering pyrantel pamoate or mebendazole.

RFR method: can search for the causative pathogen organisms, f.i. Ascaris larvae or seldom other worm's larvae. Can eliminate the larvae in the lung, but should solely be used after medical treatments.

The resonances characteristically found in case of asthma bronchiale are:
The resonant frequencies of Mycoplasma pneumoniae are: 321-328 kHz
The resonant frequencies of Mycoplasma fermentans are: 442-451kHz
The resonant frequencies of Coxsackie viruses are: 360-366 kHz
The resonant frequencies of Ascaris lumbricoides (adults and larvae) are: 384, 402-410, 586 kHz

If these can not be found, one should look for other microorganisms, too! Asthma resonances depend on the origin of asthma and on secondary infections. The time of its treatment has to be long-lasting untill all pathogens are eliminated. Prednisolon can help to stop the allergic and immune processes of asthma.

11.12. Moldosis

Many types of molds can cause allergic reactions in the lung and in other parts of the body. Organic dusts containing molds or mold proteins may cause hypersensitive pneumonitis and asthma-like reactions.

Hypersensitive pneumoconiosis (f.i. extrinsic allergic alveolitis, allergic interstitial pneumonitis, mold dust pneumoconiosis) is an inflammation in and around the tiny air sacs

of the lung (ie. Alveoli) caused by an allergic immune reaction to inhaled molds, organic dusts, and, less often, chemicals, too.
But a small number of people inhaling these common dusts will develop allergic reactions, and only a small proportion of those, who develop allergic reactions, will suffer an irreversible damage to their lungs. Generally, a person must be exposed to these antigens continuously or at least frequently before sensitivity and disease will develop. Lung damage appears to result from a combination of allergic reactions type III and type IV. Exposure to dusts causes a lymphocyte sensitization and antibody formations leading to the immune inflammation of the lungs and to the building up of white blood cells in the walls of the alveoli. Functioning lung tissue may be replaced or destroyed, leading to symptomatic diseases. Symptoms may include cough, chills, and shortness of breath, fever, loss of appetite, nausea, anxiety, and vomiting.
In a slower form of the allergic reaction, cough, bellows, asphyxia, and shortness of breath or breathlessness may develop over days or weeks, and sometimes may become so severe that the person needs to be hospitalized.
Diagnosis: by pulmonary function tests, x-ray, antibody examinations, allergy testings, lung biopsy, thoracoscopy, etc.
Prevention: the best prevention is to avoid exposure to the antigen, but this may be not possible if a person cannot change his job or his lodgings. To eliminate or reduce the quantity of mold and dust or to wear protective masks may help to prevent the recurrence. The use of Na- and K-hypochlorit disinfection of living quarters can also be of help.
Treatment: symptomatically, by administering corticosteroid anti-inflammatory drugs, as well as by desensibilization.
RFR method: detects and may eliminate the molds.
As to its frequencies, see Chapter 7.2.

11.13. Bronchopulmonary Aspergillosis

Allergic bronchopulmonary aspergillosis, an allergic lung disorder which often mimics pneumonia, is characterized by asthma, eosinophilic airway and lung inflammation, and by an increased number of eosinophils in the blood.
The disease is caused by an allergic reaction to a fungus, most commonly *Aspergillus fumigatus, Aspergillus flavus, Aspergillus glaucus, Aspergillus niger* and *Aspergillus terreus.* This fungus flourishes in soil, in decaying vegetation, foods, dusts and water. A person inhaling the fungus might become sensitized and develop allergic asthma like process. Other fungi, including Penicillium, Candida, Curvularia, and Helminthosporium, can cause an identical illness. In some people, there can develop an even more complex allergic reaction of the airways and the lungs, an illness, which is a typical farmer's disease. Aspergillus species are ubiquitous molds found in organic matter. Though more than 100 species have been identified till now, the majority of all human illnesses are caused by *Aspergillus fumigatus, Aspergillus niger* and, less frequently, by *Aspergillus flavus* and *Aspergillus clavatus.* The transmission of fungal spores to the human host happens via inhalation. The human host's defense against the inhaled spores starts with ciliary actions of the mucous cells of the respiratory tract. Macrophages and neutrophils encompass, engulf and eradicate the fungus. However, many species of Aspergillus produce toxic metabolites that inhibit macrophage and neutrophil phagocytosis. Corticosteroids do also impair the macrophage and neutrophil functions. Immune deficiency (f.i. HIV disease, chronic granulomatous disease, pharmacologic immunosuppression) also contributes directly to the neutrophil dysfunction ad the decreased numbers of neutrophils. In case of immunosuppressed individuals a vascular invasion by the fungus is much more frequent and may lead to infarction, hemorrhage, and necrosis of lung tissue. Persons suffering from **Chronic necrotizing pulmonary aspergillosis** get granuloma formations and alveolar consolidations. Aspergillus may cause

the host hypersensitivity reactions and direct angioinvasions. Respecting the lungs there are more other syndromes caused by this fungus, such as **Allergic bronchopulmonary aspergillosis (ABPA),** *Aspergilloma*, and **Invasive aspergillosis**. In case of immunodeficiency the infection can be *hematogenously disseminated* affecting other organs (f.i. the myocardium, endocardium, kidney, eyes, liver, bones etc.), too, causing there inflammations and even abscesses.
ABPA can occur among patients suffering from asthma bronchiale and cystic fibrosis and have a hypersensitivity reaction to A fumigatus colonization of the tracheobronchial tree. The very rare *allergic fungal sinusitis*, the *bronchocentric granulomatosis* and the socalled *malt worker's lung* are also caused by Aspergillus species.
Aspergilloma (see Chapter 7.2.8.2.) develops in a preexisting cavity in the lung parenchyma. Certain diseases which make patients susceptible to this aspergilloma are f.i. tuberculosis, sarcoidosis, cystic fibrosis and empysematous bullae.
The **symptoms of Invasive aspergillosis**, a rapidly progressive, often fatal infection among severely immunosuppressed patients, (f.i. profoundly neutropenic ones, bone marrow transplants, solid organ transplants, those afflicted with AIDS or having chronic granulomatous diseases), are as follows: fever, cough, dyspnea, pleuritic chest pain, and in more serious cases hemoptysis as well. If the infection gets into the blood vessels, multifocal infiltrates develop, wedge-shaped, pleural-based and cavitary. There can happen a dissemination into other organs, particularly the CNS.
Aspergillus pneumonia differs from pneumonias caused by other pathogens, as it does not directly destroy the lung tissues. These fungi colonize the asthmatic mucus in the airways and cause recurrent allergic eosinophilic inflammations of the lung. This chronic inflammation may even cause bronchiectasis and scarring.
Chronic necrotizing pulmonary aspergillosis is a subacute process usually found among patients with immunosuppression, most commonly associated with lung diseases, alcoholism, long-lasting corticosteroid therapy, etc.
The symptoms of allergic bronchopulmonary aspergillosis are usually alike with those of progressive asthma bronchiale, being: wheezing, shortness of breath, mild fever, malaise though even brownish flecks or plugs may appear in the coughed-up sputum.
Diagnosis: according to the symptoms, by repeated chest x-rays, CT, by microscopical examination of the fungus itself, along with excess eosinophils in the sputum, eosinophilia, specific antibody tests, etc.
Treatment: by administering antiasthma-drugs, especially corticosteroids, antifungal drugs and symptomatically. As Aspergillus appears everywhere in the environment, it is difficult to avoid it.
RFR method: detects and may eliminate the aspergillus species.
The resonant frequencies of Aspergillus flavus are: 434-438, 464-468, 504 kHz
The resonant frequencies of Aspergillus glaucus are: 387-389, 534-539 kHz
The resonant frequencies of Aspergillus niger are: 350-359, 393-397 kHz
The resonant frequencies of Aspergillus terreus are: 344-348, 380-387 kHz
The resonant frequencies of other, non-identified species are: 346-351, 376, 387, 390-397, 466-470 kHz
The resonant frequencies of Penicillium chrysogenum are: 308, 352-356, 372, 444, 495, 547 kHz
The resonant frequencies of Penicillium notatum are: 292, 329, 366, 410, 420, 423, 472-476, 562-578 kHz
The resonant frequencies of Penicillium rubrum are: 334-338, 392, 466-474, 520-524 kHz
The resonant frequencies of Helminthosporium are: 404-407, 492-497 kHz
All patients having allergic bronchopulmonary aspergillosis have an infection caused by Mycoplasma pneumoniae, too. In my opinion, this bacterium sensitizates the host's

immune system to fungi, and causes allergic reactions, provoking a hypersensitive response. *Mycoplasma pneumoniae* activates the most important primary hypersensitive mediators. If this bacterium is eliminated, the hypersensitive state can still remain, as the allergic cascade is going along.
The first step to take is to check and eliminate Mycoplasma pneumoniae!
The resonant frequencies of Mycoplasma pneumoniae are: 321-328 kHz
In the course of elimination the allergic reaction gets worse, making it necessary to administer also antihistamine drogs and glucocorticoids as well. After eliminating M. pneumoniae, one should eliminate the other pathogens present, too, f.i. *Aspergillus*, *Penicillinum* and *Helminthosporium* species.

11.14. Cystic Fibrosis

Cystic fibrosis is a hereditary disease with certain glands which produce abnormally thick secretions causing several symptoms concerning first of all the lungs and the digestive tract. It is the most common chronic lung disease affecting children and young adults, causing often early death. Cystic fibrosis is caused by a defective gene, due to which the mucous cells are only able to produce abnormally thick and sticky fluid, called mucus. This disease affects nearly all the exocrine glands. The sticky fluid is mostly built up in the breathing passages of the lungs and in the pancreas, the organ that helps the digestion. This collection of mucus leads to life-threatening lung infections and serious digestion problems as well. The illness may also affect the sweat glands and the reproductive system. The secretions of the pancreas and the intestines can become so thick, that they may block the glands completely. The mucus can make the airways totally narrow so that, due to decreased oxydation, the pathogens may more easily multiply. The sweat glands, parotid glands, and small salivary glands secrete fluids containing more salt than normal. Nowadays cc 40% of patients suffering from cystic fibrosis are more than 18 years old, the average span of life is approximately 35 years, which proves a dramatic increase owing to the development in therapy of the last three decades, nethertheless, severe lung complications do still lead to death.
Symptoms of the illness are characteristic: there is no bowel movements in the first 24 to 48 hours of life, stools are pale or clay colored, foul smell, salty-tasting skin in case of infants. Later on, in childhood, recurrent respiratory infections caused by Pseudomonas aeruginosa, pneumonia and sinusitis, coughing or wheezing, loss of weight, diarrhea, delayed growth and fatigue come about.
Diagnosis: by lung function test, x-ray, CT scanning, secretin stimulation test, sweat chloride test, trypsin, chymotrypsin and fat examinations of the stool.
Treatment: symptomatically, including the prevention and the treatment of lung complications, good nutrition, physical activity, psychological and social support, etc.
RFR method: can heal the secondary bacterial, **fungal and viral infections.**
The most frequently found resonances in case of cystic fibrosis are:
The resonant frequencies of Pseudomonas: 330-335 kHz
The resonant frequencies of other infections: 370-373, 393, 396-403, 409-412, 442-451, 488-489, 535-536, 570 kHz

11.15. Pulmonary Fibrosis

Many diseases, especially those that involve immune system abnormalities, fibrosing alveolitis, and intestinal pneumonia can cause pulmonary fibrosis.
Common causes of pulmonary fibrosis are:
1. Immune system abnormalities such as rheumatoid arthritis, scleroderma, polymyositis, Sjögren's Syndrome and systemic lupus erythematosus.

2. Infections caused by microorganisms, such as *viruses*, *rickettsias*, *mycoplasmas*, *mycobacterium tuberculosis*.
3. Mineral dusts, such as silica, carbon, metal dusts, asbestos, beryllium, cadmium, etc.
4. Organic dusts, such as molds, bird droppings, fungi spores, aflatoxin, dust mites, etc.
5. Gases, fumes and vapors such as chlorine, sulfur dioxide, etc.
6. Therapeutic or industrial irradiation.
7. Drugs and poisons, such as methotrexate, busulfan, cyclophosphamide, gold, penicillamine, nitrofurantoin, sulfonamides, amiodarone, paraquat, etc.
8. *Mycoplasma pneumoniae* is one of the causative factors which plays a role in the allergic and autoimmune processes of the lungs.

Symptoms are generally: coughing, loss of appetite, weight loss, tiredness, weakness and vague chest pains. In the late phase of the disease, as the level of oxygen in the blood decreases, the skin may take a blueish tinge, the ends of the fingers may become thick or club-shaped. Pulmonary congestion loading the heart may cause heart failure, named cor pulmonale.

Diagnosis: by x-ray, CT HRCT, pulmonary function tests, biopsy, bronchoscope examination The analysis of the blood gases show a low level of oxygen in the blood.

Treatment and Prognosis: in case of an active inflammation in the lung caused by immunological processes corticosteroids, gamma intrferon, cyclophosphamide, methotrexate, azathioprine, colchicine, and mycophenolate mofetil may be of help. Supportive therapy f.i. oxygen therapy, drugs for the heart failure, anticoagulants etc. The prognosis greatly varies: most patients are continuously getting worse, some can survive for many years; a few will die within several months.

RFR method: detects the resonant frequencies of the causative viruses, rickettsias, mycoplasmas, or mycobacteria causing disseminated tuberculosis, B.Fragilis, etc., and can eliminate these microorganisms.

As to the resonant frequencies of viral infections, see Chapter 5.
As to the resonant frequencies of rickettsial infections, see Chapter 6.1.1.1.
As to the resonant frequencies of mycoplasmal infections, see Chapter 6.18.1.4.

11.16. Pulmonary Wegener's Granulomatosis

Wegener's granulomatosis, a potentially fatal disease, is characterized by a severe granulomatous inflammation of the blood vessel walls affecting the sinuses, the lungs, the kidneys, and the skin clinicaly showing lumps named granulomas. Sometimes only the nasal passages, the airways and the lungs are involved. In this condition, Due to this granulomatous vasculitis the affected lung tissues may be destroyed.

This inflammation damages the tissues of important organs by limiting the blood flow supplying them.

The first **symptoms** of Wegener's granulomatosis are vague, including frequently upper respiratory tract symptoms, joint pains, weakness and tiredness.

The most often occuring sign of Wegener's granulomatosis is the involvement of the upper respiratory tract, experienced in nearly every patient. Symptoms include sinus pain, discolored or bloody fluid coming from the nose, and, occasionally, nasal ulcers. A common sign of the disease is an almost constant rhinorrhea („runny nose") or some other cold symptoms which do not respond to the usual treatment rather becoming increasingly worse. Rhinorrhea is caused by a nasal inflammation or a sinus drainage which is painful. A hole developing in the cartilage of the nose, may lead to its collapse which phenomenon is named saddle-nose deformity. The eustachian tubes, which are important for the normal ear function, may become blocked, causing chronic ear problems and loss of hearing. Bacterial infections can cause a socalled Wegener's-related sinusitis with congestion and chronic sinus pain. The in most cases affected lung may not show any symptoms,

nethertheless coughing, hemoptysis, shortness of breath, and chest discomfort can be present. Fever, loss of weight and tiredness can also come about. Chest x-ray examinations may show cavities or dense areas in the lung looking as if they were the signs of cancer.
This severe systemic granulomatous vascular disease can attack the kidney, the muscles, the eyes, the skin and the nervous system likewise.
People suffering from active Wegener's granulomatosis often have ANCA (antineutrophil cytoplasmic antibodies) in their blood. A positive ANCA test, can be useful to support a suspected diagnosis of Wegener's granulomatosis, though the only sure way to diagnose the disease is by performing biopsy of an involved organ, usually the sinuses, the lung, or the kidney). The examination of biopsy helps not only t confirm the presence of this disease, but helps also by offering a different diagnosis.
Wegener's granulomatosis might be a polyetiologic disease though its exact etiology of is as yet uncertain. No geographic or occupational factors have been identified. There may exist an association between the disease and the HLA-DR2 and HLA-B8 antigens, indicating a cetain familial predisposition. There are also etiological data concerning a hypersensitive reaction to inhaled antigens, as well as other data relating to an infectious origin.
Diagnosis: by its clinical signs, x-ray, CT, ultrasound, ANCA blood tests, and histological examination.
Treatment: symptomatically, by administering a combination of corticosteroid, cytotoxic and other immunesuppressive drugs in order to decrease the inflammation.
RFR method: detects and may eliminate viruses and bacteria perhaps present in this disease.
The most frequent resonant frequencies found in case of Wegener's granulomatosis of the lung are: 292-295, 324-327, 353, 398-401, 428-432, 442-444, 450-452, 461-469, 513-517, 540-545, 558-569 kHz
Their elimination is not easy.

11.17. Bronchiectasis

Bronchiectasis is an abnormal stretching and enlarging of the respiratory passages caused by mucus blockage. If the body is unable to get rid of the mucus, the mucus gets stuck and accumulated in the airways. This blockage and an accompanying infection cause a chronic inflammation, leading to the weakening and widening of the passages. The weakened passages can become scarred and deformed, and allow more mucus and bacteria to accumulate, which results in more infections and more blocked airways.
Bronchiectasis, a chronic, obstructive, pulmonary lung disease, manifested by inflamed and easily collapsible airways, is characterized by air flow obstructions and by an impaired clearance of secretions. This abnormal dilation affects the proximal medium-sized bronchi (greater than 2mm in diameter) and is caused by the destruction of the muscular and elastic components of the bronchial walls, and can be either congenital or acquired. There are certain extraodinary forms of bronchiectasis f.i. traction bronchiectasis, which is a distortion of the airways secondary to a mechanical traction on the bronchi from fibrosis of the surrounding lung parenchyma. Though the airways may become dilated in this case, other manifestations of bronchiectasis are lacking. Exposure to toxic gas, f.i. chlorine gas and ammonia may often cause cystic bronchiectasis and irreversible damage to the bronchial airways.
Connatal bronchiectasis affecting infants and children originates from the developmental arrest of the bronchial tree. The more commonly acquired forms occur among older children and adults after an infectious insult, an airway obstruction, and/or by the damage of the immune system. Most of these patients did never smoke. The damage of the bronchial walls is the result of the inflammatory process caused by the pathogen and the host's inflammatory mediators f.i. inflammatory cytokines, nitric oxide, and neutrophilic

proteases. If the peribronchial alveolar tissue is damaged as well, a diffuse peribronchial fibrosis might develop. The most important functional finding of this abnormal bronchial dilatation, bronchial wall destruction and transmural inflammation is the severely impaired clearance of secretions from the bronchial tree, in which way, colonization and infections with pathogens are made easier.
Complications include chronic bronchial infection, recurrent pneumonia, empyema, pneumothorax, lung abscess and cancer. Hemoptysis, though common, does rarely cause death. Amyloidosis and metastatic abscesses were severe risks in the pre-antibiotic era but are nowadays seldom met with. Its mortality is related to progressive respiratory failures and cor pulmonale.
Symptoms: chronic productive cough and a daily, mucopurulent sputum production lasting from months to years characterize this illness. Dyspnea, pleuritic chest pain, wheezing, generally a mild hemoptysis, fever, weakness, and loss of weight can come about, too. Hemoptysis without any sputum ie. dry bronchiectasis is usually a sign of tuberculosis. Patients usually suffer from repeated attacks of bronchitis and pneumonia, requiring often antibiotics, though the pathogens remain often unknown.
In regard to bronchiectasis, the most frequent bacterial infections are caused by *Pseudomonas aeroginosa, Escherichia coli, Hemophilus influenzae, Klebsiella pneumoniae, Staphylococcus aureus, Streptococcus species* and *Chlamydia.*
The most frequent viruses causing infections in this illness are: *RSV, Adenoviruses, Influenza A and B type, CMV,* and *Herpes viruses* and *HCoViruses.*
Other serious infections associated with bronchiectasis are: Allergic bronchopulmonary *aspergillosis* and *HIV* disease.
Congenital anatomic defects which may be associated with bronchiectasis are: bronchopulmonary sequestration, Williams-Campbell syndrome (congenital cartilage deficiency), Mounier-Kuhn syndrome (tracheobronchomegaly), Swyer-James syndrome (unilateral hyperlucent lung), Yellow-nail syndrome, Alpha 1-antitrypsin deficiency
Diagnosis by a compatible clinical history of characteristic chronic respiratory symptoms, microbiological sputum analysis, x-ray, pulmonary function tests, CT scan, bronchscopy, bronchography, immunological examinations, etc.
Treatment: symptomatically, there is no specific medical therapy. Medications may include the administering antibiotics (f.i. aminoglycosides, synthetic penicillin, third-generation cephalosporins, fluoroquinolones), beta-agonists, steroid inhalers, expectorants, etc.
RFR method: detects and may eliminate the pathogen microorganisms!
In case of bronchiectasis many a bacteria and viruses can be found.
The resonant frequencies of the most frequent bacteria to be met with are:
Mycoplasma pneumoniae: 307, 320-324, 337-344, 346-352, 362, 397-404, 499, 524 kHz
Mycoplasma fermentans: 329, 349-353, 361-362, 403-404, 441-445, 448-450, 492-498, 505 kHz
Pseudomonas aeruginosa: 323-325, 330-340, 351-358, 372, 380, 388-397, 401, 414, 438, 447-448, 496, 579 kHz
Escherichia coli: 317-320, 323-328, 354-358, 390-398, 408-412, 454-458, 478 kHz
Haemophilus influenzae: 335-338, 374-375, 452, 482-490, 494, 564 kHz
Klebsiella pneumoniae: 372, 381, 390-392, 397-406, 414-423, 429 kHz
Staphylococcus species: 281, 324-331, 345, 372, 380-384, 397, 402, 445, 482, 491, 537, 557, 567 kHz
Streptococcus species: 288, 311-321, 330, 337, 345, 351-353, 358-379, 382-387, 391, 397-410, 426-433, 450, 478, 508, 516, 542 kHz
Chlamydia: 317-319, 375-386, 429, 444, 480-486 kHz
The resonant frequencies of the most frequent viruses to be met with are:
Respiratory Syncytial Virus: 364, 377-385 kHz

Cytomegalovirus: 304-307, 348-351, 405-410, 534 kHz
For the frequencies of other microorganisms see their respective Chapters.

11.18. Pleurisy (Pleuritis)

Pleurisy is the inflammation of pleura, which is a 2-layered sac holding the lungs, separating them from the chest wall, the diaphragm and the heart. Pleurisy usually develops if an agent, usually a virus or a bacterium, irritates the pleura.
The pleura lining, the inside of the chest, is named parietal pleura, while the pleura covering the lungs is named visceral pleura. In case of healthy persons, the parietal and the visceral pleura are separated by a thin layer of fluid, which lightens the lungs to easily expand and contract when breathing. Pleurisy can cause pain in breathing and may even product a large amount of fluid collecting in the pleural sac, which condition is the socalled pleural effusion. Pleuritis without this fluid accumulation is named dry pleurisy.
Pleurisy can disappear by its own, but can get worse, too, so that the fluid has to be drained away from around the lungs. Some people develop scar tissue i.e. adhesions after having had pleurisy. They then can suffer chronic pain or shortness of breath.
The most common symptom of pleurisy is the chest pain, usually beginning suddenly. The pain can vary from vague discomfort to an intensive stabbing pain. It may only be felt if the person is breathing deeply, or coughs, or it can even hurt more and continuously, and gets worse when breathing deeply and when coughing. The pain is caused by the inflammation of the outer pleural layer and is usually felt in the chest wall right over the locus of the inflammation.
If a large amount of fluid accumulates, it may separate the pleura layers, ad the chest pain ceases. Large amounts of fluid can cause difficulty in expanding one or both lungs when breathing, causing respiratory distress.
Blood in the pleural space called hemothorax, usually result from a chest injury. Very seldom, a blood vessel may even rupture into the pleura space. Pus in the pleural space called empyema can accumulate when pneumonia or a lung abscess spreads into the pleural space. A high cholesterol content of the fluid in the pleural space results in a long-lasting pleural effusion caused f.i. by tuberculosis, rheumatoid arthritis and tumor. Regardless of the cause and type of the fluid in the pleura space, shortness of breath and chest pain are generally always felt.
The major causes of pleurisy are f.i. pneumonia, pulmonary embolism, rheumatoid arthritis, lupus erythematosus, cancer, tuberculosis and other infections caused by viruses, bacteria, fungi, amebas. Allergic reaction caused by drugs such as procainamide, isoniazid, phenitoin and other chemical substances f.i. asbestos.
Common causes of pleural effusion include pneumonia, heart failure, cirrhosis, blastomycosis, coccidioidomycosis, tuberculosis, histoplasmosis, criptococcosis, rheumatoid arthritis, tumors, pulmonary emboli, pancreatitis and lupus erythematosus.
Diagnosis: by auscultation, x-ray, CT, ultrasound, thoracoscope and by he indentification of the pathogen agents.
Treatment: depends on the particular cause of the disease and its symptoms. If the cause is for example a bacterial infection, antibiotics are to be prescribed supported by RFR method.
RFR method:
If the cause is a **viral infection,** one can eliminate it by RFR method, as to the resonant frequencies, see Chapter 5.
If the cause is an autoimmune disease, it can be effectively treated by RFR method, as to the resonant frequencies, see their respective Chapters.
Supportive therapies f.i. analgesics, codein, fluid drainage.

Tuberculosis, histoplasmosis, cryptococcosis, blastomycosis or coccidioidomycosis require all a prolonged treatment with antimicrobal drugs and it is advisable to support it with RFR method, as to their resonant frequencies, see Chapters 6.14.4.1. and 7.1.2.-7.1.5. The use of RFR method is especially useful and favourable in case of anti drug resistant microorganisms. The frequencies of these microorganisms can also be found in their respective Chapters.

11.19. Pulmonary Hypertension

Pulmonary hypertension (PH) means the increase in the blood pressure in the pulmonary arteries, veins and capillaries, (the lung vasculature), leading to shortness of breath, dizziness, fainting and some other symptoms. PH can be a severe disease causing a markedly decreased exercise tolerance and heart failure.

Primary pulmonary hypertension (PPH) is a rare disorder characterized by elevated pulmonary artery pressure without any apparent cause. It is also termed precapillary pulmonary hypertension or, more recently, idiopathic pulmonary arterial hypertension (IPAH). This diagnosis is usually made after excluding other known causes of pulmonary hypertension.

Secondary pulmonary artery hypertension (SPAH) is present if secondary to a pulmonary or a cardiac disorder the systolic pressure in the pulmonary artery is higher than 30 mm Hg or if the mean pressure in the pulmonary artery is higher than 20 mm Hg.

Cardiac and pulmonary disorders, or their combination are the most common causes of secondary pulmonary hypertension. Cardiac diseases produce pulmonary hypertension by volume or pressure overload, although the subsequent intimal proliferation of the pulmonary resistance vessels is adding an obstructive element.

Its other classification system is as follows:

WHO Group I – Pulmonary arterial hypertension (PAH)

Idiopathic (IPAH)

Familial (FPAH)

Associated with other diseases (APAH), f.i.: collagen vascular diseases (e.g. scleroderma), connatal shunts between the systemic and pulmonary circulation, portal hypertension, *HIV* infection, drugs, toxins, or other disorders and diseases.

Associated with venous or capillary diseases

WHO Group II – Pulmonary hypertension associated with heart disease

Atrial or ventricular disease

Valvular disease (e.g. mitral stenosis)

WHO Group III – Pulmonary hypertension associated with lung diseases and/or hypoxemia

Chronic obstructive pulmonary disease (COPD), interstitial lung disease (ILD)

Sleep and breathing disorder, alveolar hypoventilation

Chronic exposure to high altitude

Developmental lung abnormalities

WHO Group IV – Pulmonary hypertension due to chronic thrombotic and/or embolic disease

Pulmonary embolism in the proximal or distal pulmonary arteries

Embolization of other causes, such as tumor cells or parasites

WHO Group V – Miscellaneous

The classification does not include sickle cell disease, nor the infection by Human Herpes Virus-8, associated also with Kaposi's sarcoma.

The most common cause of pulmonary hypertension is associated with atrial, ventricular or valvular damages of the heart leading to pulmonary venous hypertension (WHO Group II) and can be caused by *Coxsackie viral* infections. Pulmonary venous hypertension is likely to develop owing to the systolic or diastolic malfunction of the left ventricle or to valvular

dysfunctions such as mitral regurgitation, mitral stenosis (in consequence of *chronic bacterial* infections), aortic stenosis (atheroma and/or calcification), or aortic regurgitation. It usually manifests itself as pulmonary edema or pleural effusions. As the malfunctioning heart does not pump efficiently, the blood fails to leave the pulmonary circulation in its timely manner, leading to abnormally high pressure in the pulmonary veins. The increased pressure in the pulmonary veins can be transmitted back to the pulmonary arteries.

The most common causes of pulmonary arterial hypertension (PAH, WHO Group I) include *HIV* infection, scleroderma and other autoimmune diseases (caused by *mycoplasma*, *HTLV*), cirrhosis *(Hepatitis viruses)* and portal hypertension, sickle cell disease, congenital heart disease and others. *Human Herpes Virus-8*, also associated with Kaposi's sarcoma, has been demonstrated in patients with PAH, suggesting that this virus may play a role in its development. Recent studies have been unable to find an association between Human Herpes Virus-8 and idiopathic pulmonary arterial hypertension.

Genetical predisposition: In case of a positive family medical history, the disease is termed familial pulmonary arterial hypertension (FPAH). IPAH and FPAH are now considered to be genetic disorders linked to mutations in the BMPR2 gene, which encodes a receptor for bone morphogenetic proteins, as well as mutations in the 5-HT(2B) gene, which encodes a serotonin receptor. There seems to be an association between idiopathic PAH (not only PAH caused by heart malformations) and the Down syndrome (trisomy 21).

Pulmonary embolism *(HSV and nanobacteria)* can also lead to pulmonary hypertension, acutely as well as chronically (WHO Group IV). The treatment concerning these two conditions are vastly different. *Schistosomiasis,* due to the obstruction of pulmonary vessels with the parasite, is a very common cause of pulmonary hypertension in endemic tropical areas such as that of the river Nile.

Lung diseases, including COPD, interstitial lung disease such as Pickwickian syndrome or obesity-hypoventilation syndrome, and possibly also sleep apnea, lowering the oxygen concentration in the blood (causing hypoxia) are well known causes of pulmonary hypertension (WHO Group III). Other causes can be sarcoidosis, histiocytosis X and fibrosing mediastinitis (WHO Group V).

If none of these causes can be found, the disease is termed idiopathic pulmonary arterial hypertension (IPAH). There appears to be a not causative link between IPAH and thyroid diseases.

The pathogenesis of SPAH can be influenced by three predominant pathophysiologic mechanisms:

(1) hypoxic vasoconstriction,

(2) the decrease of the pulmonary vascular bed area, and

(3) volume/pressure overload.

Hypoxic vasoconstriction

Chronic hypoxemia causes pulmonary vasoconstriction by a variety of actions affecting the pulmonary artery endothelium (*nanobacteria* release endothelin 1-3 from the endothelium) and the smooth muscle cells (hypertrophic cardiac muscle cells infected by *Coxsackie* viruses, or/and by *ECHO viruses*), as well as the down-regulation of endothelial nitric oxide synthetase and the reduced production of the voltage-gated potassium channel alpha subunit. Chronic hypoxemia leading to pulmonary hypertension can occur in patients suffering from Chronic Obstructive Pulmonary Diseases (COPD), high-altitude disorders and hypoventilation disorders.

COPD is the most common cause of SPAH. These patients have a survival rate less than 5 years, a severely mismatched ventilation perfusion and a nocturnal or exercise-induced hypoxemia. Other disorders, such as obstructive sleep apnea, neuromuscular disorders, and disorders of the chest wall, may lead to hypoxic pulmonary vasoconstriction and eventually to SPAH.

Obliteration of the pulmonary vasculature

A variety of causes may decrease the cross-sectional area of the pulmonary vascular bed, primarily due to the disease of the lung parenchyma. Patients with collagen vascular diseases have a high risk of developing SPAH, particularly those suffering from systemic sclerosis and CREST (calcinosis cutis, Raynaud's phenomenon, esophageal motility disorder, sclerodactyly, and telangiectasia) syndrome. A mild-to-moderate elevation in the mean pulmonary artery pressure occurs secondary to acute pulmonary embolism. Chronic pulmonary embolisation can result in progressive PAH. *HIV* infection as well as certain drugs and toxins are also known to cause PAH.

Volume and pressure overload

Disorders of the left heart may cause SPAH, caused by volume and pressure overload. The pulmonary blood volume overload is caused by left-to-right intracardiac shunts, f.i. in case of atrial or ventricular septal defects. A left atrial hypertension causes a passive rise in the pulmonary arterial systolic pressure maintaining a driving force across the vasculature. Later on, this persistent pulmonary hypertension will be accompanied by vasculopathy. This can occur secondary to a left ventricular dysfunction, mitral valvular disease, constrictive pericarditis, aortic stenosis, and cardiomyopathy.

The pulmonary venous obstruction is a rare cause of pulmonary hypertension. It may occur secondary to mediastinal fibrosis, anomalous pulmonary venous drainage, or pulmonary venoocclusive disease.

Pulmonary hypertension is usually influenced by two principle causes: genetical predisposition and infection.

The Symptoms of pulmonary hypertension can develop but gradually, patients delay going to physicians for years. Shortness of breath, fatigue, non-productive cough, angina pectoris, fainting or syncope, peripheral edema around ankles and feet can often be experienced and sometimes even hemoptysis (coughing up blood) as well. Pulmonary arterial hypertension (PAH) is typically not accompanied by orthopnea and paroxysmal nocturnal dyspnea, while pulmonary venous hypertension typically is.

Diagnosis: by special physical examinations (auscultation, ECG, echocardiography ultrasound, Swan-Ganz catheter, etc.) and symptomatically.

Biopsy of the lung is usually not indicated unless the pulmonary hypertension is thought to be due to an underlying interstitial lung disease.

Treatment: symptomatically and by giving Prostaglandins, Endothelin receptor antagonists, Phosphodiesterase type 5 inhibitors, etc. and sometimes surgically.

RFR method detects and can eliminate all pathogen microorganisms.

The most frequent resonances are those of:

Nanobacteria: 324-325, 375-381, 560-568 kHz
Coxsackie and ECHO viruses: 307-314 kHz
Herpes Simplex Virus-1: 291-294, 344-346 kHz
Human Herpes Virus-8: 331, 426, 508-510 kHz
Mycoplasma species: 321-324, 331, 428, 442-451, 493-495 kHz
Schistosoma species: 325, 336, 352-354, 433, 443, 472-474 kHz

12. HEART INFECTIONS AND BLOOD VESSEL INFECTIONS

Patients with pulmonary vascular congestions are particularly susceptible to pulmonary infections, but an infection anywhere in the body may precipitate heart failure. The resulting fever, tachycardia, cardiac hypoxemia and the increased metabolic demands may place a further burden on the overloaded, but compensated myocardium of a patient with chronic heart disease.
The development of acute rheumatic fever and a variety of allergic or infectious processes affecting the myocardium may further impair the myocardial function of patients with preexisting heart disease.
If using the RFR method, it is allowed to examine solely compensated patients suffering fom heart disease. By eliminating certain pathogen viruses (such as *Coxsackie viruses*, *Cytomegaloviruses*, etc.) and certain bacteria (f.i. *Staphylococcus, Streptococcus, Borrelia B. sensu lato, Chlamydia,* etc.) the released toxins and other effects can cause problems affecting the heart, so that the RFR method can be used only under the supervision of a cardiologist.
The coronary endothelium synthetizing and releasing vasoactive substances, plays a fundamental role in the basal and dynamic regulation of the cardiac circulation assuring the balance in the normal function of endothelium derived relaxing and contracting substances. Endothelin-1 (2, and 3) are potent endothelial derived vasoconstrictor substances. *Coxsackie virus* and certain other microorganisms living in the endothel are able to release and mobilise endothelines. Endothelin-1 induces vasoconstriction, vascular and myocardial hypertrophy, which are independent risk factors for cardiovascular morbidity and mortality.
The suddenly released, pathogen derived toxins can cause tachycardia, cardiac hypoxemia, coronary arterial and myocardial failures. Among patients with chronic but compensated ischemic heart disease, a fresh, clinically often silent infarct may further impair the ventricular function and aggravate the heart failure, so that a complex ECG and a cardiac testing needs to be done, before examining by the RFR method. Be careful!
All developed symptoms may be very dangerous.

12.1. Rheumatic Cardiac Fever

In case of Rheumatic cardiac fever there are inflammations of the joints (arthritis) and the heart (carditis) present, usually resulting from a streptococcal infection of the throat. Rheumatic fever, being though not an infection, can develop from a chronic streptococcal A type infection, i.e. a *Streptococcus pyogenes infection. Streptococci Group A* are the most virulent species for people, their natural hosts. This *Streptococcus pyogenes* can cause strep throat, tonsillitis, blood infections, scarlet fever, glomerulonephritis, pneumonia and rheumatic cardiac fever, with its toxins damaging the cardiac tissue. Though the illness is an inflammatory, allergic, and maybe an autoimmune reaction, too, provoked by the infection, affecting many parts of the heart, antibiotics are nethertheless widely used to treat the streptococcal infection at its early stage. In this ilness every organ of the body is involved, but the immune responses are insufficient, inadequate, characteristic to autoimmune processes. Rheumatic cardiac fever damages the musculature of the heart, may cause cardiomyopathia, endocarditis and mitral and aortic valve stenosis. If the stenosis is significant, the blood pressure in the atrium will increase, so also in the veins of the lungs, resulting in heart failure, due to which a pulmonary edema will develop.
Symptoms of rheumatic fever vary greatly, depending on which parts of the body become inflamed. Typical symptoms begin several weeks after the disappearance of a streptococcal

sore throat. Major symptoms of rheumatic fever are arthritis, carditis, fever, Sydenham's chorea, erythema marginatum, chest pain and small bumps nodules under the skin. Joint pain and fever usually are the most common first symptoms. One or several joints suddenly become painful and feel tender when touched. They may also be red, hot, swollen and may contain fluid. Ankles, knees, elbows and wrists are usally affected, so may the other joints be involved, too. The acute rheumatic carditis first manifests itself by the appearance of the heart murmurs of either mitral or aortic regurgitation and with the usual symptoms of carditis in general. Pericarditis may occur causing precordial pain, and a friction rub may become audible. This carditis occur usually together with joint pain and fever. Tachycardia, heart failure, chest pain, tiredness, weakness, swellings in the feet, ankles and legs are often present.

Some symptoms of heart failure in case of children are different from those of adults. They may experience shortness of breath, nausea, vomiting, stomach ache and a hacking cough. Tiredness, heart murmurs, tachycardia, valvular lesions are also characteristic. A flat painless rash with a wavy edge (erythema marginatum) may appear as the other symptoms subside.

Diagnosis: by auscultation and according to the respective symptoms, high erythrocyte sedimentation, antibody testing and measuring, ECG, echocardiograph, etc. Rheumatic carditis is almost always associated with significant murmurs.

Treatment: by promptly administering effective antibiotic for this pathogen streptococcus for a long time (penicillin) as well as high dose NSAID f.i. aspirin and corticosteroids. In case of severe carditis, corticosteroid may be given to prevent tissue damage.

RFR method: is to be used together with NSAID and antibiotic treatment! Detect and measure the pathogen Streptococcus!

The most often resonant frequencies of Streptococcus pyogenes are: 358-368, 407-412 kHz

The other frequencies: 313-321, 388, 404, 508-509, 520, 538-539 kHz

12.2. Endocarditis, Myocarditis and Pericarditis

Infective endocarditis is a bacterial, less often a fungal or a viral infection of the endocardium and the heart valves. **Infective myocarditis** is a bacterial or a viral infection of the myocardium. **Acute and chronic pericarditis** is an inflammation of the pericardium, beginning suddenly, being often painful; and characterized by pericardial fluid containing fibrin, red blood cells and white blood cells being in the pericardial space. Acute pericarditis has many a cause, ranging from viral to fungal infections, bacterial infections and life threatening cancers, and can be caused even by AIDS, systemic lupus erythematosus, rheumatoid diseaseas, etc.

Bacteria, viruses and fungi can cause endocarditis either by entering the bloodstream or, in rare instances, by contaminating the heart during open-heart surgery they can lodge on heart valves and infect the endocardium. Accumulations of bacteria with blood clots on the valves can break loose and travel to vital organs, where they can block the arterial blood flow. They can cause stroke heart attacks, infections and damages to the pericardium, myocardium and endocardium. A few bacteria in the blood may not cause immediate **symptoms,** but the *bacteremia* may develop into septicemia, a severe blood infection causing typically high fever, chills, shaking and low blood pressure. A person with septicemia or rheumatoid fever is particularly at risk to get a form of carditis.

Acute bacterial endocarditis usually begins suddenly, with high fever and high heart rate, tiredness, and tends to get rapidly an extensive heart valve damage. Other symptoms of acute and sub acute bacterial endocarditis may include chills, joint pain, pale skin, rapid heartbeats.

Usually, **acute pericarditis** causes fever and chest pain, which typically extend to the left shoulder and sometimes down the left arm. The pain may be similar to that of a heart

attack, except that it tends to be made worse by lying down, coughing, or even deep breathing. Pericarditis may cause cardiac tamponade, a potential fatal condition.
Chronic pericarditis is an infectious inflammation resulting in fluid accumulation or in the thickening of the pericardium beginning gradually and being long lasting. Symptoms of chronic pericarditis include shortness of breath, coughing and fatigue.
Myocarditis is the result of an infectious process, but it may also be present in hypersensitivity diseases such as in acute rheumatic fever, or can be caused by radiation therapy, chemical poisons, physical agents, or drugs. *Coxsackie A and B viral strains*, the *poliomyelitis*, *influenza*, *adenoviruses*, *ECHO viruses* and *rubella viruses* are the best-documented etiologic agents. In case of these diseases, only transient ST-T wave abnormalities can be noted and the myocardial involvement does not influence the course of the disease. Myocarditis is frequently associated with acute pericarditis, especially if caused by *Coxsackie B viruses* or *ECHO viruses*.
Toxoplasmosis relatively often cause acute myocarditis among newborns. Toxoplasma myocarditis may suffer many a member of a family.
Aspergillus myocarditis, an opportunistic infection affects mostly immunocompromised persons.
Chagas' disease, caused by *Trypanosoma cruzi,* produces a form of myocarditis occurring in well-defined acute and chronic forms.
Certain tricyclic antidepressants, the phenothiazines, various aerosol propellants, emetine, daunorubicin or rubidomycin may have myocardial side effects.
Diagnosis: by physical examinations, ECG, x-ray, CT, MRI, cardiac catheterization, blood pressure measuring, bacterial and fungal culturing, antibody detection, etc. Sometimes bacteria can't be cultured from blood samples. In cardiac infections there may be many a resistant bacterium or fungus present.
Prevention: people with heart valve abnormalities, artificial valves or with connatal defects have to be administered with antibiotics before starting dental and surgical procedures.
Treatment: depending on the causative agent, f.i. by administering antibiotics with a bactericid effect, fungicide drugs, and according to its symptoms. Repeated laboratory control is necessary.
RFR method: measure! Detects and eliminates all pathological microorganisms.
The most frequent resonances of the Coxsackie virus A1, A2, A5, A8, A9, A16 are: 406-411 kHz
The most frequent resonances of the Coxsackie virus B1 are: 287-290, 300, 360-370, 392, 407-408, 426 kHz
The most frequent resonances of the Coxsackie virus B2 are: 287-293, 297-301, 360-362, 407-408, 443, 546 kHz
The most frequent resonances of the Coxsackie virus B3 are: 287-293, 297-301, 333-335, 407-409, 444, 498 kHz
The most frequent resonances of the Coxsackie virus B4 are: 307-308, 360-366, 419-426, 430, 534-544, 552-554 kHz
The most frequent resonances of the Coxsackie virus B5 are: 287-291, 331, 360-362, 396, 472, 533, 553-555 kHz
The most frequent resonances of the Coxsackie virus B6 are: 336, 340-343, 350, 366-376, 407-416, 498, 564 kHz
The most frequent resonances of the Streptococcus pyogenes are: 358-368, 407-412 kHz
The most frequent resonances of the Streptococcus family are: 288, 312-321, 337, 345, 351-353, 363,-375, 366-376, 381-385, 391, 397-413, 425-426, 432, 447, 450-453, 478, 508, 516, 542 kHz
The most frequent resonances of the Staphylococcus aureus are: 376-384 kHz

The most frequent resonances of the Toxoplasma are: 393-398, 436, 444 kHz
The most frequent resonances of the Tripanosoma are: 322, 358, 370, 393-398, 412, 423-431, 434-451, 460-465, 520 kHz
The most frequent resonances of the Aspergillus are: 346, 356, 380-387, 394, 466, 536 kHz

12.3. Cardiobacterial Endocarditis

Cardiobacterium hominis is a gram-negative, nonmotile, aerobic bacillus normally residing in the respiratory tract. The caused illnesses are associated with a local infection in the mouth and in 5-10% of the cases occur with the inflammation of the native valves. Endocarditis caused by *C. hominis* accounting for 0.1% of all endocarditis cases, the mitral and the aortic valves are most often affected. Bacteremia together with endocarditis caused by *C. hominis* usually occurs in case of a preexisting structural heart disease and a prosthetic heart valve as well. Many patients have a history of a recent dental procedure and of a poor dentition.

Symptoms include: fever, tiredness, thoracalgia, a peripheral embolic phenomenon, rapid heartbeats, clubbing, petechiae, anemia, loss of weight, sweating, joint pain, splenomegaly and rarely even arthritis, glomerulonephritis and hematuria. People with a birth defect or with an abnormality which allows the blood to leak from one part of the heart to the other (for instance, from one ventricle to the other) also have an increased risk of endocarditis of this type.

The presence of but a few cardiobacteria in the blood may not cause immediate symptoms, nethertheless, bacteremia may sometimes develop into septicemia, causing typically high fever, chills, shaking and a low blood pressure.

Diagnosis: by bacterial culturing, ECG, TEE, by examination of rheumatoid and other factors, etc.

Treatment: by administering ceftriaxone, ampicillin-sulbactam, or ciprofloxacin for 4 weeks.

RFR method: detects and may eliminate the Cardiobacterium hominis and the pathogens of other coinfections as well.

The most frequent resonance frequencies of the Cardiobacterium hominis are: 378-385, 503-510, 550-555 kHz

Use the RFR method of the *C. hominis* together with Ceftriaxone, and detect its elimination. One must be careful, as embolization may occur.

12.4. Endocarditis Caused by Veillonella

The species of the genus Veillonella are small, non-fermentative, anaerobic, Gram-negative cocci. The species Veillonella may be a part of the normal flora of the mouth, the gastrointestinal and the vaginal tract. *Veillonella dispar*, *V. atypica*, *V. parvula*, *V. alcalescence* and *V. montpellierensis* have been cultured from human specimens. They only seldom cause human infections. Veillonella species were sometimes the only etiologic agents identified in serious infections such as in case of meningitis, osteomyelitis, prosthetic joint infections, pleuropulmonary infections, endocarditis and bacteremia. A new species, *V. montpellierensis*, has recently been isolated from the gastric fluid of a newborn and from the amniotic fluid. Veillonella species but very seldom cause endocarditis.

Diagnosis: by bacterial culturing, PCR.

Treatment: by administering effective antibacterial drugs, f.i. cephapirin and gentamycin, oral penicillin V, ampicillin and metronidazole, oral clindamycin, ampicillin, etc. Veillonella can be penicillin resistant.

RFR method: detects and may eliminate the Veillonella species in case of an infection or a disease.
The most frequent resonances are: 400-405, 508-511, 527-530 kHz
This list is not complete.

12.5. Mitral Valve Stenosis and Aortic Valve Stenosis

Mitral valve stenosis, a narrowing of the mitral valve opening, increases the resistance to blood flow from the left atrium to the left ventricle. Similarly, the aortic valve stenosis, a narrowing of the aortic valve opening, increases the resistance to blood flow from the left ventricle to the aorta. These kinds of stenosis are almost always the result of a rheumatic fever. In certain parts of the world, where the antibiotic therapy is already since many decades easily available, there occurs mitral and aortic valve stenosis mostly among older people, who had rheumatic fever in their childhood. In the rest of the world, rheumatic fever is common, and leads to mitral or aortic valve stenosis concerning teenagers, adults and sometimes-even children. One or more attacks of acute rheumatic fever can be the cause of a predominant or pure mitral stenosis concerning approximately two-thirds of adult patients. In this condition the valve leaflets can be diffusely thickened due to a fibrous tissue or due to calcific deposits. Typically, if the mitral valve stenosis is caused by rheumatic fever, the mitral valve leaflets become partially to be fused together. In case of a severe stenosis, the blood pressure increases in the atrium and in the veins of the lungs, resulting in heart failure and in the development of pulmonary edema. The enlargement of the left atrium can be associated with an atrial fibrillation or an irregular heartbeat, causing chest pain and angina on exertion. Such an insufficient blood supply will damage the heart muscles. The resulted heart failure leads to fatigue and shortness of breath on exertion. Rheumatic fever can cause also a tricuspid valve stenosis and a tricuspid valve regurgitation. Pulmonary valve stenosis, which but rarely concerns adults, originates usually from birth defect.
Diagnosis: by x-ray, ECG, echocardiography, ultrasound and cardiac catheterization.
Prevention: a mitral valve stenosis can be prevented only by the prevention of rheumatic fever, a childhood illness that sometimes occurs following an untreated streptococcal throat infection.
Treatment: by administering beta-blockers, digoxin, verapamil, diuretics, alternatively by heart surgery.
People with mitral valve stenosis should be administered preventive antibiotics before dental or surgical procedures to reduce the risk of a heart valve infection and the progression of stenosis.
RFR method can have only a preventive effect. Detects and eliminates the Streptococcus!
Valve stenosis is the end of a long process. Untreated streptococcal infections can lead to mitral valve stenosis and aortic valve stenosis. Treatment has to begin as soon as possible and be controlled for a long time!
The most frequently found resonant frequencies concerning streptococcal infections of this kind are: 288, 307-308, 310-321, 324, 337, 340, 345, 351-353, 362-376, 381-385, 391, 397-413, 418, 426, 432, 440-443, 447, 450-453, 478, 486, 508-511, 516-520, 542 kHz
The list might be not yet complete, as there are other Streptococcus subspecies having other resonance frequencies.

12.6. Cardiomyopathy

Cardiomyopathy is the end state of a long, complicated process and is a progressive disorder with altered structures and impaired functions of the muscular wall of the lower chambers of the heart. Many a known disease can cause cardiomyopathy, but there can be still cases of unknown origin.
Pathologic classification of cardiomyopathies is as follows:
1. Primary myocardial involvement: idiopathic, familial, alcoholic, peripartum, endocardium fibroelastosis, endomyocardial fibrosis, etc.
2. Secondary myocardial involvement: amyloidosis, hemochromatosis, sarcoidosis, connective tissue disease, neuromuscular disease, neoplastic disease, glycogen storage disease, lipoidoses.
The classification of cardiomyopathies can also be made in the following way:
1. Congestive cardiomyopathies: characterized by cardiac dilatation, congestive heart failures, arrhythmias, emboli, and murmurs of mitral and tricuspidal regurgitations.
2. Restrictive cardiomyopathies: characterized by restriction to ventricular filling.
3. Hypertrophic cardiomyopathies: with or without obstruction to ventricular outflow.
The dilated congestive cardiomyopathy is a certain group of heart disorders, where the ventricles are enlarged, the heart isn't able to pump enough blood for the body's needs, thus this state leads to heart failure. Coronary artery disease results in inadequate blood supply to the heart muscles, which can lead to permanent injuries. The remaining uninjured heart muscles then get stretched to compensate for the lost pumping action. If there is no adequate compensation, a dilated congestive cardiomyopathy will develop. Mural thrombi are frequently present in the left ventricle, left atrium and right atrium. On histological examinations the major changes seen are fibrosis, myocardial degenerations and hypertrophy.
Acute inflammation of the heart muscles from a viral infection may weaken the heart muscles and produce dilated congestive cardiomyopathy, named *viral cardiomyopathies.* Infections caused by *Cytomegaloviruses* and *Coxsackie viruses* are the most frequent cause of viral cardiomyopathies. These microorganisms cause acute or chronic inflammations in the myocardium, there are released endothelin substances and the chronically high endothelin-1 level results in vascular and myocardial hypertrophy.
Acute inflammation of the heart musculature *caused by a bacterial infection* may weaken the heart muscles and produce a long lasting dilated cardiomyopathy.
Dilated congestive cardiomyopathy also can be caused by drugs such as alcohol, cocaine and antidepressants. *Alcoholic cardiomyopathy* may develop after a permanently heavy alcohol abuse, which causes a toxic state suppressing the immune defense which furthers the developing of viral infections. *Pregnancy, connective tissue diseases,* f.i. rheumatoid arthritis but rarely cause dilated congestive cardiomyopathy.
If the cardiomyopathy results from an infection, the first symptoms will be sudden fever and flu like symptoms. Whatever the cause, the heart rate will speed up, the blood pressure will be normal or low, fluid will be retained in the legs and the abdomen, and the lungs will be filled with fluid. Due to the enlargement of the heart the heart valves open and close but improperly, which leads to the leakage of the ventricules. This process affects also the heart rhythm.
Diagnosis: by physical examinations and according to the symptoms, x-ray, ECG, echocardiography, MRI.
Treatment: by administering f.i. nitrites, nitrates, beta-blockers, calcium channel blockers, anticoagulants, angiotensin converting enzyme inhibitors, diuretics, etc.
RFR method: detects the viruses or the bacteria and eliminates them! This treatment can stop the process of the disease, but cannot reverse it.
The resonant frequencies of Cytomegalovirus are: 305, 327, 349, 406-413, 512, 543-548 kHz

The most frequent resonances of the Coxsackie virus A1, A2, A5, A8, A9, A16 are: 406-411 kHz
The most frequent resonances of the Coxsackie virus B1 are: 287-290, 300, 360-370, 392, 407-408, 426 kHz
The most frequent resonances of the Coxsackie virus B2 are: 287-293, 297-301, 360-362, 407-408, 443, 546 kHz
The most frequent resonances of the Coxsackie virus B3 are: 287-293, 297-301, 333-335, 407-409, 444, 498 kHz
The most frequent resonances of the Coxsackie virus B4 are: 307-308, 360-366, 419-426, 430, 534-544, 552-554 kHz
The most frequent resonances of the Coxsackie virus B5 are: 287-291, 331, 360-362, 396, 472, 533, 553-555 kHz
The most frequent resonances of the Coxsackie virus B6 are: 336, 340-343, 350, 366-376, 407-416, 498, 564 kHz
The most frequent resonancies found in case of Cardiomyopathy are: 407-413 kHz

12.7. Angina Pectoris

Angina pectoris is a clinical syndrome caused by a transient myocardial ischemia. **Angina**, also named angina pectoris, is a temporary chest pain or a feeling of pressure occuring if the heart muscles don't receive enough oxygen. Certain diseases can result in myocardial ischemia as well as in pain syndromes. Approximately four-fifths of all patients with angina pectoris are men, and those, younger than fifty years of age also are mostly men. Patients usually describe their chest sensation seldom as a pain, mostly as heaviness, pressure, smothering, tightness, choking or squeezing. The typical and most important diagnostic feature of angina pectoris is its relation to exertion and emotion. The discomfort is usually coming on during physical or emotional stress and will be relieved by rest. Anger, fright, hurrying, or sexual activity also may bring on the syndrome. This phenomenon is thought to be related to increased total body oxygen requirements associated with the continuous contractions of the antigravity muscles of the arms and shoulder girdles. The pain may come also during or after eating. Exposure to extreme cold and warm temperatures or to wind may accentuate or precipitate the symptom.
Myocardial ischemia may cause pain in the neck, the jaw, the throat, the chest. Sharp pains, especially those, localized to the left submammary area and of short duration, are rarely caused by myocardial ischemia, but pains described as knife-like and cutting occasionally mean and describe a real angina.
Variant angina, also named Prinzmetal's angina, is characterized by a chest pain of cardiac origin, i.e. an angina occuring usually at rest and cyclic. It is often accompanied by ventricular arrhythmias. The characteristic ECG demonstrates S-T segment elevations during the attacks of the pain, in contrast to the characteristic S-T segment depression in case of the typical effort angina syndrome. Variant angina presents transmural ischemia, often due to the spasm of one of the major coronary arteries.
Usually, angina results from coronary artery diseases, but it can also be caused by abnormalities of the aortic valve, especially by aortic valve stenosis, aortic valve regurgitation, as well as by hypertrophic subaortic stenosis. Besides the variant angina caused by arterial spasms, a *severe anemia*, reducing the supply of oxygen to the heart muscles, also can trigger angina.
The most important pathogenic factor of angina pectoris is the chronic *Coxsackie virus infection*, which increases the demand on oxygen of the myocardium. This higher oxygen demand of the myocardium can cause an ischemic syndrome. In case of a *Coxsackie viral cardiomyopathy* the angina is typically triggered by physical activities and stress because this chronic viral infection alters the function of beta-adrenerg receptors and the accommodation, provoking spasms and myocardial hypoxia. Angina pectoris is therefore

not only a coronary arterial disease, as the beta-adrenerg receptors of the wall of the coronary arteries and the myocardium together will be ill in case of a Coxsackie viral infection and these cause together an anginal symptom. The coronary endothelium synthesizes and releases vasoactive substances in such a way playing a fundamental role in the basal and dynamic regulation of the cardiac circulation. This results a balance in the normal function of endothelium derived relaxing and contracting substances. Endothelin-1 (and 2, 3) are potent endothelial derived vasoconstrictor substances. The *Coxsackie virus* and certain other microorganisms infect the coronary endothel and are able to release and mobilise endothelins by inhibiting the heparin system. Endothelin-1 induces vasoconstriction, vascular and myocardial hypertrophy, which are independent risk factors for the cardiovascular morbidity and mortality. Decreased heparine level can lead to formation of thrombus and infarct.

Diagnosis: by detecting S-T elevation during the attack of an angina with ECG examination and symptomatically. Continuous ECG monitoring, coronary arteriography, echocardiography, radio nucleotide imaging, angiography, tolerance testing, and x-ray examination could be necessary in certain cases.

Differential diagnosis: by distinguising it from an old myocardial infarction, from S-T elevations caused by drug side effects, and from an acute myocardial small infarction.

Treatment: by administering nitrates, f.i. nitroglycerin, cardiac glycosides, diuretics, beta-adrenergic blocking drugs, by surgery f.i. inplanting coronary shunt, forming coronary bypass or angioplasty. The primary risk factors, f.i. elevated blood pressure and cholesterol levels are promptly to be treated. Smoking is an important risk factor concerning coronary diseases.

RFR method: should only be used during the asymptomatic period controlled by ECG. Detects and eliminates the Coxsackie virus and other viruses!

The most frequent resonances found in case of angina pectoris are: 291, 300-304, 331-346, 360-366, 370-372, 393-397, 403, 407-416, 425-426, 443-450, 471-472, 553-554 kHz

One must to be careful during the measuring and the treatment aswell!

12.8. Sudden Death caused by Coxsackie Viral Cardiac Infection

12.8.1. Sudden Infant's Death Syndrome (SIDS)

Myocarditis can cause heart failure and even sudden death. SIDS is the leading cause of death among infants, aged 1 month to 1 year old. These symptoms are often caused by viruses, being usually the *Coxsackie virus*. The heart failure of infants causes fatigue, i.e. getting tired when eating, sweating due to drinking, rapid breathing, rapid heart rate. This permanent heart damage can be sometimes so severe, that a heart transplant may become to be necessary. Sometimes, however, there is no permanent damage to be experienced.

Treatment: Symptomatically. There is no effective antiviral drug for such viruses.

RFR prevention: detects and eliminates f.i. the *Coxsackie virus*. (See Chapter 5.1.5.2.2.)

12.8.2. Sudden Cardiac Syndrome

Sudden Cardiac Syndrome (i.e. Rapid cardiac death of sportsmen) is associated with an acute myocardial ischemia developing as the result of a *Coxsackie B viral* infection. Viral infections of the heart are significant causes of morbidity and mortality of persons of all ages. *Enteroviruses*, especially the strains of *Coxsackie viruses group B* are thought to be the most common cause of viral myocarditis. The pathogenesis of this kind of viral myocarditis is well described. Once a Coxsackievirus group B infects a heart muscle cell, it produces proteases that can affect dystrophin, a structural protein in the muscle. The cleavage of dystrophin can play a role in the release of the virus from the myocyte. The

innate immunity of cardiac myocytes affecting Coxsackieviruses group B elicits a suppression of cytokine signaling, which is responsible for some cardiac inflammatory changes. Whether this viral myocarditis will progress into a dilated cardiomyopathy depends on the persistence or clearance of the viruses.
These infections can lead to heart muscle damage-associated unstable angina and to acute coronary syndrome (sport death). Myocardial infarctions (MIs, acute heart attacks) have two forms, in which the heart muscles are damaged. These types are named according to the appearance of the electrocardiogram (ECG) as non-ST segment elevation myocardial infarction (NSTEMI) and as ST segment elevation myocardial infarction (STEMI). The acute cardiac syndrome is usually associated with a secondary coronary thrombosis. A cardiac chest pain can also be influenced by anemia, bradycardia and tachycardia. All these can cause nausea and vomiting, as well as shortness of breath. In many cases, the sensation of the patients are "atypical", their pain can be experienced in different ways.
The heart attack is a medical emergency, people experiencing chest pain are advised to alert their emergency medical services in order to get a prompt protection with an external defibrillator which can save their life from a primary ventricular fibrillation. Heart attacks are the cause of death of apparently "healthy" sportsmen and women. Their most important risk factor is an infection caused by Coxsackievirus group B.
Symptoms: chest pain is the most common symptom of the acute myocardial infarction often described as a sensation of tightness, pressure and squeezing. The chest pain due to ischemia of the heart muscle is termed angina pectoris. Pain radiates mostly to the left arm, but may also radiate to the lower jaw, neck, right arm, back, and epigastrium, where it may imitate heartburn. Shortness of breath (dyspnea) occurs when the damage to the heart limits the output of the left ventricle, causing a left ventricular failure and a consequent pulmonary edema. Other symptoms include diaphoresis, weakness, light-headedness, nausea, vomiting and palpitation. Loss of consciousness (caused by inadequate cerebral perfusion and cardiogenic shock) and even sudden death (owing to the development of ventricular fibrillation) can occur in case of myocardial infarctions. This syndrome may even be free of symptoms.
Cardiogenic shock may occur as its **complication** in the acute setting soon after the myocardial infarction or in the weeks following it. Cardiogenic shock is defined as a hemodynamic state in which the heart cannot supply an adequate amount of oxygenated blood for the tissues of the body.
Diagnosis: by ECG, load ECG, coronary angiogram, echocardiogram, by measuring creatine kinase-NB fraction, troponin, glycogen phosphorylase isoenzyme BB of the serum, etc.
Treatment: in case of **acut emergency:** the use of cardiac defibrillator. By administering Glyceryl trinitrate (nitroglycerin) sublingually, antiplatelet drugs, beta blockers, ACE inhibitors, aldosterone antagonist agents, omega-3 fatty acids, etc.
Prevention: by the RFR method, which can detect and eliminate Coxsackie viruses group B.
The most frequent resonances of Coxsackie B virus are: 406-409 kHz
As to the other frequencies, see the Chapter of Coxsackie virus.

12.9. Brugada Syndrome

The clinical manifestations of this genetic predisposed Brugada syndrome (BS) are caused by dysfunctions in the cardiac ion channels. This disorder is characterized by coved or saddle-shaped ST-segment elevations in the leads V_1-V_3 of ECG. It is associated with complete or incomplete right bundle-branch blocks and T-wave inversions, too. Polymorphic ventricular tachyarrhythmias often develop in case of BS, which may lead to ventricular fibrillations, syncopes, cardiac arrest and even to a sudden cardiac death. The BS is in association with the mutation in the gene SCN5A. The gain-of-function of these

SCN5A mutations may also cause long QT syndrome type 3. This mutation is mostly found among people in Laos and Thailand. This genetic predisposition together with an infection by *Coxsackie virus* can cause the Brugada Syndrome. If the action potential heterogeneously occurs in the infected myocardium, it may generate phase 2 reentries which can cause ventricular tachycardia and ventricular fibrillation as well. The large transmural voltage gradients generated by the short action potentials in the epicardial right ventricular outflow are thought to be the origin of the ECG patterns of BS. These specific alterations in the cardiac electrical activity, mainly affect the right ventricle. A prolonged syncope and the aborted cardiac arrest may cause nightmares, seizures, other neurological deficits, hypoxial brain damages, hypertrophic cardiomyopathy, can cause connatally long QT syndrome, aberrant coronary artery origin from the aorta and right ventricular dysplasia.

Diagnosis: by ECG examinations, which show the characteristic alterations mentioned above, hypercalcemia and hyperkalemia, creatine kinase level of the serum, by testing to find the mutation in SCN5A. Echocardiography and MRI should be performed mainly in order to exclude an arrhythmogenic right ventricular dysplasia and also to assess other potential causes of arrhythmias, such as hypertrophic cardiomyopathy, unsuspected myocardial injury, myocarditis, or aberrant coronary artery origins.

Treatment: by the implantation of a cardiac defibrillator, the sole treatment in severe cases. Quinidine can be effective in less serious states.

RFR method: detects and may eliminate the *Coxsackie virus*.

The most frequent resonances are: 407-412 kHz

12.10. Temporal Arteritis (Giant Cell Arteritis)

Temporal arteritis (TA) is a chronic, inflammatory immune disease of the large arteries of the temporal region. The symptoms overlap with those of *polymyalgia rheumatica* syndrome, and some authorities say, that they are two variations of one disease. There are some evidences showing the involving of both the humoral and the cellular immunity in the affected elastic arterial tissue. The elastic lamina will become highly fragmented and can even be absent in some areas of these arteries. The intima is more thickened than can be expected concerning the age of the patient alone. Thrombosis may occur at the locus of the inflammation. The segmental lesions of TA may involve the superficial temporal arteries, too. In case of polymyalgia rheumatica the aorta is frequently involved, aneurysms and dissections are recorded. The external and internal carotid arteries and the vertebral arterial system can also be involved. The inflammation and the occlusion of the ophthalmic and the central retinal arteries may lead to blindness.

TA is a disease of elderly people, affecting men and women nearly equally. The symptomatic involvement of the arteries is frequently preceded by systemic symptoms, including fever, sweats, malaise, fatigue, anorexia and loss of weight, a very high erythrocyte sedimentation rate of the blood is also characteristic. The fever is usually caused by chronic infections. The other group of symptoms include those of polymyalgia rheumatica. The TA belongs to the polymyalgia rheumatica syndrome, according also in my opinion.

Diagnosis: by physical examinations, blood tests, confirmation by biopsy.

Treatment: by administering corticosteroid drugs.

RFR method: detects and may eliminate the pathogen microorganisms.

The most frequent immunomodulating infections are caused by pathogens as follows:

HTLV-1: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz

Mycoplasma fermentans: 442-444, 447-451 kHz

Epstein-Barr Virus: 372, 383 kHz

Other most frequent frequencies found in case of TA are: 334, 339-348, 409, 446-451, 480, 544 kHz

12.11. Kawasaki Syndrome

Kawasaki syndrome is an inflammatory disease primarily affecting children younger than age 5, causing skin rash, fever, generalized enlarged lymph nodes, inflammation of the heart and the joints. This infection may cause a complete thrombotic occlusion of the coronary arteries, aneurysms together with myocardial infarctions which latter can be the immediate cause of death. The developed cardiovascular sequelae of infected children range from asymptomatic coronary artery ectasis or aneurysm formation to giant cell coronary arteritis and aneurysms with thrombosis. Though inflammatory infiltrates can be found also in the myocardium, the pancreas, the kidney and the biliary tract, no significant sequelae do persist in these nonvascular tissues. This syndrome surpasses the rheumatic fever as the most often experienced cause of acquired heart diseases. This self-limiting disease has an infectious etiology, can cause periodic epidemics in certain regions of the world. The characteristic fever, the adenopathy and the eye signs also point to infection. The most frequent causative pathological agents are *Coxsackie virus A 1-14, 16, 21-22, 24, ECHO viruses 1-14, 19-20, 22, 25, 28, 30, Human T-cell Lymphotropic Virus* and *Mycoplasma.*

The development of myocarditis, a congestive heart failure, pericarditis with pericardial effusion, mitral or aortic insufficiency and dysrhythmias can be observed in the early phase of the disease. Later on the development of aneurysms can be predictive by the severity of the disease. The disease is more common among boys.

The subacute phase of the illness is characterized by a persistent irritability, anorexia, a conjunctival inflammation, fever, thrombocytosis, desquamation of the fingertips and toes. Aneurysm formation can also occur during this time. Children are at the greatest risk of sudden death in this phase of the disease.

The chronic phase can show cardiac complications. Its duration has a lifetime significance as the aneurysm formed in childhood may rupture in adulthood.

Diagnosis: by clinical laboratory examinations, ECG, EEG, MRI or PETscan.

Treatment: by administering gammaglobulins, anti-inflammatory agents, antiplatelet agents and surgery.

RFR method: detects and may eliminate the pathogen microorganisms.

The most frequent resonances of the Coxsackie viruses are: 294-322, 407-408, 409-412 kHz

The most frequent resonances of the ECHO viruses are: 317-319, 369, 379, 401-404, 470-471, 526 kHz

The most frequent resonances of the Mycoplasmas are: 442-451, 493-495 kHz

The most frequent resonances of the Human T-cell Lymphotropic Viruses are: 311-321, 339, 354, 359, 365, 370-376, 428, 482, 523, 526-530 kHz

The most frequent resonances of the Epstein-Barr Virus are: 372-383, 518-519 kHz

The most frequent resonances of the rarely found Cytomegalovirus are: 305, 348-350, 408-410, 545-550 kHz

12.12. Churg-Strauss Syndrome

Systemic vasculitis, a group of vasculitis diseases can affect several organ systems and cause severe disabilities.

Churg-Strauss syndrome is characterized by a systemic autoimmune vasculitis of the medium and small vessels, leading to their necrosis. It involves mainly the blood vessels of the lungs, the gastrointestinal system and the peripheral nerves, but may also affect the heart, the skin and the kidneys. It is a rare, non-inheritable, non-transmittable and often miss-diagnosed disease. Churg-Strauss syndrome was formerly sorted to the systemic vasculitis group of Polyarteritis nodosa (PAN) because of their similar granulomatous

histopathologic alterations. The symptoms of Churg-Strauss syndrome include fatigue, weakness, fever, arthralgias, abdominal pain, hypertension, renal insufficiency and neurologic dysfunctions as well, this latter depends on the stage of the disease. The disease has three distinct stages.

The first stage usually involves the sinuses and is characterized by the onset or the worsening of pre-existing allergies of the air-ways.

The second stage is characterized by the symptoms of an acute asthma-like chronic disease with granulomatous inflammation of the respiratory tracts and by a necrotizing, pauci-immune glomerulonephritis.

The third and final stage can involve various organ systems. This stage is by far the most painful and life threatening. The patients suffer from severe nerve pains in their legs, arms and hands. Purple exanthems can appear on the skin, sores in the mouth or the nose. Besides the lungs and the kidneys the disease can also affect the heart and the liver. Reduced renal function, proteinuria, gastrointestinal hemorrhages and infarctions, pancreatitis, the involvement of the central nervous system and a cardiomyopathy can all be present.

Having the symptoms of the first and second stages people can live for many years before progressing to the final stage.

Infections caused by *Herpes Virus*, *Coxsackie virus*, *Borrelia Burgdorferi sensu lato*, *Mycoplasmas* and *HTLV* are usually present in this type of systemic vasculitis disease.

Diagnosis: symptomatically, by diagnostic markers including eosinophil granulocytes and granulomas in the affected tissues, by ANCA antibody testing, etc.

Differential diagnosis: by distinguishing it from other vasculitis illnesses: f.i. Wegener's granulomatosis, other CNS vasculitis diseases, Henoch-Schönlein purpura, essential cryoglobulinemic vasculitis etc.

Treatment: by administering glucocorticoids and other immunosuppressive drugs f.i. azathioprine, methotrexate and cyclophosphamide. In many cases the disease can be brought into a type of chemical remission by drug therapy, but the disease will be chronic. The treatment can only be symptomatic and can not heal this disease.

RFR method: detects and may eliminate all pathogen microorganisms.

The most frequent resonances are: 290-294, 343-345, 353-363, 370-376, 378-388, 416-420, 442-451, 493-495 kHz

12.13. Polyarteritis Nodosa

Polyarteritis nodosa (PAN), is a rare systemic, focal or segmented immune vasculitis of the small-sized and medium-sized arteries. Though all organs can be affected, PAN involves most of all the kidney, the peripheral nerves, the joints, the gastrointestinal (GI) tract and the skin. The manifestations of the disease are diverse and complex, ranging from a benign cutaneous form to a severe disseminated form. PAN is also observed as a complication of *Hepatitis B*, *Hepatitis C* and other *viral* infections together with *mycoplasma*, which can provoke the autoimmune process. This systemic vasculitis characterized by necrotizing inflammatory lesions of the small-sized and medium-sized muscular-type arteries, preferentially at the vessel bifurcations, can result in microaneurysm formations, thrombosis, aneurysmal ruptures with hemorrhages and infarctions.

Microscopic polyangiitis (MPA) causes necrotizing vasculitis with a few or no immune deposits affecting the small vessels (capillaries, venules, arterioles). Various infections, most frequently *mycoplasma* and *superantigens* have been suspected as causes of a persistent antigen, stimulus subsequently leading to immune complex formations. Mycoplasma antigens are adsorbed by the cell walls. The resultant immune complexes activate the complement cascade, which activate and attract neutrophils. Antineutrophil cytoplasmic antibodies (ANCA) appear to play a significant role in the damaging of endothelia.

Symptoms of PAN: despite the often occuring intense *myalgias*, the creatine kinase levels in the blood remain usually within the values of references. *Painful skin ulcerations, livedo reticularis, ischemia* and *gangrene* are the most common skin manifestations of PAN. A non deforming asymmetric *arthritis*, usually involving the larger joints of the lower extremities is in the early stage of the disease rather common. Mesenteric thrombosis and ischemia causing consequent intractable *abdominal pain. Loss of weight*, infarction, bowel perforation, hemorrhages, pancreatitis, appendicitis and cholecystitis can also come about. PAN does always cause vascular nephropathy, in contrast to MPA, which causes glomerulonephritis. This nephropathy can lead to profound *hypertension* and *oliguric renal failure*. The acute necrotizing renal arteritis in case of PAN can lead to thrombosis and renal infarction causing severe *costophrenic pain and tenderness*.
Coronary arteritis, the signs and symptoms of which may include hypertension (most common), *tachycardia* unrelated to fever, *congestive heart failures, cardiomegaly, pericardial friction rubs*, and *arrhythmias*. Multiple, usually asymmetric, sensory and motorous mononeuropathies do often occur. The sciatic nerve is mostly affected. The onset of the sensory neuropathy may be sudden, accompanied by *pain* and *paresthesias* radiating in the distribution of the affected peripheral nerve, followed by motor deficits of the same peripheral nerve within hours or days.
A **secondary PAN-like syndrome** may be associated with systemic autoimmune diseases such as rheumatoid arthritis and Sjögren's Syndrome. Likewise, also certain *viral* infections are associated with this c-PAN. Polyarteritis may occur at any time among patients who are HbsAg positive. *Other viral infections* including *HIV, CMV, parvovirus B-19, HTLV-1* and *HCV* as well as bacterial infections, such as *Streptococcus* and *Mycoplasma* species or seldom *Chlamydia*, may also be associated with arteritis, occasionally even with PAN, while, in contrast to *HBV*, these viruses are associated with many an other type of vasculitides, too. Hairy cell leukemia can be joined by PAN, though, in most of these cases, the patients are also HBV-positive. Co-infections are frequently detectable with *CMV, EBV, HSV1 and 2, Herpes Zoster and Borrelia B.s.l.*
Diagnosis: symptomatically, by nonspecific inflammatory changes, such as normochromic anemia, polymorphonuclear leucocytosis, thrombocytosis and eosinophilia, by elevated erythrocyte sedimentation rate (usually >60 mm/h), CRP, ANA, Cryoglobulins, depressed serum C3 and C4 levels, Rheumatoid factor, p-ANCA, c-ANCA etc.
Treatment: conventionally and symptomatically, as there is no specific therapy. By administering high doses of corticosteroids and immunosuppressive drugs, such as Cyclophosphamide.
RFR method: detects and may eliminate the various pathogen microorganisms.
The most frequent resonances are: 324, 370-376, 384, 392-402, 442-451, 475-480-484, 508, 513-518, 528 and 534 kHz
As to the other frequencies, see the referring chapters of *Hepatitis viruses, Cytomegalovirus, Epstein-Barr Virus, HTLV*, etc.

12.14. Leukocytoclastic Vasculitis

Leukocytoclastic vasculitis (LCV), also named Hypersensitivity vasculitis or Hypersensitivity angiitis, is caused by a hypersensitivity reaction to a known drug, auto-antigens or to infectious agents, f.i. bacteria. Though many possible causes can be responsible for this condition, the exact cause is not found in regard to 50% of the patients. Immune complexes are present in the vessel wall, attracting polymorphonuclear leukocytes, which, in turn, release tissue-degrading substances leading to inflammatory processes. LCV is a histopathologic term commonly used to denote a small-vessel vasculitis. The disorder may be localized to the wall of the skin vessels, or may be manifested in other organs, too. These other affected organs are the joints, the gastrointestinal tract and the kidneys. If there is no internal involvement, the prognosis is

usually good. The disorder can be acute or chronic. Formerly, circulating immune complexes were believed to be the cause of LCV. Though immune complexes are involved in the pathogenesis of LCV, autoantibodies, such as antineutrophil cytoplasmic antibody (ANCA), other inflammatory mediators and local factors, involving the endothelial cells and adhesion molecules play all an important role in the pathogenesis of the illness. *Mycoplasma species, HTLV, beta-hemolytic streptococci, Borrelia B.sensu lato, Hepatitis B and C viruses and HIV* may all be co-factors in the development of this disease.

Symptoms include itching, burning, pain, but there may also be asymptomatic lesions, presence or absence of fever, arthralgia, arthritis, myalgia, abdominal pain, diarrhea, hematochezia, cough, hemoptysis, sinusitis, paresthesia, weakness and hematuria. Vasculitis of the skin may occur in the absence of any systemic involvement. It may only be an eruption, or it may occur in association with collagen vascular disorders, paraproteinemia, can be caused by certain foods, by medications, as well as by various infections, or, rarely, even by malignancy.

LCV symptoms can be connected with inflammatory bowel diseases and collagen vascular disorders, particularly rheumatoid arthritis, lupus erythematosus, or Sjögren's Syndrome as well.

LCV lesions are usually maculopapulous exanthems or palpable purpura, being round, 1-3 mm in diameter, forming sometimes plaques, ulcerating in certain cases. Palpable purpura is observed most often on the legs, but any surface of the body can be involved. Sometimes, the purpuric lesions are barely palpable. Urticarial lesions may also occur concerning some patients, this type of lesions may precede the purpuric lesions.

Patients with hypocomplementemic urticarial vasculitis often suffer from chronic obstructive pulmonary diseases, so that careful examinations of the heart and the lungs are needed.

Henoch-Schönlein purpura (HSP), this postinfectious systemic hypersensitivity vasculitis affecting children and jung adults, occuring together with arthralgia, gastrointestinal symptoms f.i. abdominal pain and glomerulonephritis mostly with hematuria, shows IgA type immuncomplex deposits in the wall of the small vessels of the skin and the kidney. The palpable purpura typically appears on the legs and the buttocks, but may also be found on the arms, the face and the trunk. Subcutaneous edema of the hands, the feet and the scalp often come about. The abdominal spasms are often accompanied by nausea, vomiting, constipation and diarrhea with blood and mucus in the stools. HSP and IgA nephropathy are related disorders. Both illnesses have elevated serum IgA levels and identical findings on renal biopsy; though IgA nephropathy involves almost exclusively young adults and affects predominantly the kidneys. HSP can develop following infections caused by *beta-hemolytic streptococci, Hepatitis B, Herpes Simplex Virus, Parvovirus B19, Coxsackie virus, ECHO virus, Adenovirus, Helicobacter pylori, measles, mumps, rubella, HTLV, Mycoplasmas.* Non infective agents, such as *drugs* including antibiotics, f.i. vancomycin and cefuroxime, ACE inhibitors, f.i. enalapril and captopril, anti-inflammatory drugs, f.i. diclofenac, and other drugs, f.i. ranitidine, streptokinase, etc. can also cause Henoch-Schönlein purpura.

LCV may be associated with *bacterial endocarditis* (beta-hemolyticus streptococci), too.

Diagnosis: symptomatically, by histologic foundings of leukocytoclastic vasculitis, most prominently in postcapillary venules. By serologic studies. ANA, ANCA and rheumatoid factor examinations should be maid concerning patients with no obvious cause of their illness.

Treatment: By administering high doses of corticosteroids with or without immunosuppressive agents (f.i. cyclophosphamide, azathioprine, methotrexate and colchicine) as there is no specific therapy. Chronic forms, that primarily involve the skin should be treated with nontoxic modalities whenever possible, avoiding the use of systemic

corticosteroids, immunosuppressive agents, or both. By administering NSAIDs in the treatment of their pain.
RFR method: detects and may eliminate all pathogen microorganisms.
In case of LCV the most important is the elimination of Mycoplasmas (frequently the Mycoplasma fermentans) and HTLV, which pathogens provoke the hypersensitivity and the immune processes.
The most important resonances are: 293, 324, 336, 341, 364-367, 370-376, 384, 392, 414-420, 423, 432-433, 442-451, 454-456, 475-479, 482, 487-493, 493-497, 561 kHz
The most frequent resonances found in case of Henoch-Schönlein purpura are: 303, 306-321, 324, 360-375, 409-413, 416-420, 442-451, 493-495, 576 kHz
The RFR method of LCV can be the most effective form of all therapies.

12.15. Hypertension

High blood pressure, named also „the silent killer", is generally a symptomless condition in which the abnormally high pressure in the arteries increases the risk of problems such as stroke, heart failure, aneurysm, heart attack, retinopathy and kidney damage. Patients with elevation of arterial pressure show usually no symptoms and their blood pressure abnormality often arouses attention only incidentally, f.i. at their periodic physical examinations (during military service, life insurance, or in case of an infection). As hypertension can cause secondarily damages of certain organs and reduce the span of life, it has to be treated. For many people, the expression hypertension means excessive tensions, nervousness and stress, but the medical term hypertension means a condition of elevated blood pressure, regardless of its cause.
In case of adult people high blood pressure is defined as if the systolic pressure at rest averages 140 mm Hg or more, and the diastolic pressure is more than 90 mm Hg. In case of almost everybody the blood pressure increases with age.
The renin- angiotensin- aldosterone system regulates the blood pressure. The decrease in blood pressure causes the releasing of renin. Renin, in turn, activates angiotensin, a hormone causing constriction of the muscular walls of the arterioles, increasing thus the blood pressure. Angiotensin triggers also the release of aldosterone hormones in the adrenal gland, which causes the retaining of sodium salts and the excretion of potassium in the kidneys. As sodium retains water, the blood volume will expand and the blood pressure will be increasing.
The acute hypertensive crisis, a very dangerous condition, can cause hypertensive encephalopathy and requires an emergency medical treatment.
Certain causes of secondary hypertension are:
Kidney diseases:
Renal artery stenosis
Pyelonephritis
Glomerulonephritis
Kidney tumors
Polycystic kidney disease
Injury to the kidneys
Radiation therapy affecting the kidneys
Hormonal disorders:
Hyperaldosteronism
Cushing's syndrome
Pheochromocytoma
Acromegaly
Adrenogenital syndrome
Drugs:
Oral contraceptives

Corticosteroids
Cyclosporine
Erythropoetin
Cocaine
Alcohol abuse
Licorice
Nervous system:
Psyhogenic stress
Chronic stress
Diencephalic syndrome
Familial dysautonomy
Poliomyelitis
Polyneuritis
Increased intracranial pressure
Spinal cord section
Other causes being:
Pregnancy complicated by preeclampsia
Coarctation of the Aorta
Acute intermittent porphyria
Acute poisoning, f.i. lead poisoning

In cases of hypertension appearing after an asymptomatic latent period, the clinical manifestations, which reflect the underlying pathologic sequelae of the hypertensive state, usually become apparent. The effects of the cardiac, renal and central nervous system, developing due to the accelerated vascular damages, are the most prominent, and, if unaltered by therapy, they often ultimately result in a symptomatic illness causing even death. I will not discuss all cases of hypertension in my book, I shall only pick up one among the many secondary hypertensions, the *poststreptococcal glomerulonephritis*. The hypertension associated with poststreptococcal glomerulonephritis is mostly related to the overloading of fluids and, additionally, perhaps to the effect of the increased activity of the renin-angiotensin system. Fluid removal and, if needed, an antihypertensive medication should be used in order to control the blood pressure elevation. During the acute phase of the disease bed rest is advisable, though during convalescence normal activity may be undertaken. Poststreptococcal glomerulonephritis may occur after a streptococcal pharyngitis or tonsillitis or even after a streptococcal pyodermal skin infection. The latent period between the streptococcal skin infection and the glomerulonephritis is about 2-4 weeks. Several specific nephritogenic serotypes may cause pharyngitis-associated glomerulonephritis, while a hypertension of a pyoderma-associated nephritis origin may be induced by streptococcal infections of other *serotypes* (*A2*).
A persistent fever can warn of either a continued streptococcal infection or a systemic disease associated with an acute glomerulonephritis, f.i. systemic lupus erythematosus or polyarteritis nodosa. Hypertension is to be found in approximately 60 percent of the patients suffering from acute poststreptococcal glomerulonephritis. In more severe cases the hypertension may be associated with encephalopathy and retinopathy including hemorrhages, exudates, and papilledema. Severe headaches, somnolence, nose-bleeding, dizziness, a flushed face and even convulsions may be the manifestations of this encephalopathy. If a patient has a severe, long-standing and untreated high blood pressure, its symptoms, such as headache, fatigue, nausea, vomiting, shortness of breath, restlessness and a blurred vision occur caused by the damage of the brain, the eyes, the heart and the kidneys.
Therapy: of the poststreptococcal glomerulonephritis (by administering antibiotics, steroids or other antiinflammatory drugs), see Chapter 15. Hypertension treatment with

angiotensin converting enzyme inhibitors and angiotensin II blockers in order to lessen the blood pressure by dilating the arteries. They are particularly useful in case of whites, young people, people with heart failure, people with proteinuria caused by chronic kidney diseases. Other antihypertensive drugs f.i. thiazides, betaadrenergic blockers, calcium antagonists, direct vasodilatator drugs can also be useful.

RFR method: eliminates every *streptococcus* strains, as well as the *Herpes viruses*, the *Nanobacteria* and the *Chlamydia*! Steroid is very important in the glomerulonephritis treatment, because of the autoimmune component of the pathogenesis of this illness. The autoimmune inflammation can cause irreversible damages in the kidneys, though the corticosteroid drugs, as a side effect, can increase the blood pressure!

The most frequent resonance frequencies of the streptococcus family are: 313-321, 360-375 kHz

As to the other streptococcus resonance frequencies, see Chapter 6.16.2.2.

The most frequent resonances found in case of hypertension are: 370-373, 383-384, 394-403, 408-412, 442-444, 447-451 kHz

The antibiotic treatment can not always kill the streptococcus species (therefore bacterial culture resistance examinations are to be advised). This RFR method doesn't decrease the high blood pressure directly and has no effect on the autoimmune process. It is advised to control the streptococcus resonances for a long time by measuring. The RFR method is very effective in the eliminating of the *Streptococcus*, *Herpes Simplex Virus-1* and *Nanobacteria*. After an effective usage of the RFR method the hypertension may be temporarily increased. If the eliminating is definitive the blood pressure will decrease (see Nanobacteria).

12.16. Atherosclerosis

Atherosclerosis, a form of arterial degenerations, is the major cause of death all over the world, but most often in industrialized societies. Arteriosclerosis is the term for the thickening and the induration of the arterial wall. A certain type of arteriosclerosis is the atherosclerosis, a disorder found in most cases of arteriosclerotic heart diseases and of coronary artery diseases, which also plays a major role in the pathology of the cerebrovascular diseases. The mortality of atherosclerosis is the very first of all other causes of death in Europe and the USA. Arteriolosclerosis is a disorder representing alterations in the small arteries, which are particularly common in case of hypertension. A lesser degree of sustained hypertension causes characteristically the hyalinization of arterioles; while a more severe or malignant hypertension produces typically fibrous and elastic hyperplasia and, moreover, even the necrosis of the media and intima. Atherosclerosis is a process, in which fatty materials and, later on, calcium accumulates under the inner lining of the arterial wall. Atherosclerosis can affect the arteries of the brain, the heart, the kidneys, the eye and the arteries of other organs, as well as the arteries of the muscles of arms and legs. If atherosclerosis develops in the arteries supplying the brain, a stroke may occur; but if developing in the coronary arteries, a heart attack may occur. In case of atherosclerosis modified monocytes migrate from the bloodstream into the wall of the artery and will be transformed into cells accumulating fatty materials. A thickening atherosclerotic plaque is filled with soft, cheese-like substances consisting of various fatty materials, mostly cholesterols, modified smooth muscle cells and connective tissue cells. Atherosclerotic arteries lose their elasticity, and while the atheromas grow, the arteries will narrow. Later on the atheromas collect calcium deposits, become brittle and may rupture. This ruptured atheroma may also spill its fatty contents and trigger the formation of a blood thrombus. The clot may further narrow or even occlude the artery, or may be detached and float downstream, where it can cause an occlusion.

The ischemic heart disease (synonymous with coronary heart disease and arteriosclerotic heart disease) is a useful indicator of atherosclerosis. A sudden closure of a partially

compromised vessel by a subsequently lysed small thrombus and embolus, as well as a spasm can cause ventricular fibrillations, but none of these need precede in a fatal arrhythmia.
Cerebrovascular diseases are only occasionally associated with atherosclerosis. These cerebrovascular diseases include cerebral hemorrhage and cerebral thrombosis. Cerebral thrombosis, cerebral infarction or a softening without any embolus is usually caused by atherosclerosis.
Atherosclerosis is a multicausal condition, its two major factors being the hyperlipidemia and the hypertension. Hypertension and hyperlipidemia together give a higher incidence of premature human ischemic heart diseases, than if alone. Hypertension and hyperlipidemia appear to be independent from each other and to have an additive effect on the accelerating of atherosclerosis. This fact itself does not explain the etiology of atherosclerosis and the variability in the susceptibility to complications or in the extent of lesions among individuals with one or both of these major risk factors.
Endothelin-1 can induce local and systemic vasoconstriction, vascular and myocardial hypertrophy, which are independent risk factors of the cardiovascular morbidity and mortality. Alterations of the endothelin levels have been observed in a number of cardiovascular and renal disorders.
Nanobacterium, a pathogen microorganism can cause local and systemic high and hyperactive endothelin levels and pathophysiologic vascular contractions. Hypercholesterolemia has a stimulative effect on the endothelin system, so that the levels of LDL and the activity of the endothelial endothelin receptors will be increased. The endothelin receptor system plays a role in a number of vascular diseases, f.i. in the endothelin-1-induced vasoconstriction of the coronary arteries. This pathogen *nanobacterium* has an important effect on the endothelin receptor, too. Endothelin-1 works as a proinflammatory mediator in case of various diseases, as well as in atheriosclerosis.
The physiological role of endothelin 1-3 is its vasoconstrictor effect inhibiting posttraumatic bleeding. Nanobacterium is able to mobilize endothelin 1-3 causing thus peripheral vasoconstriction. Nanobacteria can build up calcium carbonate apatite membranes with rough surfaces along the endothel cells provoking thus the aggregation of thrombocytes. The organism in turn protects against this process with cholesterin deposits. Neither can this new surface produce heparin, so that the danger of developing thrombosis does remain. In this case an adequate bloodflow can be ensured only with an enhanced bloodpressure which causes thus new problems.
After getting infected with nanobacteria the atherosclerotic process mentioned above develops but very slowly, lasting for 30-50 years, but in case of a coinfection with *Mycoplasma fermentans* inhibiting the immune responses, nanobacteria are apt to grow faster. Other infections caused by immunosuppressive microorganisms, such as *HTLVs, EBV,* etc can have similar harmful effects.
Inflammatory mechanisms play an important role in the developing of vascular diseases and the inflammatory plasma markers correlate with the severity of the diseases. The nanobacteria and *certain viruses* can play an important role in the vascular inflammatory processes. Nanobacteria are heparin inhibitors. The decrease of the heparin level can increase the aggregation of thrombocytes and the risk of thrombosis. Cholesterol and triglycerides are the clinically most important lipid components of the plasma. Together with phospholipids and certain proteins (apolipoproteins) they compose the four major classes of plasma lipoproteins. The glycerides and the cholesterols are transported in large particles, i.e. chylomicrons from the small intestine into the blood via the lymphatics. Very low-density lipoproteins (VLDL) carry endogenously synthesized triglycerides, to be removed by the muscles, the heart, the adipose tissues and other tissues of the body as well. The major remnants of VLDL metabolism are the low-density lipoproteins (LDL). LDL are catabolized mostly by hepatocytes. The high-density lipoprotein (HDL) contains

phospholipid and cholesterol complexes with apolipoproteins, the bulks of which differ from those of VLDL and LDL. HDLs are involved in the metabolism of VLDLs and in this enzymatic reaction cholesterol will be esterified in plasma. The age-related increase in cholesterol are mainly associated with the rise in the LDL concentrations, and the increase in triglycerides with the rise in VLDL concentrations. In case of an ischemic heart disease and stroke, the concentrations of LDL or LDL+VLDL are increased similarly to cholesterol. The relation of triglycerides and VLDL to these diseases is in connection with the rise in cholesterol and in VLDL. There are 5 types of hyperlipoproteinemia occuring in about an equal frequency concerning patients suffering from premature ischemic heart diseases.

The other important risk factors in the development of atherosclerosis are smoking, abnormal glucose tolerance, physical inactivity, obesity, genetic factors, infections caused by *Coxsackie virus, Cytomegalovirus* and *other viruses,* stress factors, and *certain pathogens* provoking high blood pressures.

Diagnosis: by serum analysis and lipid analysis, by blood tension measuring, ECG, EEG, echocardiography, by blood flow measuring.

Treatment: should be complex, affecting all causative factors.

RFR method can be effective in case of inflammatory processes, where the pathogen microorganisms play an important role in the developing of the disease. The atherosclerotic process does only slowly develop during the patient's life, so that the elimination of nanobacteria with the RFR method can from time to time slecken or prevent this process.

RFR method detects the viral components and is able to eliminate the viruses.

The most frequent resonances found in atherosclerosis are: 294-298, 305-310, 324-326, 336-349, 370-387, 397, 407-414, 442-451, 512, 543, 555-558, 560-569 kHz

The RFR method can only be supplementary to the conventional medical therapy.

12.17. Diabetic Angiopathy

Diabetic angiopathy is a disease of the blood vessels, characterized by alterations in the walls of the blood vessels that interfere with normal blood flow. The illness can affect the large blood vessels causing macroangiopathy, and the small blood vessels, causing microangiopathy. Interestingly, the small vessel disease can be minimized by tight blood glucose controls, but the large vessel disease can not be influenced by tight blood glucose controls.

These diabetic small vessel diseases can cause proliferative retinopathy and macular edema, which can lead to severe vision loss or blindness; peripheral neuropathy, particularly combined with damaged blood vessels, can give possibility to get severe infections and gangrene, sometimes requiring even limb amputation. The diabetic nephropathy can lead to renal failure.

The complications of diabetic large vessel diseases include ischemic heart diseases, affecting the large and the small vessels as well, stroke and peripheral vascular diseases, with the risk of amputation.

Diabetes mellitus is the most often cause of adult kidney failures worldwide. It is also the most common cause of amputation, usually of the toes and the feet, which must be made because of gangrene, the result of this peripheral vascular disease. The diabetic retinal damage (resulted from microangiopathy) is the most common cause of blindness among non-elderly adults. A number of studies have found that those with diabetes are more at risk for dry eye syndrome. Advanced glycosylation end products are believed to play a role in the pathogenesis of angiopathy resulting from diabetes mellitus.

Diabetes mellitus in frequently combined with hypertension. This chronic hypertension with small vessel vasculopathy is characterized by lipohyalinosis, fibrinoid necrosis and by the development of Charcot-Bouchard ancurysms, affecting the penetrating arteries mostly

of the brainstem, but also the lenticulostriates, the thalamoperforators, the paramedian branches of the basilar artery, the superior cerebellar arteries and the anterior inferior cerebellar arteries. *Nanobacteria* can cause calcification in the heart, the aorta, and the large blood vessels. Nanobacterial mineral formation is a specific biogenic process. The minerals grow directly on the nanobacteria, forming parts of their envelop. These bacteria live on the vascular endothel membrane and can narrow the blood vessel lumen.
Infections caused by *Mycoplasma fermentans* and *other Mycoplasma species* can often be found among people suffering from diabetes mellitus. *Nanobacteria*, *Mycoplasma*, and a special *virus* cause the blood vessel damage in diabetes. Chronic inflammatory responses are to be seen in the areas where these microorganisms are present. *Nanobacteria*, *Mycoplasma* and *virus* components cause the diabetic macroangiopathy and microangiopathy. The diabetic angiopathy is a proliferative and inflammatory blood vessel disease.
Diagnosis: by examination of retina, renal function tests, ECG, angiographies, peripherial limb tonometry, biopsy and other functional tests.
Treatment: symptomatically.
RFR method: detects the microorganisms and eliminates them!
The frequencies of the Nanobacterium: 294-298, 324-325, 336-345, 466-469, 372-387, 556-558, 560-568 kHz
The frequencies of the Mycoplasma fermentans: 312, 329-330, 353, 361, 404, 442-451, 493-495, 505 kHz
Other frequencies see in Chapter 24.1.

12.18. Antiphospholipid Syndrome and Mycoplasma

The Antiphospholipid syndrome (APS) is characterized by a variety of hematologic and vaso-occlusive manifestations, and is associated with antibodies to negatively charged phospholipids. The alterations of the APS include hemolytic anemia, thrombocytopenia, venous and arterial occlusions, livedo reticularis, pulmonary manifestations, recurrent fetal losses, neurologic manifestations (stroke, transverse myelitis, Guillain-Barré syndrome) as well as a positive Coombs test, anticardiolipin antibodies and lupus anticoagulant activity of the blood.
The recurrent venous or arterial thrombosis and the fetal losses associated with typical laboratory abnormalities are accompanied by persistently elevated levels of antibodies directed against membrane anionic phospholipids (cardiolipin and phosphatidylserine) as well as against their associated plasma proteins, predominantly the beta-2 glycoprotein I (apolipoprotein H) and by the presence of circulating anticoagulants. This disorder may also be associated with SLE and other autoimmune diseases.
There occurs an alteration of the homeostatic regulation of the blood coagulation; though, the mechanisms of the thrombosis are not yet exactly defined. A certain hypothesis postulates a defect in the cellular apoptosis, exposing membrane phospholipids to the binding of various coagulation proteins. If once bound, a phospholipid-protein complex is formed, a neoepitope will be uncovered and will subsequently become the target of autoantibodies.
It seems, as if the antiphospholipid syndrome were connected with mycoplasmal infections, so that f.i. the *Mycoplasma fermentans*, *Mycoplasma penetrans* and *Mycoplasma pneumoniae* were playing an important role in the development of APS.
A cerebrovascular accident is usually thrombotic but can also be embolic, f.i. in case of Libman-Sacks endocarditis in SLE. The cardiac valvular disease may be severe enough to require a valve replacement. The APS may also contribute to an increased frequency of

myocardial infarctions. The recurrently occuring pulmonary emboli and thrombosis can lead to a life-threatening pulmonary hypertension.
Though they are not included in the classification criteria, the following clinical features, may be attributed to the APS:
Nonthrombotic neurologic symptoms, such as migraine headaches, chorea, seizures, transverse myelitis, Guillain-Barré syndrome and sometimes dementia;
Heart murmur or cardiac valvular vegetations;
Hematologic abnormalities, such as thrombocytopenia and hemolytic anemia;
Livedo reticularis;
Unexplainable adrenal insufficiency;
Avascular necrosis of bone in the absence of other risk factors;
Pulmonary hypertension.
Unlike inflammatory autoimmune diseases, the histologic studies of the skin or the other involved tissues reveal a noninflammatory, bland thrombosis without any signs of perivascular inflammations or vasculitis. Similarly, the biopsy samples from the affected kidneys demonstrate glomerular and small arterial microthrombi.
A catastrophic APS is a serious and often-fatal manifestation characterized by multiorgan infarctions occuring over days to weeks.
Due to these infections the immune response will decrease and the infections will cause a chronic process.
Diagnosis: by autoantibody examinations, such as anticardiolipin antibodies, anti–beta-2 glycoprotein I antibodies, by measuring the activated partial thromboplastin time (aPTT),
By LA tests such as dilute Russell viper venom time (DRVVT), CBC count (thrombocytopenia, Coombs-positive hemolytic anemia), by Mycoplasma serology, and by CT scanning or MRI of the brain (CVA), the chest (pulmonary embolism), or the abdomen (Budd-Chiari syndrome). Doppler ultrasound studies are recommended for a possible detection of DVT.
Treatment: the therapeutic agents are based on their anticoagulant properties and their benefits are to be weighed carefully because of their significant risks. A life-long administration of a moderately high-intensity warfarin is the standard therapy regarding the recurrent thrombotic events. Heparin therapy is often administered. For obstetric patients with APS, the standard therapy has to be the subcutaneously administered unfractionated heparin or a LMWH and aspirin in low-doses.
RFR method: detects and may eliminate the Mycoplasma!
As to the resonance frequencies of the Mycoplasma, see Chapter 6.18.
Check these resonance frequencies, treat and give anticoagulant therapy for a long time.

12.19. Intravascular Coagulation Syndrome

Intravascular coagulation syndrome (ICS) is a condition, characterized by the development of many a thrombus in the vascular system. The pathogenesis, the diagnosis and the management of this disease are extremely complex. The principal target protein of the ICS is the fibrinogen. The principal enzymes, attacking the fibrinogen, are thrombins and plasmins. Thrombin cleaves two bonds, releasing fibrinopeptides A and B from the amino-terminal ends of the alpha and beta chains of fibrinogen. One peptide is cleaved more rapidly, and following its release, the fibrin molecules will polymerize forming a visible coagulum. The ICS is characterized by quantitative and qualitative changes of the plasma fibrinogen.
The normal concentration of fibrinogen reflects a balance between its synthesis and catabolism. The synthesis happens in the liver and normally can be increased up to tenfold.
The reticuloendothelial system, particularly in the liver, removes the fibrin and certain activated coagulation factors from the circulation. The suppression of reticuloendothelial

activity enhances the susceptibility to intravascular coagulation. The inhibition of fibrinolysis promotes the occurrence of thrombosis of organs.
The most naturally acting circulating anticoagulants are actually antibodies to specific clotting factors. Heparin administration will prevent the changes of coagulation. Some different bacterial and viral infections inhibit the production of heparin in the vascular intima. The bacterial protease enzymes may catalyse the fibrinogen-fibrin transformation.
The clinical symptoms will depend on the localization of the thrombus and on the fact whether this agglutination will be a process localized or generalized. Many different symptoms can be developed, depending on the fact, which of the arteria or vena is affected, and to what degree the blood flow has been decreased.
The ICS can develop in the cardiovascular and the pulmonary system, the brain circulation system, in the vessels of the eye and often in the limb arteria or the limb vena such as in the vena femoralis.
The most important form of the ICS is the stroke. In case of stroke there develops a cerebral infarction caused by the lack of blood flow and by an insufficient oxygen content and energy supply of the brain. A stroke can be either ischemic or hemorrhagic. In case of an ischemic stroke, the blood supply to a part of the brain is cut off, either due to atherosclerosis or due to a blood clot blocking a blood vessel. In case of a hemorrhagic stroke, a blood vessel burst, preventing a normal flow, allowing the blood to leak into an area of the brain to destroy it.
A stroke can occur if an inflammation or an infection narrows the blood vessel leading to the brain. *Nanobacteria* can reduce the blood flow to the brain in the capillary and sclerotic blood vessels, releasing endothelin 1-3, substances inhibiting or stopping the capillary circulation. Strokes usually begin suddenly, develop rapidly, and cause brain damage within minutes. Less commonly, strokes may continue to worsen lasting from several hours to a day or two, because of the steadily enlarging necrotic area of the brain tissue. The progression is usually, but not always, interrupted by somewhat stable periods during which the area temporarily stops to get enlarged or some improvement can be experienced. Many different symptoms can occur, depending on which part of the brain is affected. Strokes can cause edema or swelling in the brain. This swelling is particularly dangerous as the skull allows but little space for expansion. The resulting pressure can damage the brain tissue causing neurological problems which can get worse even if the stroke itself doesn't spread any more.
The Intravascular Coagulation Syndrome can cause a shock reaction and the intravascular coagulation may be generalized.
Sepsis is one of the most common disorders associated with disseminated intravascular coagulation. Classically, the sepsis is caused by gram-negative microorganisms such as *meningococcus*, but the syndrome may be encountered in case of infection caused by *gram positive bacteria, plasmodia* and by *viruses,* f.i. by *EBV.*
Purpura fulminans is an acute eruptive skin disorder occuring most often among children after a bacterial or viral infection. Following a latent period after the infection, purple purpuric lesions, possibly with a necrotic center occur on the limbs, the back, the buttocks, or the face. Laboratory signs of a disseminated intravascular coagulation can be observed; histologically there can be found a widespread thrombosis of capillaries and venules with a perivascular inflammatory reaction. Heparin can stop the progression of the disease.
Diagnosis: by examination of the coagulation parameters of the blood, symptomatically, by neurological examinations and others: by CT, MRI, angiography, echo, etc.
Treatment: symptomatically, by administering anticoagulant drugs, antibiotics and anti-inflammatory drugs, ACTH or steroid therapy may also be indicated, as well as anticholesterin, antilipid therapy and antihypertensive therapy, rehabilitation, etc.
RFR method: detects and may eliminate the pathogen microorganisms.

The most frequent resonances are: 294, 307, 318-320, 324-325, 332, 371-381, 389, 402-403, 410-418, 437-438, 440-454, 460-461, 477, 504, 512, 534, 555, 560-568, 576-579 kHz

12.20. Venous Disorders. Varicose Veins

Varicose veins are dilated, tortuous superficial veins with incompetent valves. The origin of this varicosity of the veins can be either primary or secondary. In case of primary varicose veins there is no causative deep venous disease and this form of varicosity generally takes a benign course. Varicosities, occuring secondary to obstructions and valvular incompetences of the deep venous system, are much more serious. The greater and lesser saphenous systems are most commonly involved, but the secondary branches of the superficial system of veins can sometimes also become dilated. Varicose veins most often appear after the age of twenty, but in case of women they often develop in puberty, during pregnancy and by the commencement of the menopause. Besides being unsightly, varicose veins often ache and the patient's legs feel tired. Many people, however, even some with very enlarged veins, may have no pain. The skin of the lower part of the leg and the ankle is often itching, leading to scratching and redness or a rash, which is often incorrectly attributed to dry skin. The symptoms of the varicosity worsen if the veins are fully stretched. In case of decompensation f.i. hot weather, warm baths, too much walking, trauma etc. there can occur complications, such as dermatitis, phlebitis or bleeding. This dermatitis shows a red, scaling, itchy area later on getting brownish, usually on the inside of the leg above the ankle. In certain more serious and complicated cases the scratching or a minor injury of the skin can lead to painful ulcers which heal but slowly. Hereditary factors are dominant in the development of this disorder. The vascular endothelium synthesizing and releasing its vasoactive substances regulate the basic and dynamic functions of the circulation. This results in the balance between the relaxing and the contracting substances and between the antiaggregate and anticoagulate substances f.i. the heparin aswell.

Herpes Simplex Virus-1 can exist in the endothel wall. This virus is able to inhibit the heparin release, and increase the development of vasorelaxing substances. Vein walls infected by herpes are dilated, the elastic membrane and the endothel will be demaged by an inflammation caused by the viral infection, so that thrombosis, or, more frequently, a superficial thrombophlebitis may develop. Familiar herpes infections may have a role in the development of this disease.

Diagnosis: symptomatically, by ultra sound, x-ray, examinations using contrast drugs, and by Trendelenburg test.

Treatment: the majority of patients with symptomatic primary varicose veins should be treated with compression stockings. If the deep veins and the perforating channels are patent and competent, it is very unusual, that the primary varicose veins will lead to stasis, pigmentation and ulceration.

Complications:

Thrombophlebitis: Thrombophlebitis encompasses a spectrum of symptoms and signs of an acute superficial or deep venous disease, the end point of which is thrombosis of the involved segment. The known precipitating factors responsible for the thrombosis of the venous system of the extremities include trauma, infection and chemical irritations. Its risk factors are prolonged bed rest, severe illness, immobilization, malignancy particularly of the pancreas, the lung and the gastrointestinal system, estrogens, oral contraceptives, polycythemia vera, congestion, disseminated intravascular coagulation, heart failure, postoperative state.

The great saphenous vein is most commonly involved in this superficial venous inflammation.

In case of deep vein thrombosis there occur a pathological blood clotting somewhere in the deep veins. The attachment of a thrombus to the vein wall can rapidly be experienced, and

if the process progresses it can get beyond the margin of the valve cups into the lumen of the vein. At this stage, the venous outflow may be completely obstructed, with a secondary venous thrombosis. The most dangerous sequel is the release of all or a portion of the thrombus, resulting in pulmonary embolism. It is important to recognize that the inflammation of the vein wall is not the dominant part of this sequence. The most dangerous and potentially lethal emboli arise from the iliac and femoral veins. Thrombophlebitis causes pain, tenderness and erythema at the locus of the inflammation of the involved vein. The local swelling can be prominent. It is important to know, that deep venous thrombosis often develops only with discrete symptoms and signs. In most cases edema, an increased calf girth and a local deep tenderness when palpitated are the most reliable physical findings, though a deep venous thrombosis can occur sometimes also without these physical findings.

Diagnosis: symptomatically, by Doppler ultrasound examination, ultrasonic velocity detector, plethysmographic methods, contrast venograms.

Differential diagnosis: by distinguishing it from bacterial cellulites, lymphangitis, myositis, muscle cramps, arthritis and Baker's cyst.

Treatment: by controlling the thrombotic process from the beginning. By administering anticoagulant drugs such as: heparin, warfarin etc. In case of infections: Doxicyclin. By compression therapy and other supportive methods.

RFR method: often detects the frequencies of the Herpes family, see also Chapter 5.2.4. Do never treat an acute thrombosis with RFR method!

The most frequent resonances are: 290-294, 318, 344-352, 371-387, 396-397, 403, 410-418, 442-454, 460, 504, 555 kHz

Postphlebitic syndrome: patients having one single episode, or multiple episodes of deep venous thrombosis frequently get irreversible changes in their veins, f.i. chronic occlusion and destruction of the venous valves, which can lead to other morbidity. These chronic damages can lead to the development of ulcers, difficult to manage.

12.21. Malignant Atrophic Papulosis (Degos Disease)

Malignant Atrophic Papulosis (MAP) is a multisystem disorder involving small-caliber blood vessels. The disease is characterized by the narrowing and occlusion of their lumen due to a pathological intimal proliferation and thrombosis, leading to ischemia and the infarction of the involved organ. This illness differs from other vasculitis disorders in that, that the inflammation is not a prominent component of the disease. The disease may involve and damage the blood vessels in the gastrointestinal and genitourinary tracts, the central and peripheral nervous system, the skin, heart, lungs, eyes, pancreas, adrenals and the kidneys. It is a progressive, occlusive disorder of the small- and medium-size arteria, leading to tissue infarction, first involving the skin. Degos disease can occur either in a limited, benign cutaneous form or as a lethal multiorgan, systemic variant.

Its etiology is a combined process: a *viral infection*, a pathological immune-"autoimmune" response (provoked by *Mycoplasma* and/or *HTLV*), hypersensitivity, and a genetic predisposition factor, all leading to an endothelial dysfunction and small vessel vasculitis (as an answer to the viral infection), where a coagulopathy is also present. In most cases no circulating immune complexes, antiendothelial antibodies, and anticardiolipin antibodies can be isolated. Nevertheless, antiphospholipid antibodies of uncertain significance were identified in some cases.

Symptoms: The recognition of the skin lesions is important for an accurate diagnosis. The early lesions are pinkish papules appearing in bouts, about 2-5 mm in size, occurring on the trunk and the extremities, but usually avoiding the face, palms, soles, and scalp. Within a few days, these papules become umbilicated, with depressed centers. At the time of investigation, most of the papules have already reached their atrophic stage, appearing as porcelain-white lesions covered with a fine scale surrounded by an erythematous border of

1-2 mms. Each single, red, painful, cutaneous-subcutaneous papules remain usually stable, with no tendency to spread or to coalesce with the neighboring lesions.
Patients with MAP may have abdominal complaints, such as abdominal pain, distention, cramps, nausea, vomiting, diarrhea, or constipation. Some patients experience weakness, fatigue, loss of weight and symptoms of malabsorption.
MAP was found to be accompanied with other systemic diseases, f.i. rheumatoid arthritis, HIV infection, with antiphospholipid antibodies and antiphospholipid syndrome, etc. Some patients complain of neurologic symptoms, such as facial and acral paresthesia, headache, dizziness, seizures, hemiplegia, aphasia, paraplegia and gaze palsy. These patients may have a variety of eye problems, including diplopia, ptosis and visual-field defects. They may experience weakness, shortness of breath, and chest pain as well.
Its provoking infections can be caused by species of the *Herpes virus group*, many different species of the *Coxsackie* and/or *ECHO virus* group, *HTLV*, *HPV* and *Mycoplasma* groups (mostly *M. fermentans*) and *nanobacteria*.
Diagnosis: by plethysmography, ultrasonic velocity measures, arteriography, functional tests, biochemical tests on cholesterol and other parameters and physical examinations.
Treatment: symptomatically and depending on the results of the biochemical tests.
The RFR method, can detect and may eliminate the viral and mycoplasmal infective agents.
The most frequent resonances are: 288-303, 317-321, 324, 344-347, 352-363, 370-376, 404-409, 416-426, 442-451, 459-464, 476-479, 517-519, 560-568 kHz

12.22. Thromboangiitis Obliterans (Buerger's Disease)

Thromboangiitis obliterans (named also Buerger's disease) is characterized by chronic inflammations and thrombosis of the arteries and veins of hands and feet. Its main symptom is the pain in the affected areas. Ulcerations and gangrene of the extremities are common complications, often resulting in the need for amputation of the involved extremity. This life-shortening disease is associated with the use of smoking tobacco products and smokeless ones. Infections caused by *Nanobacteria*, *Streptococci* and *Mycoplasma fermentans* activating immunological reactions may play an important role in the pathogenesis of the disease. Most patients suffering from Thromboangiitis obliterans are regular smokers, though there do occur some cases among patients who smoke but moderately, or use smokeless tobacco. It has been postulated that Buerger's disease is caused by "autoimmune" reactions (i.e by attacking the body's own intima and media layers of blood vessels) triggered by certain tobacco constituent and infectious microbiological factors.
Diseases with which this illness may be confused are f.i. atherosclerosis (in which case in order to prevent an acute trombosis caused by Nanobacteria the build–up of cholesterol plaques in the arteries can be observed), endocarditis (a streptococcal infection of the lining of the heart), other types of vasculitis, severe Raynaud's phenomenon associated with connective tissue disorders such as SLE and scleroderma, clotting disorders of the blood, etc. In case of Buerger's disease the "cork-screw" appearance of the arteries is resulting from vascular damages, particularly those of the arteries in the region of the wrists and ankles. Angiograms can show occlusions (blockages) and stenoses (narrowings) in many areas of the arms and legs. The changes are particularly apparent in the blood vessels at the lower right hand portion. Calciphylaxis of the Nanobacteria is often a fatal complication of the end stage vascular disease characterized by a systemic deposition of calcium phosphate salts (calcification) in the medial layer of the arteries and in the connective tissues. The calcification of the media is followed by the fibrous hyperplasia of the intima with the obliteration of the arterial lumen and by tissue gangrene. These calcification processes may cause thrombosis, though the human organism protects and develops a cholesterin sheet. These cholesterin sheets protect the blood coagulation on the

intima of the calcium layer. Nanobacterium releases endothelin 1-3 from the intima, which substances cause a strong vasoconstriction.
Mycoplasma antigens may provoke an immune-autoimmune process. The role of tobacco is not cleared up as yet, though its vasoconstrictive effect is well known.
Diagnosis: by diagnostic criteria.
Treatment: by vascular surgery, by administering anticoagulants, anti-inflammatory agents and by abstaining from all tobacco products.
The RFR method can detect the causative microorganisms.
The most frequent resonances of Buerger's disease are: 338-339, 344, 353-356, 359-362, 378-380, 385-387, 405-407, 424-426, 442-451, 479-481, 485-489, 493-495 kHz

13. GASTROINTESTINAL DISORDERS ASSOCIATED WITH INFECTIONS

Gastrointestinal symptoms occur not only concerning the infectious diseases attacking primarily the gastrointestinal tract, but frequently are also present as the manifestations of systemic diseases, f.i. anorexia, nausea and vomiting may be experienced in case of congestive disorders and uremia, diarrhea and constipation may be present as a consequence of a metabolic derangement caused f.i. by changes of electrolytes or by alterations in the thyroid function.

13.1. Esophageal Diseases

13.1.1. Gastroesophageal Reflux Disease

Gastroesophageal reflux disease (GERD) is characterized by the backflow of the stomach contents upward into the esophagus. Gastroesophageal reflux is a physiologic phenomenon experienced intermittently by most people, occuring particularly after eating. GERD can develop if the amount of the gastric fluids getting into the esophagus by reflux exceeds the normal limits, causing symptoms with or without esophageal mucosal injuries.

This pathological gastroesophageal reflux is mostly caused by an infection with *Helicobacter pylori* causing gastric inflammations of the cardia, and by functional (frequent transient lower esophageal sphincter (LES) relaxation) or mechanical (hypotensive LES) disorders of the LES.

Certain drinks (f.i. coffee, alcohol), medical drugs (f.i. calcium channel blockers, nitrates, beta-blockers), and hormones (f.i. progesterone) can decrease the pressure of the LES, too. Obesity is a predisposing factor for GERD, probably due to the increased intra-abdominal pressure.

The gastroesophageal junction must be located in the abdomen in a way that the diaphragmatic crura can assist the action of the LES, functioning thus as an extrinsic sphincter. The presence of a hiatal hernia disrupts this synergistic action and can promote the reflux.

This disorder can affect adults, children and newborns as well. Infants often experience a post-feeding vomiting. Most infants outgrow these symptom without medication, but there exsist some cases, which will need a more aggressive treatment.

Symptoms: the typical symptoms are heartburn, regurgitation and dysphagia. An abnormally occuring reflux can cause atypical symptoms, such as coughing, chest pain, wheezing and esophagitis, too.

Additional atypical illnesses, caused by an abnormally occuring reflux phenomenon, can affect the lungs (f.i. pneumonia, asthma bronchiale, idiopathic pulmonary fibrosis), the vocal cords (f.i. laryngitis, cancer), the ears (f.i. otitis media) and the teeth (f.i. enamel decay).

Chronic esophagitis caused by GERD can be classified into the following 4 grades based on its severity:

Grade I - Erythema

Grade II - Linear nonconfluent erosions

Grade III - Circular confluent erosions

Grade IV - Stricture and Barrett's esophagus

Barrett's esophagus occurs if the squamous epithelium of the esophagus is replaced by the intestinal columnar epithelium. Barrett's esophagus is present in 10% of the patients suffering from GERD and is a predilection state of the development of adenocarcinoma. In

such cases the inflammatory process of GERD is combined with a coinfection caused by *HPV* and with an infection caused by pathogens with immunosuppressive effects f.i. *HTLV, Epstein-Barr Virus, Cytomegalovirus, HIV* or *mycoplasma*.
Diagnosis: symptomatically, by barium esophagogram, esophagogastroduodenoscopy, esophageal manometry, etc.
Treatment: by administering histamine (H2)-receptor antagonists (f.i. ranitidine, cimetidine, nizatidine, etc.) which are the first line agents for patients showing mild-to-moderate symptoms of grades I-II esophagitis. H2 receptor antagonists are effective only in the healing of mild esophagitis forms caused by GERD and for providing maintenance therapy to prevent relapses. Tachyphylaxis has been observed, suggesting that the pharmacologic tolerance can reduce the long-term efficacy of these drugs.
Proton pump inhibitors (f.i. omeprazole, lansoprazole, rabeprazole, esomeprazole, etc.) work by blocking the final phase in the H+ ion secretion by the parietal cells. They have few adverse effects and are well tolerated for long-term use.
Surgical treatment: indicated concerning cc 10% of patients suffering from the progressive form of the disease, to prevent severe complications f.i. strictures or Barrett's esophagus. This surgical treatment should be done at the earliest possible stage to avoid the serious consequences.
RFR method: detects and may eliminate the gastric pathogens.
The most frequent resonances of Helicobacter pylori are: 344, 346, 355-358, 360-361, 372-379, 449-452, 544, 554 kHz

13.1.2. Mallory-Weiss Syndrome

Mallory-Weiss syndrome can be characterized by esophageal bleeding caused by a mucosal tear in the esophagus as a result of forceful coughing, vomiting or retching. It is often associated with infectious hepatitis and alcoholic hepatitis. There is some evidence that the presence of a hiatal hernia is a necessary predisposing condition. This syndrome is characterized by vomiting blood (hematemesis) after a violent retching or vomiting, though it may show also the presence of digested **blood** in the stool (melena), where retching may be absent. In most cases, the bleeding stops spontaneously after 24-48 hours, though, nevertheless endoscopic or surgical treatments can be sometimes required. This condition can sometimes be fatal.
Anyone of the disorders initiating vomiting may result in the development of a Mallory-Weiss tear, developing as a linear laceration at the gastroesophageal junction as the esophagus and the stomach are cylindrical. This cylindrical shape allows to occur longitudinal tears more easily than circumferential ones. These tears might occur either by a rapid increase of the intragastric pressure and distention, which increases the forceful fluid ejection through the esophagus, or, might be secondary to a significant change in transgastric pressure (in case of differences in the pressure across the gastric wall) as a negative intrathoracic pressure and a positive intragastric pressure cause the distortion of the gastric cardia, resulting in a gastric or esophageal tear. Due to these factors, Mallory-Weiss tears do more commonly occur among people with hiatal hernia.
Symptoms: abdominal pain, hematemesis, dizziness, syncope and melena. Hematemesis is the dominant symptom in case of all patients diagnosed as having a Mallory-Weiss tear. The diagnosis does not depend on the amount of hematemesis as it can vary from blood flecks or streaks of blood mixed with gastric contents or mucus to several ounces of bright red bloody emesis. In case of children suffering from a Mallory-Weiss tear the hematemesis is mostly preceded by one or more episodes of nonbloody emesis.
The causative factors of this syndrome can be infectious hepatitis and alcoholic hepatitis, rarely infectious gastroenteritis, gastric outlet obstruction, ulcers, hiatal hernia, malrotation, volvulus and certain inflammatory conditions of the stomach and intestine.
Diagnosis: by endoscopy.

Treatment: symptomatically and by cauterization or injection of epinephrine during the index endoscopy procedure in order to stop the bleeding, by using ice pack, balloon catheter, etc.
Prevention: by eliminating the infective pathogens causing acute infectious hepatitis and gastroenteritis, by prohibiting the drinking of alcohol and by restauration of the intestinal flora.
RFR method: can detect and eliminate viruses in the liver.
As to the frequencies of the hepatitis viruses, see their special Chapters.

13.2. Peptic Ulcer

Peptic ulcers (PU) are round or oval sores in the lining of the stomach, the duodenum and the esophagus. A shallow or incipient ulcer is named erosion. Pepsin, a digestive protease enzyme, together with hydrochloric acids produced in the lining of the stomach, digest foods, especially proteins. PU can develop in the lining of the digestive tract exposed to high acid and digestive enzymes, f.i. in the esophagus, the gastric and the duodenal region. Gastric ulcers occur most often in the antrum or at the junction of the antral and the fundic tissue. Usually single, they are often located in the lesser curvature or in the prepyloric area. The repeated regurgitation of the stomach acids into the lower parts of the esophagus can cause inflammation and esophageal ulcers. Duodenal ulcers, the most common types of the PUs, occur in the duodenum, in the first few cm length of the small intestine just below the stomach.
Ulcers coming about due to severe stress, or some severe illness, burns or traumas, are named stress ulcers. PUs develop caused by microcirculation damages. Many people with PU have *Helicobacter pylori* infections, bacteria being the major cause of this illness. They can interfere with the host's normal defense mechanisms fighting against the stomach acids, and may produce a toxin which contributes to the formation of ulcers.
Symptoms can vary depending on the location of the ulcer and on the person's age. Children and elderly people may not have the usual symptoms or may have no symptoms at all. Esophagitis and esophageal ulcers cause pain when swallowing and when lying in bed. Duodenal ulcers have typical symptoms including burning, aching, soreness, feeling of emptiness and hunger. The symptoms of the gastric ulcers are just the same as said before, but cause also bloating, nausea and vomiting after eating. More severe symptoms show the complications of peptic ulcers, like penetration, perforation, bleeding and obstruction.
Diagnosis: symptomatically, by endoscopy, barium contrast x-ray, gastric fluid analysis, blood tests, Helicobacter pylori examinations, etc.
Treatment: by administering absorbable antacids, ulcer drugs f.i. Sucralfate, H2 antagonists, f.i. omeprazole, lansoprazole and antibiotic combinations (treating Helicobacter pylori), f.i. bismuth subsalicylate, tetracycline derivates, amoxicillin, metronidazole, misoprostol, etc. and by surgery.
RFR method: should be used together with antibiotic therapy. Detects and eliminates the pathogen flora.
The most frequent resonances are: 345-347, 352-357, 365-372, 376-378, 411-413, 440-451, 552-555 kHz
The most frequent resonances of the Amoxicillin resistant strains are: 347-350, 358-360, 379, 415, 418, 556 kHz
The most frequent resonances of the polyresistant strains are: 346-351, 450-456, 557-561 kHz

13.3. Gastritis

Gastritis is the inflammation of the stomach lining. The lining of the stomach resists irritation and can usually withstand very strong acid. Nevertheless, the stomach lining can become irritated, acutely or chronic inflamed for several reasons. Gastritis can be caused by bacterial, viral and fungal infections, by allergy and stress. Histopathologically it can be erosive, atrophic, eosinophilic and plasma cell gastritis, though in case of Ménétrier's disease the gastritis is hyperplastic and hypersecretory. Bacterial, fungal and viral gastritis can be influenced well with RFR method.

Bacterial gastritis commonly results from an infection by *Helicobacter pylori* bacteria growing in the mucus-secreting cells of the stomach lining. No other bacteria are known to grow in the normally acidic stomach, but many types of bacteria may grow if the stomach produces only a small amount of acid or none at all. Such bacterial growths may cause temporary or persistent gastritis.

Phlegmonous gastritis is a very rare condition characterized by acute upper abdominal pains, signs of peritonitis, fever, purulent and ascitic fluid, nausea or vomiting, though only by normal serum amylase levels. This gastritis form is of bacterial origin, most often caused by *streptococci*, *pneumococci,* though *staphylococci*, *Escherichia coli* and the *gas-forming bacteria* can also be responsible for the disease.

Viral and fungal gastritis may occur among people with impaired immune systems.

Eosinophilic gastritis may result from allergic reactions to roundworm infestations. In case of this type of gastritis the eosinophil cells accumulate in the stomach wall, while the abdominal pain and vomiting are usually caused by the narrowing or the blockage of the stomach outlet to the duodenum.

Erosions may develop regarding any type of gastritis and in case of a normal mucosa as well.

Erosive gastritis is an important and frequent cause of upper gastrointestinal bleedings. Examining by endoscopy, the multiple bleeding erosions are usually diffusely distributed throughout the gastric mucosa or localized to the fundus, the body, or the antrum. Histologically, the mucosal destructions caused by erosions do not extend below the muscularis mucosae and can heal completely. At the same time, erosions can be observed in various stages of evolution and regression. If the erosions get enlarged and extend below the muscularis mucosae into the submucosa, they form acute ulcers. An accidental or suicidal ingestion of strong alkali (f.i. lyes) or acids (f.i. hydrochlorid or carbolic acids) can cause the necrosis of the gastric walls, particularly in the prepyloric region.

Hypersecretory gastropathy is a more frequent cause of gastric mucosal hyperplasia, than the Ménétrier's disease. The majority of such patients probably have peptic and duodenal ulcers. Patients suffering from this illness differ from those with classic Ménétrier's disease by having hypersecretion of gastric acid rather than hypochloridia. Unlike persons suffering from Zollinger-Ellison Syndrome, their blood gastrin levels are normal. Epigastric pain is the most often complaint besides anorexia, nausea, vomiting and loss of weight.

Chronic gastritis is a histological diagnosis and not a clinically recognizable entity because it was never proved to be the direct cause of the symptoms. The diagnosis can be made only by biopsy. There are two main types; i.e. the fundal gland gastritis and the pyloric gland gastritis. In case of some persons, however, both gastric areas are involved, often in a patchy manner.

Chronic fundal gland gastritis is a very common histologic finding affecting the mucosa of the gastric body and the fundus diffusely, sparing the antrum, particularly of older people. There are three pathologic types of this illness, i.e. the superficial gastritis, the atrophic gastritis and the gastric atrophy. Acute and chronic inflammation as well as epithelial abnormalities of the superficial, nonglandular mucosa are present in gastritis of superficial type. In case of atrophic gastritis there is a partial loss of fundal glands and an increased number of lymphocytes and plasma cells within the lamina propia. In case of

gastric atrophy, the fundal glands mostly disappear and are almost completely replaced by mucous or intestinal cells; without any histological evidence of inflammation. The **hyposecretory, atrophic type of gastritis** does especially occur among patients suffering from thyroid diseases, idiopathic iron deficiency anemia and pernicious anemia. Chronic antral gastritis and pyloric gland gastritis can be asymptomatic. Gastric cancer is often associated with pyloric gland gastritis involving the antrum, as well as the fundal gland areas. All types of gastritis may be secondary infected by bacteria, fungi and viruses.

Diagnosis: symptomatically, by gastroscopy, biopsy, x-ray, by Helicobacter pylori tests. By culturing bacteria and fungi.

Treatment: by treating the Helicobacter pylori infection causing the symptoms, by administering drug combinations f.i. Amoxicillin, Clarithromycin and Omeprazole, by administering vitamin B12 in case of deficiency, symptomatically by administering antacids, etc. Concerning the treatment of worms, see Chapter 9.

RFR method: detects the pathogenic microorganisms and eliminates them.

The most frequent resonances of Helicobacter pylori are: 344-346, 352-358, 365-373, 375-379, 442-451, 554-555 kHz

As regards the most frequent resonances of Candida and other fungi, see Chapter 7.3.1.

13.4. Gastroenteritis

Gastroenteritis is the term for a group of conditions usually caused by infections and characterized by symptoms such as loss of appetite, nausea, vomiting, mild to severe diarrhea, cramps and discomfort in the abdomen. The developed electrolyte imbalance can cause life-threatening dehydratations in case of very ill, very young and elderly patients. The epidemics of diarrhea among infants, children and adults are usually caused by microorganisms spread in water or food, generally after their being contaminated by infected feces.

Viral gastroenteritis occurs in sporadic endemic cases and in acute outbreaks in summer or winter. The latter is termed winter vomiting disease or intestinal flu. The majority of the cases is not caused by enteroviral infections, but is caused by *reovirus*-like agents such as *Norwalk* virus and other *Calici viruses* in the stool. *Coxsackie virus types B-3 and B-4,* as well as *ECHO viruses, types 1, 3, 6 to 9, 11 to 14, 18, 19, and 22 to 24,* often cause gastroenteritis. Attacks of vomiting and diarrhea occurring in winter outbreaks due to these *ECHO viruses* are brief, debilitating illnesses. Malaise and many a watery stools herald their onset. Fever is rarely present. Abdominal cramps and myalgia may occur. The viral gastroenteritis is highly contagious, wildly spreading in families, getting all ill at the same time or following each one after the other. *Reoviruses* and *adenoviruses* can cause sporadically similar gastroenteritis cases.

Bacterial gastroenteritis: epidemics of diarrhea affecting infants, children and adults are usually caused by bacteria. These infections also can be transmitted from person to person. The most frequent bacterial gastroenteritis form is caused by Salmonella species. The different Salmonella serotypes show a marked variation in the invasivity and capacity of causing diseases among people. For example, *Salmonella enterica serovar. anatum* causes characteristically asymptomatic intestinal infections rarely invading the bloodstream. In contrast, *Salmonella cholera-suis*, the most invasive serotype, frequently causes bacteremia and metastatic infections. The toxins of certain bacteria enhance the amount of electrolytes and water secreted by the cells in the intestinal wall. One such toxin is responsible for the watery diarrhea occuring in case of cholera. Certain other bacterial toxins cause special damages far from the intestinal cells, the toxins of *Shigella flexneri* cause f.i. brain damages. A toxin produced by *Escherichia coli* can cause traveler's diarrhea and outbreaks of diarrhea in hospital nurseries. Some bacteria, such as certain strains of *E. coli*, *Campylobacter*, *Shigella* and *Salmonella* can invade the lining of the intestines. They

damage the underlying cells, causing tiny ulcerations bleeding, allowing a considerable loss of fluid containing proteins, electrolytes and water. *Salmonella* may produce not only asymptomatic intestinal infections among human beings but also various clinical syndromes, f.i. acute gastroenteritis and enterocolitis as well. Bacteremia, paratyphoid fever and localized infections, such as osteomyelitis and endocarditis can also come about. The clinical syndromes caused by infections of *Salmonella* can not always be sharply differentiated, they sometimes overlap each other. Salmonella infections are among the most prevalent communicable diseases caused by bacteria in the world today. These infections are transmitted in the majority of cases from animals to man and occasionally from man to man, lasting usually but brief and are self-limited and mild. The most common Salmonella species are: the *S.typhi murium, S. cholera-suis, S. Newport, S. Heidelberg, S. agona, S. wein, S. infants, S. Saint-Paul, S. Thompson, S. Derby, S. javiana and S. enteritidis.* Shigellosis is an acute or chronic, self-limited infection of the intestinal tract of men characterized by diarrhea, fever and abdominal pain. The disease is frequently named bacillary dysentery, but the term Shigellosis is preferred. Species causing shigellosis are: *S. dysenteriae, S. flexneri, S. boydii, S. sonnei.* Enteral pathogenic bacteria can often be multidrug resistant.

Parasitic gastroenteritis: certain intestinal parasites, particularly *Giardia lamblia* and *Cryptosporidium* stick to, or invade the lining of the intestines, causing nausea, vomiting, diarrhea and a general feeling of sickness. These parasites are most often acquired by drinking contaminated water.

Ulcerative gastroenteritis and colitis may be caused by *Amebic Dysentery.* Certain worms can also cause gastroenteritis.

Diagnosis: symptomatically, by culturing bacteria, fungi, etc. By gastroscopy and colonoscopy.

Treatment: by administering antibiotics, antifungal and antiparasitic drugs and symptomatically.

RFR method: detects and may eliminate the bacteria, viruses and fungi together with medical treatment.

The most frequent pathogens found in case of gastroenteritis are:

Escherichia coli: 288-294, 317-319, 323-328, 334-340, 342, 352-358, 390-397, 408-412, 426, 435, 443, 478, 489 kHz

Capylobacter jejuni: 340, 350-376, 386, 415, 469, 568-569 kHz

Helicobacter pylori: 355-362, 377-379, 449-452 kHz

Shigella groups: 310, 313, 315-321, 369, 388-398, 403-410, 423-425, 496, 499, 506 kHz

Clostridium groups: 325-327, 344, 360, 364-367, 382, 396-400, 512-513 kHz

Pseudomonas: 312, 324, 330-335, 339, , 372-374, 377-380, 388-397, 401, 414, 428, 496, 558, 579 kHz

Salmonella groups: 279, 318, 329-339, 354, 360-370, 380-396, 428, 452, 497, 558 kHz

Proteus groups: 320-329, 333-339, 345-352, 408-416, 426, 516, 522-529, 535 kHz

Staphylococcus groups: 324-332, 345, 357, 372-382, 397-402, 434, 445, 462, 482, 491, 537, 557, 562-567 kHz

Enterobacter groups: 351, 373-375, 418 kHz

Entamoeba histolytica: 300, 322, 336, 398, 430-441 kHz

Adenovirus: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz

Herpes Simplex Viruses and Herpes Zostes Virus: 290-294, 301-310, 328, 331-340, 344-346, 352-365, 397-402, 413, 416-421, 425, 431-433, 449-450, 458-459, 478, 483-486, 533 kHz

Coxsackie viruses: 300-304, 331-336, 341-346, 360-366, 393-396, 416, 426-430, 443-445, 471-473, 533, 554 kHz

Enterohepatitis virus: 477, 487, 564 kHz

Calicivirus: 340-346 kHz

Cytomegalovirus: 305, 349, 407-412, 512, 530-536, 548 kHz
Giardia lamblia: 340, 415-416, 420-430, 516 kHz
Cryptosporidium: 294, 326, 357, 363, 448, 457-459, 492 kHz
Ascaris: 293, 308, 400-410, 452 kHz
Taenia group: 318-319, 339, 411, 431-437, 440-500, 534, 554 kHz
Enterobius: 395, 418-427 kHz
Candida albicans: 297, 308, 332-338, 345, 352, 362, 372, 380-390, 403, 410, 420-426, 443-450, 453, 460, 474, 488-490, 504, 520, 554, 570-580 kHz
This list of pathogens is not yet complete, many other microorganisms may play a role in the etiology of bowel diseases.

13.4.1. Salmonella Infections

The three Salmonella species have more than 2200 different serologic types. Human salmonella infections cause a great number of clinical illnesses, f.i. enteric fever (typhoid or paratyphoid fever), acute gastroenteritis, bacteremia, chronic inflammation of the gallbladder and the urinary tract and other localized infections. The three Salmonella species are: *Salmonella typhi*, *Salmonella cholera-suis* and *Salmonella enteritidis*. The most frequently isolated serotypes are *S. typhimurium, S. enteridis, S. Newport, S. Heidelberg, S. infantis, S. Saint-paul, S. Thompson, S. cholera-suis, S. typhi, S. derby and S. javiana.* Salmonellae, these motile gram-negative bacilli, do not ferment lactose and sucrose but ferment glucose. Almost all serotypes produce gas.

Typhoid fever is an acute systemic disease caused by an infection of *Salmonella typhi.* It is characterized by malaise, fever, abdominal discomfort, transient rash, splenomegaly and leucopenia. The most prominent complications are the intestinal hemorrhages and perforations. Typhoid bacteria are shed in feces and urine of infected people. An inadequate hand washing after defecation or urination may spread Salmonella typhi to food or water supplies. About 3 percent of the people are infected with salmonella typhi and, remaining untreated, will shed bacteria in their stool for longer than one year.

The **Symptoms** of the disease begin 7-14 days after being infected, consisting of fever, headache, joint pain, sore throat, constipation, loss of appetite, abdominal tenderness and pain. Less commonly, painful urination, cough, slow heartbeat, extreme exhaustion and nose-bleed can develop. Delirium, stupor, coma and shock may occur in severe cases. Sometimes the infection causes pneumonia-like symptoms, or symptoms similar to those of a urinary tract infection. An infection of the gallbladder and the liver also may occur. Bacteremia occasionally leads to the infection of the bones, i.e. osteomyelitis, the heart valves i.e. endocarditis, the lining of the brain i.e. meningitis, the kidneys i.e. glomerulitis, or the genital or urinary tract. Muscle infection caused by Salmonella can lead to abscesses. The most prominent microscopic lesion in typhoid fever is the proliferation of large mononuclear cells in many a different tissues. Mononuclear hyperplasia leads to lympadenopathy, splenomegaly and to a significant enlargement of lymphoid tissues in the intestines, especially in the Peyer's patches of the terminal ileum.

Nontyphoidal salmonella infections can produce gastrointestinal upset, enteric fever and specific localized infections. Infected meat, poultry, raw milk, eggs and egg products are common sources of Salmonella bacteria causing nontyphoidal infections. Being though carriers, some infected people show no symptoms at all.

Diagnosis: by blood culturing. By typisation of bacteria.

Differential diagnosis: rickettsioses, brucellosis, tularemia, leptospirosis, psittacosis, infectious hepatitis, mononucleosis infectiosa, atypical pneumonia, miliary tuberculosis, malaria, lymphoma, rheumatic fever, etc.

Treatment: symptomatically and by antibiotic therapy f.i. Chloramphenicol, Doxycyclin. If the person is delirious, comatose or in shock, corticosteroids may be used to reduce their brain inflammation.

RFR method: together with the antibiotic treatment it detects and eliminates.
The frequencies of Salmonella typhimurium are: 339, 354-356, 380-387, 390-395, 428 kHz
The frequencies of Salmonella enteritidis are: 329-330 kHz
The frequencies of Salmonella paratyphi are: 318, 361-371, 384, 392, 432-434, 452, 497 kHz
The frequencies of Salmonella infantis are: 329, 339, 365-369, 497 kHz
The resonant frequencies of other Salmonella species are: 318, 329, 337-339, 354-356, 361-368, 384-386, 388-395, 418-430, 497, 553, 558-559 kHz
This list is not yet complete.

13.4.2. Cholera

Cholera is an acute illness, caused by the colonization of the small intestine by *Vibrio cholerae*. The disease is characterized by its epidemic occurrence and the symptoms of massive diarrhea with rapid depletion of extracellular fluids and electrolytes in more severe cases. The enterotoxins, which are protein-products of Cholera bacteria, cause the secretion of an immense amount of fluid rich in salt and minerals produced by the cells of the small intestine. Cholera is spreading by ingesting seafood or other foods and water contaminated by the excrements of infected people.
The *Vibrio cholerae* is a curved, aerobic, gram-negative bacillus with a single polar flagellum. It is rapidly motile and possesses both O and H antigens. The serologic identification is based on the differences in the polysaccharide O antigen. Poor sanitation appears to be primarily responsible for the continued presence of cholera, though host factors, such as relative or absolute achlorhydria can also play an important role in the susceptibility to infection. The enterotoxin-induced electrolyte secretion occurs in the absence of any demonstrable histological damage of the intestine epithelial cells or of the capillary endothelial cells of the lamina propria.
Vomiting generally follows or precedes occasionally the onset of diarrhea. As the saline depletion progresses, severe muscle cramps will come about, usually involving the calves. The severely ill cholera patient is typically cyanotic, with a pinched face, scaphoid abdomen, poor skin turgor and thready or absent peripherial pulses. The voice of the patient is faint, high-pitched, often inaudible, tachycardia and tachypnea can also be experienced. In case of inadequately treated patients, the cause of death can be hypovolemic shock, metabolic acidosis and uremia, resulting from acute tubular necrosis.
Diagnosis: by laboratory analysis of blood, stool or other body fluids. Rapid diagnosis is possible either by directly observing immobilization of vibrios by type-specific antiserums using dark-field or phase-contrast microscopy or by identifying the pathogens by immunofluorescent methods.
Prevention: using boiled water, by avoiding uncooked vegetables and inadequately cooked fish or shellfish. By cholera vaccine.
Treatment: By administering Doxycyline, Tetracycline, Chloramphenicol and symptomatically, by the substitution of fluids given intravenously.
RFR method: should only be used together with the medical treatment.
The most frequent resonances are: 280-284, 300-305, 330-342, 353, 372, 403-410, 428-435, 460-470, 495, 529 kHz

13.4.3. Cyclosporiasis, Traveler's Diarrhea

Cyclospora cayetanensis, a coccidian protozoan parasite, can cause intestinal infections, the socalled „traveller's diarrhea". Cyclosporiasis is endemic in Bangladesh, Brazil, Chile, China, Cuba, the Dominican Republic, Egypt, Guatemala, Haiti, India, Indonesia, Jordan, Mexico, Morocco, Nepal, Nigeria, Pakistan, Peru, Puerto Rico, Romania, Saudi Arabia, Tanzania, Thailand, Turkey, Venezuela, Viet Nam, Zimbabwe. Ilnesses caused by

Cyclosporiasis can occur seasonally in Guatemala (from May to August), in Haiti (from January to March or April), in Nepal (from May to August) and in Peru (from December to May), often disappearing at times for months. In several countries Cyclospora species was found in source waters as well.
Contamination of food and drinking water can lead to human ingestion and infection.
Symptoms: are characterized by cyclical diarrhea accompanied by fatigue, malaise, anorexia, nausea, loss of weight and abdominal cramps interspersed with periods of remission. Low-grade fever and malabsorption may come about. If left untreated, the diarrhea may continue for weeks and months. The Cyclospora infection affects both immunocompetent and immunocompromised individuals as well (see also Chapter 8.11.).
RFR method: detects and may eliminate the parasite. It is advised to combine RFR method with the trimethoprim-sulfamethoxazole therapy.
The most frequent resonances of the Cyclospora species are: 322-325, 360-362, 507-509, 547-556 kHz
Immunocompromised hosts require an oral antibiotic therapy for a longer time, to be followed by prophylaxis to prevent its recurrence. It is necessary to examine the cause of the immunocompromised state and to treat other, eventually found pathogens, too.

13.4.4. Intestinal Flukes

Fasciolopsis buskii is the most frequently infecting intestinal trematode causing disease among people. *Echinostoma ilocanum* also cause often human infections, while *Heterophyes heterophyes* and *Metagonimus yokogawai* are less often causatives of human intestinal fluke infections. Intestinal flukes, only rarely causing human intestinal infections, are *Gastrodiscoides hominis*, *Phaneropsolus bonnei* and *Prosthodendrium molenkampi.*
Fasciolopsis buskii, attaches itself to the duodenal and jejunal mucosa; however, in case of severe infections, it may attach itself to the ileum and the colon, too. Intestinal flukes cause inflammations, ulcerations and mucous secretions at the locus of their attachment. Severe infections may cause *intestinal obstruction and malabsorption leading to hypoalbuminemia, protein-losing enteropathy and an impaired vitamin B-12 absorption.* Intestinal flukes are endemic in the Far East and Southeast Asia. *Heterophyes Heterophyes* can also be found in the Nile delta region of Egypt. Intestinal flukes are endemic in some parts of North Africa, too. The cercariae of these flukes encyst on various plants, such as water caltrop, water chestnut, lotus (on the roots), water bamboo, and other aquatic vegetables. Human beings can get infected by consuming these raw vegetables. The metacercariae attach themselves to the wall of the human duodenum, becoming adult worms in approximately 3 months. The adult worms cause *traumatic, toxic and obstructive damages to the intestinal mucosa.* At the locus of their attachment, deep inflammatory ulcerations can be observed. Large numbers of worms provoke an excessive mucous discharge and can obstruct the lumen of the bowel. The absorbed *metabolites of the adult worm may cause intoxication and sensitization.*
Echinostoma ilocanum worms are small, elongated flukes measuring 5-15 mm in length and 1-2 mm in width. In case of an Echinostoma infection, the adult worm, attached to the intestinal wall of people, produces eggs that pass in the feces. When the eggs reach water miracidia will develop and penetrate the first intermediate hosts: snails. Being inside the snails, they develop into sporocysts, mother rediae, daughter rediae and cercariae in the course of 6-7 weeks. The cercariae leave the snails in order to encyst in the second intermediate hosts, which can be freshwater snails, fish, tadpoles and vegetables. People are infected by ingesting raw or undercooked second intermediate hosts. Inside of the human hosts, the flukes attach themselves to the small intestinal mucosa and can produce, depending on the severity of infection, *shallow ulcers with mild inflammation and/or local necrosis.* Mild infections are asymptomatic, but heavy infections produce *diarrhea, flatulence and intestinal colic* similar to that observed in case of fasciolopsiasis.

Heterophyes heterophyes and *Metagonimus yokogawai* are important human parasites. *H. heterophyes* is a small fluke, measuring 1-1.8 mm in length and 0.3-0.7 mm in width, with a broadly rounded posterior end. The oral sucker is subterminal, its size being one third of the size of the ventral sucker. These flukes are to be found in the infected human intestine, jejunum and ileum. The worms produce eggs, which are excreted in the feces and into the water. The first intermediate hosts, the snails, ingest the eggs. In the snails, the eggs hatch and undergo their developmental cycle, forming cercariae, which emerge from the snails and encyst on the second intermediate hosts: brackish or freshwater fish. In their second intermediate hosts, the cercariae transform into metacercariae, which infect people upon ingesting of raw or undercooked fish. In case of human infections, the flukes attach themselves to the small bowel causing *ulcers, mild inflammation and superficial necrosis.* Their clinical presentation includes *diarrhea, dyspepsia, and intestinal colic.* Due to their small size, the eggs, and sometimes the adult flukes, can enter the blood vessels and embolize into the brain, producing symptoms similar to *cerebral hemorrhages.* Eggs may also enter the mesenteric lymphatics and travel to the heart, causing *myocarditis, chronic congestive heart failure and even death.*

Metagonimus yokogawai is closely related to H heterophyes. It measures 1-2.5 mm in length and 0.4-0.75 mm in width. The ventral sucker is located to the right of the midline. Its life cycle is similar to that of H heterophyes. Metacercariae infect human beings after ingestion of raw or undercooked fish. The flukes then invade the mucosa of the small intestines, causing *inflammation and ulcerations.* Eventually the flukes become encapsulated. M yokogawai can occasionally embolize into other organs. Patients infected with M yokogawai suffer from *mucous diarrhea and vague abdominal complaints.* The prognosis of the illness is usually good, excepting the case of embolization. This infection but rarely causes death, occuring only regarding patients with a heavy worm burden, with severe cachexia and prostration. In cases of infection with H heterophyes or M yokogawai, death may occur with embolization of the eggs to the heart or the brain, though most patients remain asymptomatic. Patients with moderate infections have occasional *loose stools, some loss of weight, malaise, and, occasionally, generalized abdominal pain.* Severe infections, where toxic diarrhea alternate with constipation and hunger pangs are the first symptoms, usually occuring towards the end of the incubation period. As the infection progresses and the worm burden increases, *edema of the face, the abdominal wall and the lower limbs can be observed, as well as ascites and generalized abdominal pain. Anorexia, nausea, and vomiting* are also common. The diarrhea usually persists, becoming greenish-yellow and malodorous. M yokogawai eggs, if present in the heart, can cause the symptoms of *myocarditis.*

Intestinal flukes are endemic in areas where abundant snail hosts exist (eg. China, Vietnam, India and other parts of Asia).

Summarized Intestinal Trematode Infections

Infection	Source	Geographic Distribution
Fasciolopsiasis	Freshwater plants (water caltrop, water chestnut)	China, Thailand, Bangladesh, India
Echinostomiasis	Tadpoles, freshwater snails, fish, frogs	Indonesia, Philippines, Taiwan, Thailand
Heterophyiasis	Fish	Egypt, Iran, Tunisia, Turkey
Metagonimiasis	Fish (cyprinid)	Far East, Spain, Eastern Europe

Diagnosis: symptomatically, by stool examinations. By other laboratory findings including anemia and eosinophilia. There are no serologic tests available.

Treatment: by administering Praziquantel, Albendazole and symptomatically.

RFR method: should only be used after the oral antiparasitic treatment.

The resonant frequencies of the Fasciolopsis buskii are: 426-438 kHz
The resonant frequencies of the Echinostoma ilocanum are: 422-430 kHz
The resonant frequencies of the Metagonimus yokogawai are: 435-445 kHz
The resonant frequencies of the Gastrodiscoides hominis are: 445-465 kHz
The resonant frequencies of the Prosthodendrium molenkampi are: 396-428 kHz
The resonant frequencies of the Echinostoma revolutum and ilocanum are: 422-441 kHz
This list is not complete yet, there are other subspecies and life cycle forms having other frequencies. They also should be detected and eliminated.

13.5. Crohn's Disease

Crohn's disease is a chronic regional granulomatous enteritis, the etiology of which may be associated with combined parasitic, bacterial or viral infections together with allergic and autoimmune factors. This Crohn's disease affects typically the full thickness of the intestinal wall, mostly occuring in the lowest part of the small intestine, in the terminal ileum and in patchy areas of the whole bowel system, f.i. in the colon, in the upper part of the small intestine alone and the skin around the anus as well. Stomach, mouth and the esophagus are only rarely affected. In case of Crohn's disease the dysfunction of the immune system leads to a prolongated illness of the bowel, primerily of an infectious origin. There are current theories implicating the role of microbial infections, genetic, environmental, dietary, vascular, immunologic and even of psychosocial factors as the potential causative agents. It is suggested that patients have an inherited susceptibility for an aberrant immunologic response to one or more of these provoking factors. Though there do contribute several genes to a complex phenotype; the mutations within the CARD15, the NOD2 gene (or the IBD1 gene) are thought to offer susceptibility to get this illness.
Crohn's disease is characterized by an increased production of interleukin 12 (IL-12), TNF-alpha, and interferon gamma (IFN-gamma). TNF-alpha plays a critical role in the inflammative process of this disease. The increased production of TNF-alpha by macrophages of patients suffering from Crohn's disease results in increased concentrations of TNF-alpha in the stool, the blood and the mucosa.
This illness is thought to be an immune disease developing due to the effects of certain combined infections. Such infective microorganisms are *Mycoplasmas* (f.i. *M. fermentans, M. pneumoniae), HTLV,* etc. According to the works of Naser S.A. *Mycobacterium avium subspecies, Mycobacterium paratuberculosis* can cause Crohn's disease; very similar to Johne's disease affecting cattles. *Enteroviruses and Enterobacteria* also play an important role in the triggering off the development of the disease. All these infectious agents may provoke chronic immune and autoimmune processes.
Microscopically, the initial lesion starts as a focal inflammatory infiltrate around the bowel crypts, followed by ulceration of the superficial mucosa. The inflammatory cells later on invade the deeper layers as well, in which noncaseating granulomas, fistuli, microperforations, abscess formations, adhesions and malabsorption will develop. The granulomas extend in all layers of the intestinal wall affecting even the mesentery and the regional lymph nodes. Though this granuloma formation is pathognomonic for this illness, its absence does not exclude the diagnosis.
Its most often present **symptoms** are related to a chronic inflammatory process involving the ileocecal region. The early symptoms are usually characterized by *low-grade fever, prolonged diarrhea with abdominal pain but without urgency, loss of appetite, loss of weight and generalized fatiguability.* The patients suffer from crampy or steady pain either in the right lower quadrant of the abdomen or periumbilical. The pain precedes and may be partially relieved by defecation. The diarrhea is usually intermittent and not bloody. The development of intestinal obstructions, abnormal connecting channels and abscesses are complications of the disease. Perianal fissures or fistulae are common. Fistulae can also

develop connecting two different parts of the intestines. The cologastric fistulae may cause feculent vomiting, the enterovesical fistulae can cause recurrent urinary tract infections and hematuria, the enterovaginal fistulae are accompanied by feculent vaginal discharge, while the enterocutaneous fistulae manifest as feculent soiling of the skin. Perforation of the small intestine may sometimes also come about. Obstructions are initially caused by a significant edema of the mucosa and can be associated with spasms of the bowel, too. The obstructions can be intermittent and reversible following an effective conservative and anti-inflammatory therapy. Crohn's disease is often associated with certain inflammations of other organs, i.e. the skin, the joints, the mouth and the eyes. Such skin manifestations are f.i. the *erythema nodosum and the pyoderma gangrenosum. Ankylosing spondylitis and sacroiliitis* causing hip and back pain, is often associated with Crohn's Disease, may antedate the bowel disease by several years, and can persist even after a surgical or medical remission of the disease. Peripheral asymmetric arthritis of the larger joints are probably more often associated with colitis rather than with enteritis. *Aphthous ulcers* are the most common mouth lesions. *Episcleritis, recurrent iritis, uveitis* are frequently occuring ocular illnesses of this illness. These inflammations can be of an autoimmune character. An *inadequate absorption of nutrients, amyloidosis and thromboembolic manifestations* may also come about.

The exacerbations of the illness occur usually in irregular intervals of the patient's life. The inflammation tends to recur in the same locus even if the diseased place was removed surgically. Loss of weight and generalized fatigue is usually always present.

Diagnosis: symptomatically, by examining laboratory markers (i.e. iron malabsorption, hypoalbuminemia, hypocholesterolemia, hypocalcemia, erythrocyte sedimentation, hypomagnesemia, hypoprothrombinemia, etc.). The acute phase reaction markers, f.i. CRP and Alpha-1-acid glycoprotein correlate closely with the activity of the disease. By virus examinations, bacterium examinations and parasite examinations of the stool. By toxicological examination of Clostridium difficile. By antibodies to the yeast Saccharomyces (a test result positive for ASCA and negative for p-ANCA antigen suggests the presence of Crohn's disease). By CT scan assessing extramural complications such as fistulae, abscesses, hepatobiliary and renal complications. By MRI demonstrating pelvic lesions. Ultrasound and colonoscopy differentiating tubo-ovarian origin. By endoscopy with biopsy, etc.

Treatment: symptomatically, by administering antidiarrheal and anti-inflammatory drugs, antibiotics and corticosteroids. A corticosteroid treatment may be advisable only after the elimination of the pathogens present in the intestines. The indication of immunosuppressants is questionable.

RFR method: detects and may eliminate all pathogen microorganisms.

The most frequent resonances found in case of Crohn's Disease are: 298-300, 303-307, 321-324, 339-340, 356, 370-376, 383, 392-393, 396-402, 425-426, 442-451, 479, 497-501, 565, 576-580 kHz

The first pathogen to be eliminated should be the Mycoplasma: 321-324, 442-451 kHz

Regarding the second pathogen, i.e. the Mycobacterium avium, see Chapter 6.14.4.1.

13.6. Bowel Diseases

Bowel infections can be simple bacterial infections, but also different, complex conditions combining parasitic and bacterial infections together with allergic and autoimmune components. The bile duct and the intestinal tract influence each other. Inflammatory bowel diseases can be acute or chronic disorders in which the intestines become inflamed, often causing recurring abdominal cramps and diarrhea including acute colitis, irritable bowel syndrome and ulcerative colitis. Some bacteria, such as certain strains of *Escherichia coli*, *Campylobacter*, *Shigella* and *Salmonella* invade the lining of the intestines. *Certain viruses*, such as the *Norovirus (Norwalk virus)*, *Rotavirus*, *Enterovirus*,

adenovirus and *Coxsackie virus* can cause inflammation and diarrhea. *Ascaris lumbricoides* and other worms can cause diarrhea accompanied by abdominal cramps, nausea and vomiting. The development of complex multicausal chronic diseases such as Crohn's disease and Ulcerative colitis are associated with damaged immune responses.

13.6.1. Irritable Bowel Syndrome

Irritable bowel syndrome is a disorder of motility of the entire gastrointestinal tract causing abdominal pain, constipation or diarrhea. It is a chronic illness affecting women three times more often than men. In case of this syndrome, the intestinal tract is especially sensitive to certain stimuli. Stress, diet, drugs, hormones, infections of the biliary system and irritants may cause abnormal contractions of the intestinal muscles. In case of the irritable bowel syndrome, colitis and spastic colon are accompanied by parasitic, bacterial and allergic problems. The elimination and the killing of all parasites, i.e. *fungi, bacteria and viruses*, can easily cure all these bowel symptoms. Since the reinfection is a big problem, until the patient is completely cured, one should take his/her pet away.

Symptoms: There are two major types of the irritable bowel syndrome. The *spastic colon type* is often triggered by eating, is characterized by periodic constipations or by painful diarrhea and is associated with bile duct diseases. Sometimes constipation and diarrhea change alternatively. There often appears mucus in the stool. The person may suffer bloating, gas, nausea, headache, fatigue, depression, anxiety and difficulty in concentrating.

Regarding the second type, painless diarrhea or relatively painless constipation can come about. The diarrhea usally begins very suddenly soon after eating and with extreme urgency. Flatulence caused by an increased amounts of gas in the gastrointestinal tract is a typical symptom of the irritable bowel syndrome. *Mycoplasma species* may play a role in the pathogenesis of this syndrome, see Chapter 23.9. as well.

Treatment: symptomatically and by administering antibiotics, antihelminthics, antispasmodics f.i. propantheline, antidiarrheal drugs, f.i. diphenoxylate, loperamid, antidepressants, mild tranquilizers, by diet and administering digestive enzymes, etc.

13.6.2. Acute Colitis

Acute inflammations of the colon can be caused by a number of infectious agents including viruses, bacteria and parasites. The clinical symptoms of these infections are fever, crampy, lower abdominal pain and diarrhea, which may be bloody. These infections can be caused by *Shigella, Escherichia coli, Salmonella, Pseudomonas, Entamoeba histolytica*, etc.

In case of a **hemorrhagic colitis** certain strains of *Escherichia coli* infect the large intestines producing a toxin which causes sudden bloody diarrhea and sometimes other serious complications, too. **traveler's diarrhea** can also be caused by *E. coli* infection, characterized by diarrhea, nausea and vomiting occuring commonly among travelers.

Some bacteria, f.i. certain strains of *Salmonella, Shigella, Campylobacter* and *Pseudomonas* invade the lining of the intestine. They damage the underlying cells, causing tiny ulcerations which bleed and lose a considerable amount of fluid containing proteins, electrolytes and water.

The *staphylococcal* **food poisoning** is a poisoning from eating food contaminated with the toxins of certain types of *Staphylococci*, causing generally diarrhea and vomiting.

An other type of *food poisoning* and even a **necrotizing enteritis** can be caused by eating food contaminated by the toxins of *Clostridium perfringens*.

Botulism is caused by the toxins of *Clostridium botulinum*. Certain toxins produced by this Clostridium species are highly poisonous proteins resisting the destructive effects of the protective enzymes of the human intestines. The first symptoms of botulism are usually

dry mouth, double vision, dropping eyelids and inability to focus on nearby objects. The paralysis of the nerves and muscles will develop, beginning at the face and the head, eventually reaching the arms, the legs and the breathing muscles. Without being administered with antitoxin this disease may be fatal. The type and the severity of the symptoms depend on the species and the quality of the microorganism or its ingested toxins. The symptoms vary also according to the person's resistance to the disease.

The symptoms of acute colitis occur often dramatically, characterized usually by nausea and vomiting, abdominal pain and diarrhea with or without visible blood and mucus in the stool. The loops of the intestine may be painfully distended with gas. The patient may have high fever, feel generally sick, experience an active moving of the intestinal muscles and feel extremely exhausted. Severe vomiting and diarrhea can lead to marked dehydration and to a severe decrease of the blood pressure. The excessive vomiting and diarrhea can cause a serious loss of electrolytes, resulting in the hypokalemia and hyponatremia of the blood. All of these imbalances can be potentially serious. *E. coli* toxins damage the lining of the large intestine, but if absorbed into the bloodstream, they can affect other organs, too, such as the kidney and the heart.

In case of *E. coli* infections a hemolytic-uremic syndrome can also develop. Its symptoms are hemolytic anemia, thrombocytopenia and even a sudden kidney failure. Some people suffer from seizures, strokes, or other complications of nerve damages and brain damages.

Treatment: symptomatically or by administering antibiotics, antihelminthics, analgesia.

13.6.3. Clostridium Difficile

Clostridium difficile species are spore forming bacteria and parts of the normal intestinal flora of 50% of children under age two, and less frequently, of persons over two years of age. These bacteria can cause pseudomembranous colitis and antibiotic associated diarrhea. Antibiotics can alter the colonic microflora facilitating the colonization and the growth of Clostridium difficile primarily affecting hospitalized patients. The combination of nosocomial exposure to C difficile and the loss of normal protective colonic bacteria leads to their colonization. As the bacterium proliferates, toxins A and B will be produced and released. Toxin A, an enterotoxin elicit an acute inflammatory response among animals. Toxin B, a cytotoxin is pathogenic affecting people. These toxins cause the release of proinflammatory mucosal cytokines, resulting in exuberant inflammatory exudates on the colonic mucosal surface with intervening areas of normal mucosa. These plaques consisting of inflammatory cells, disrupted crypts and cellular debrises appear macroscopically as yellow to gray pseudomembranes. This infection can cause a wide spectrum of clinical manifestations. An antibiotic-associated diarrhea, not caused by C difficile, is characterized by three or four loose bowel movements a day, usually without any other systemic complaints. It can be treated conservatively by stopping the inciting antibiotics.

Symptoms: C difficile colitis is complicated by more frequent bowel movements, abdominal pain and fever. Systemic manifestations may include dehydration, prerenal azotemia, sepsis syndrome, toxic colitis and among seriously ill patients, even death. Laboratory tests usually demonstrate a marked elevation in the white blood cell count with a left shift towards immature forms. An extreme form of fulminant colitis may require colectomy and could lead to death.

Diagnosis: By ELISA detecting C difficile toxins in the stool.

Differential diagnosis: by distinguishing it from other bacterial, fungal and viral colitis forms.

Treatment: by stopping the administration of inciting antibiotics, by administering antimotility agents (f.i. loperamide hydrochloride, diphenoxylate hydrochloride with atropine sulfate, by administration of metronidazole, vancomycin for 10 to 14 days, etc.

Prevention: after an antibiotic treatment it is advised to administer probiotica (bifidobacteria and acidophilus group bacteria) in order to win back the lost friendly bacterial flora).
RFR method: detects and may eliminate the Clostridium difficile.
The most frequently resonant frequencies are: 320-326, 342-345, 392-400, 512-513 kHz

13.6.4. Hemorrhagic Colitis

Hemorrhagic colitis is caused by certain strains of *Escherichia coli,* infecting the intestine and producing a toxin which causes sudden bloody diarrhea and sometimes other serious complications as well. The E. coli toxins damage the lining of the large intestine. If they are absorbed in the bloodstream, they can also affect other organs, f. i. the kidneys. Symptoms include trombocytopenia, a sudden kidney failure and anemia, caused perhaps by the hemolytic-uremic syndrome with the breakdown of the red blood cells. Some patients experience seizures, strokes, or other complications of the nerves and brain damages.
Diagnosis: symptomatically, by a specific testing for E. coli 01557: H7, by colonoscopy.
Treatment: by administering Doxycyclin, Chloramphenicol.
RFR method: should be used only together with the beginning of the antibiotic treatment.
The most frequently resonant frequencies are: 317-319, 323-328, 334-340, 352-358, 390-397, 408, 410-412, 422, 426, 435, 443, 478, 489, 560-570, 576-580 kHz

13.6.5. Amebiasis, Amebic Dysentery

Amebiasis is an infection of the large intestine produced by *Entamoeba histolytica.* It is an asymptomatic carrier state in case of most persons, but the infection can cause chronic, mild diarrhea and even fulminant dysenteries. Its most often appearing extraintestinal complication is the hepatic abscess, which may rupture into the peritoneum, the pleura, the lung and the pericardium. E. histolytica exist in two forms: the motile trophozoite and the cystic forms. The trophozoite, the parasitic form, dwells in the lumen and the wall of the colon, divides by binary fission, grows best under anaerobic conditions, and requires the presence of either bacteria or tissue substrates to satisfy its nutritional requirements.
Though the parasites can sometimes infect rats, cats, dogs and primates, human beings are their principal hosts and reservoirs. Asymptomatic cysts are the source of the new infections. The cysts are usually spread by contaminated food or water. Direct fecal spreading may occur if the environment is massively contaminated. The cases of Amebic Dysentery are usually sporadic, though waterborne epidemics can also occur. In some cases there are intermittent diarrhea also caused, consisting of one to four foul smelling loose or watery stools daily. The stools can contain mucus and blood as well. Loose stools alternate with periods of relatively normal ones, this disorder may even persist for month and years. Flatulence and abdominal cramping are frequent. The physical findings can only be a tender hepatomegaly and a slight pain, if the cecum and the ascending colon are palpated. Hepatomegaly is very frequent, the sigmoidoscopy usually demonstrates extensive rectosigmoid ulcerations. The invasion of the appendix may lead to the clinical signs of appendicitis. Complications can be f.i. hepatic amebiasis, pleuropulmonary amebiasis, peritonitis and pericarditis.
Diagnosis: by identifying ameba in the stool or the tissues. By macroscopic and microscopic stool examinations. Examinations with sigmoidoscopy. Serologic amebiasis tests.
Differential diagnosis: by distinguishing it from other intestinal infections and diseases.
Treatment: symptomatically, by substituting fluid and electrolytes. By administering Chloroquine diphoshate, Emetine, Metronidazole, Flagyl, Doxycyclin, etc.

RFR method: detects and may eliminate the ameba!
The most frequently resonant frequencies are: 300-336, 340-346, 374, 381-387, 397-400, 402, 410, 425, 433-442, 544 kHz

13.6.6. Ulcerative Colitis

Ulcerative colitis (UC) is a systemic chronic disease, in which the mucous layer of the large intestine becomes inflamed and ulcerated, leading to crypt abscesses and cryptitis, causing episodes of bloody diarrhea, abdominal cramps, fever and loss of weight. Remaining often a lifelong illness, this condition has a profound emotional and social impact on the affected individual. There is no etiologic difference known exactly between ulcerative colitis and Crohn's disease, though there are some markers pointing on the differencies. Serum and mucosal autoantibodies against the intestinal epithelial cells can be present in case of UC. Persons with UC are often found to have p-antineutrophil cytoplasmic antibodies (pANCAs).

The cause of UC may be associated with an immune deficiency-mediated abnormality of the humoral and the T cell-mediated immune response together with a generalized *Herpes simplex virus* infection combined with *other viral or bacterial* infections. Herpes simplex viruses are absorbed by the mucosal cells changing the antigen structure of these cells. The immune response againts these changed structures results in damages of the mucosa and other tissues. The loss of tolerance to the indigenous enteric flora is thought to be the central event in the pathogenesis of this inflammatory bowel disease. *Mycoplasmal or/and HTLV* infections can have an important role in the development of this immune alteration, as these infections can reduce the immune response of the host. Secondary infections caused by different *enteroviruses* and *enterobacteria* can happen in this way.

Genetic susceptibility (coded in chromosomes 12 and 16) can also be a causative factor of the development of the illness. A positive family history is associated with a higher risk for the development of the disease.

The laboratory findings in ulcerative colitis are nonspecific. Anemia due to a combination of iron deficiency and a chronic inflammation are common. Leucocytosis with a shift to the left is frequent, especially during acute febrile exacerbations. Hypoalbuminemia, electrolyte disturbances, a mild increase in the serum alkaline phosphatase as well as in the liver enzymes of the serum, elevations of the erythrocyte sedimentation rate are often to be found.

The Symptoms: are most often characterized by urgency and tenesmus, abdominal cramps, mild fever, dehydration, abdominal tenderness, malnutrition and loss of weight. A patient with ulcerative colitis but rarely has constipation, particularly if suffering predominantly from rectal inflammations. Most patients show only a few signs at their physical examination, though the abdomen may be rather prominent. Rectal bleedings frequently occur, with or without mucus.

Extracolonic manifestations, such as aphthous ulcers involving the intestinal bowel, the tongue, the lips, the palate and the pharynx; ocular inflammations such as iritis, episcleritis and uveitis can be also often experienced. Seronegative arthritis, ankylosing spondylitis (in case of HLA B27 positivity) and sacroiliitis, illnesses of the musculosceletal system, are often associated with UC. Skin diseases f.i. erythema nodosum and pyoderma gangrenosum, autoimmune haemolytic anaemia, deep venous thrombosis can also occur. This inflammation of the large bowel is in close relation with the liver and the production of the bile. The bile duct system is also in a state of chronic inflammation, the excretion of the acids of the bile is inhibited. This state furthers the growing of the bacteria present in the bowel. The inflammation of the bile ducts may appear many years before the appearance of any of the intestinal symptoms of UC. **Primary sclerosing cholangitis** and **chronic inflammation of the liver** are often experienced complications of the disease. In more severe cases there can develop even cirrhosis caused by chronic hepatitis and primary

sclerosing cholangitis. Other more serious but frequent complications of the disease are the *toxic megacolon* and the *development of neoplasmas* in the colon. The clinical course of chronic ulcerative colitis is variable, depending on the extent of the involvement and the intensity of the inflammatory process. Most patients have a history of intermittent symptoms interspersed with periods of normal or nearly normal states. The pseudopolyposis, the polyposis and the adenocarcinomas of the colon are caused by special *viral* infections, as for their frequency, see below.

Diagnosis: by CT, barium enema x-ray, biopsy, colonoscopy, sigmoidoscopy. By examinations of micro and macro parasites, bacteria and fungi in the stool. By increased mucosal friability.

Differential diagnosis: by distinguishing it from amebiasis, Crohn's disease, Behçet's Syndrome, bacillary dysentery, tuberculosis, Whipple's disease, ischemic colitis, chronic laxative abuse, etc.

Treatment: symptomatically and by administering diphenoxylate, loperamide, broad spectrum antibiotics, metronidazole, sulfasalazine, olsalazin, mesalamin, corticosteroids, surgery. The killing of the parasite can occur by administering metronidazole, quinacradine, mebendazole, pyrantel, praziquantel, niclosamide, yomesan etc.

RRF method in case of gastroenteritis: detects parasites, bacterium, viruses, fungi, and eliminates them!

RFR method should only be used after the beginning of the antibiotic, antifungal, and antihelmetic treatments and together with them.

The most frequent resonances found in case of gastroenteritis and other inflammative bowel diseases are: 288, 330-339, 346-354, 372, 380-383, 389, 390-393, 396-397, 401, 409-410, 418, 425-435, 442-451, 457-459, 487, 511, 544, 555-558, 560-578, 580-590 kHz

RFR method: detects and may eliminate the pathogen micro- and macroorganisms. In this chronic syndrome one can find many different resonances of the bacteria. Use RFR method together with drug therapy.

The most frequent resonances are: 290-293, 329-334, 344-345, 354-356, 365-375, 374, 390, 416-420, 442-451 kHz

The first step of the elimination is the eliminating of the Mycoplasma species.

The most frequent resonances in case of polyposis and pseudopolyposis are: 299, 318, 332, 341, 348, 353, 372, 410-412, 461, 476, 534-535, 544, 555 kHz

The most frequent frequencies of the adenocarcinoma of the colon are: 427-437 kHz

13.6.7. Celiac Disease

Celiac disease (CD) is an autoimmune disorder of the small intestine affecting genetically predisposed individuals. Triggered by a well-identified antigen i.e. gluten and its related prolamins the illness can progressively lead to the flattening of the small intestinal mucosa. The genetic susceptibility to this celiac disease is coded in well-identified haplotypes in the human leukocyte antigen (HLA) class II regions: DR17, DR5/DR7 and DR4, one of which can be expressed on the antigen-presenting cells of the mucosa as well. The antigen presenting cells of approximately 90% of the affected patients express the DQ2 heterodimer. The pathology of this illness is multifactorial. Activated Intraepithelial lymphocytes (IELs), enterocytes activated by stress and inflammation expressing an enhanced amount of MHC-I on their surfaces and the highly expressed cytokine IL-15, autoantibodies agaist tissue transglutaminase enzymes wich all have a significant role in causing damages to the epithelial cells of the small intestine. The T lymphocytes, infected by HTLVs can cause an „abnormal immune reaction" to the ingested gluten antigen among genetically predisposed individuals. Certain other environmental factors, related f.i. to the feeding practices regarding infants and to intestinal infections got in the early ages of the persons, can be also pathologic co-factors. All these factors may trigger the HTLV-infected

T lymphocytes to give the starting signal for the production of autoantibodies to the intestinal mucosa infected f.i. by *mycoplasma*, *adenovirus 12*, etc.
Celiac disease can be developing into an autoimmune disease, where the enzyme tissue transglutaminase (tTG) is the autoantigen to which the „abnormal immune response" is directed.
Three cereals contain gluten, toxic for patients with celiac disease: wheat, rye and barley. In case of CD there are often different infections of the small intestine present f.i. *Candida, Clostridium difficile, Mycoplasma fermentans, HTLV, Giardia lamblia, Herpes viruses and Adenoviruses*. The question however is, which of them has an important role regarding the developement of immune inflammations and the destruction of epithelial cells. Classic symptoms of the coeliac disease include diarrhea, loss of weight, anemia, loss of iron absorption and fatigue. Though coeliac disease is primarily a bowel disease, the bowel symptoms may also be limited or even absent. Some patients are diagnosed with symptoms related to the decreased absorption of nutrients or with various symptoms, which, though statistically linked, have no clear relationship to malfunctioning bowels. Given this wide range of possible symptoms, the classic triad is no longer a requirement of diagnosis. In case of a celiac diesease the autoantibodies can be detected by serology; even in absence of symptoms. Arthritis, arthralgia and osteoporosis and the skin disease Dermatitis herpetiformis Duhring can be common extraintestinal symptoms regarding adult patients with CD.
CD can be associated with some psychiatric disorders, too, such as depression and anxiety. These conditions can be severe but usually respond well to a gluten-free diet. CD leads to an increased risk of both adenocarcinoma and lymphoma of the small bowel, which risk can return to the baseline with dietary changes. Its frequently associated diseases are Down syndrome, Williams syndrome and Turner syndrome.
Diagnosis: there can be made four special serological blood tests when diagnosing, i.e. the detecting of IgA type antibodies against particular antigens present in the small bowel: i.e. anti-R-1-reticulin, anti-gliadin, anti-endomysium and anti-tissue transglutaminase. Generally, serology may be unreliable regarding young children, though an anti-gliadin test can be of value concerning children under five years. Serology tests are based on indirect immunofluorescence methods or ELISA.
Treatment: A lifelong avoidance of gluten ingestion is the cornerstone treatment for patients with celiac disease. Wheat, rye and barley are the grains that contain toxic peptides. Corticosteroids can rapidly control the severe symptoms of CD.
In case of a Clostridium difficile coinfection the administration of Metronidazole and/or Vancomycin is advised.
RFR method: detects and may eliminate all pathogen microorganisms present.
The most frequent resonances are: 291-293, 343-353, 370-375, 395-390, 421-426, 442-451, 572-580 kHz

13.7. Staphylococcal Food Poisoning

Staphylococcal food poisoning is an illness caused by eating food, contaminated the toxins of certain types of *staphylococci.* Symptoms include nausea, vomiting, abdominal cramping, diarrhea, headache and fever. Severe fluid and electrolyte loss may occur and cause weakness and a very low blood pressure. This food poisoning can sometimes be fatal, especially when affecting very young and elderly patients, and people weakened by long term illnesses.
Diagnosis: by bacterium culturing.
Treatment: symptomatically, by substituting fluids, electrolytes and by administering Cephalosporine, Vancomycin, etc.
The most frequent resonant frequencies are: 324-332, 345, 372-382, 397, 402, 434, 445, 462, 482, 491, 537, 557-567 kHz

13.8. Botulism

Botulism is an uncommon, life-threatening poisoning caused by the toxins of Clostridium botulinum. These neurotoxins, the most potent poisons known, can severely damage the nerves and the muscles. Clostridium botulinum species can appear also in form of spores. These spores can exist in dormant state for many years and can not easily be destructed. The first symptoms of the illness are dry mouth, double vision, drooping eyelids and an inability to focus on nearby objects. The pupillar muscles can not constrict normally if exposed to light during an eye examination; or can not constrict at all. Nausea, vomiting and diarrhea can also come about. Some people don't have gastrointestinal symptoms, particularly those patients with wound botulism. Infected patients may have difficulty in speaking and swallowing. The paralysis of the nerves can lead to the inhalation of food so that aspiration pneumonia can develop. The muscles of the arms, legs and those involved in breathing weaken progressively.

Diagnosis: symptomatically, the diagnosis can be confirmed by toxin tests and bacterium cultures from the feces.

Treatment: by antitoxin, gastric lavage. By administering Cephalosporin, Doxycyclin, Chloramphenicol, Clindamycin, Symptomatically, by substitution of plasma, fluids, electrolytes, etc.

RFR method should only be used together with the beginning of the antibioticum treatment.

The most frequent resonant frequencies are: 327, 349-353, 360-367, 530-544 kHz

13.9. Food Poisoning Caused by Clostridium Perfringens

This type of gastroenteritis is caused by eating food contaminated by enterotoxins produced by *Clostridium perfringens*. This gastroenteritis is usually mild, causes abdominal cramping and diarrhea, though it can develop into a severe disorder, with abdominal distension from gas, severe diarrhea, dehydration and shock as well.

Diagnosis: By bacterium culturing. Spores are rarely observable in smears of exudates.

Treatment: by administering f.i. Cephalosporin, Doxycyclin, Chloramphenicol. Clindamycin, etc. and symptomatically, by giving plasma, fluids, electrolytes, cardiac drugs, etc.

RFR method: should only be done after the beginning of the antibioticum treatment! Detects and eliminates the Clostridium.

The most frequent resonant frequencies are: 325, 344-348, 360-368, 394-400 kHz

13.10. Food Allergies and Intolerances

Food allergies belong mostly to the first category allergic reactions, and are adverse immune responses to food proteins. Their symptoms can include itching of and in the mouth, vomiting, hives and asthma bronchiale. In some cases these reactions can be so severe that they cause serious illnesses which might even leave to death. (See Chapter 23.6.)

Food intolerances, belong to the second category reactions and include all adverse responses to food and food additives that are not immunologically mediated (as f.i. lactose intolerance, bacterial food poisoning, pharmacologic reactions, etc.).

Many a food protein can behave in the human body as an antigen. The milk proteins of cows often cause food allergy in infancy. The first category reactions can be allergies mediated by IgE antibodies (f.i. IgE-mediated reaction to peanuts), which begin during or soon after exposure to food, while others are resulted from non–IgE-mediated mechanisms

(f.i. the protein-induced enterocolitis syndrome), which generally take several hours to develop.

The IgE-type antibodies bind themselves to high-affinity IgE receptors of circulating basophils and of tissue mast cells present in the skin, the gastrointestinal tract and the respiratory tract. The subsequent allergen exposure binds two adjacent IgE antibodies together, resulting in receptor cross-linking and intracellular signaling that initiates the release of numerous mediators, including histamine, various prostaglandins, leukotrienes, chemotactic factors and cytokines. Their effect on the surrounding tissues leads to vasodilatation, smooth muscle contraction and mucus secretion, which, in turn, are responsible for the spectrum of clinical symptoms observable during allergic reactions to food. *Mycoplasmas and Human T Lymphocyte viruses* are microorganisms playing a most important role in the development of immune system dependent food allergies (see also Chapter 23.6.).

The major food allergens are water-soluble glycoproteins that are resistant to heat, to acids and enzymes. The gastrointestinal (GI) tract is impermeable to some intact antigens. The antigen uptake is an endocytotic process, involving intracellular lysosomes. Some antigens can pass through the intercellular gaps; the penetration of antigens through the mucosal barrier is usually not associated with clinical symptoms. Under normal circumstances, the food antigen exposure in the GI tract results in a local immunoglobulin A (IgA) response and in the activation of suppressor $CD8^+$ lymphocytes residing in the gut-associated lymphoid tissue (causing thus oral tolerance). In case of genetically susceptible children, or owing to other as-yet-unknown reasons, this oral tolerance does not develop, and different immunologic and inflammatory mechanisms can occur. Whether nonimmunologic mechanisms have a role in the development of specific intolerances to food proteins is still disputable.

Some evidence suggests that a reduced microbial exposure during infancy and early childhood results in a slower postnatal maturation of the immune system and delays the progression to an optimal balance between immunity T_H1 and T_H2 (hygiene hypothesis). The T_H1/T_H2 imbalance is crucial to the clinical manifestation of allergy and asthma. The immunologic background of asthma and food allergy is similar. Certain genetic variations in receptors for bacterial products may be associated with allergic sensitization. On the other hand, certain intestinal infections may increase the paracellular permeability, allowing the absorption of food proteins without being epithelially processed. As a consequence, *mycoplasmal and HTLV* infections can be important contributors of the pathogenesis of food protein allergies. *Human T-cell Lymphotropic Viruses and Human B-cell Lymphotropic Viruses* play a most important role in the food allergy of genetically susceptible persons.

The antigen uptake has been found to be increased in children with gastroenteritis and with allergy to cow's milk. Local production and systemic distribution of specific reaginic IgE plays a significant role in the IgE-mediated reaction to food proteins. Intraepithelial lymphocytes have an important role in the pathogenesis of GI food allergy. The pathogenetic role of eosinophils in food-induced eosinophilic GI diseases has not yet been defined. The occurrence of IgG-food protein antibodies is well known. However, their actual role in the pathogenesis of clinically relevant symptoms is, at best, doubtful. The classic Ig-E-mediated food allergies are classified as immediate hypersensitivity reactions type-I. The onset of these allergic reactions is acute (from seconds to one hour) and may cause angioedema (of the face, lips, tongue, eyelids, larynx, trachea), hives, itching, rhinorrhea, nausea, vomiting, diarrhea, stomach cramps and abdominal pain. These symptoms are termed gastrointestinal hypersensitivity or anaphylaxis.

This allergic reaction may progress to an anaphylactic shock, a systemic reaction with hypotension involving several different organs and might even lead to death. Most

frequently associated allergens are peanuts, nuts, milk, eggs and seafood, though many more food allergens have been reported as triggers for anaphylaxis.
Food allergy is thought to develop more easily among patients with atopic syndrome. Patients suffering from this syndrome have a strong inherited predisposition, so that a family history of allergic diseases is indicative of atopic syndrome.
Conditions caused by food allergy are classified into 3 groups according to the mechanism of the allergic response, and can be:
1. IgE-mediated (classic): immediate hypersensitivity reaction Type-I.
2. IgE and/or non-IgE-mediated: Allergic eosinophilic gastritis, gastroenteritis and esophagitis
3. Non-IgE mediated: Food protein-induced enterocolitis syndrome such as coeliac disease.
Milk and/or soy protein intolerance (MSPI) is a non-medical term used to describe a non-IgE mediated allergic response to milk and/or soy protein during infancy and early childhood. Symptoms of MSPI are usually attributable to food protein proctocolitis.
Heiner syndrome is a lung disease due to formation of milk protein/IgG antibody immune complexes (milk precipitins) in the bloodstream after being absorbed from the gastrointestinal tract.
The most frequent food allergies are:
Cereal allergy: Quite a number of cereals, such as wheat, rye, barley, oats may cause allergic reactions among sensitive children and adults.
Coconut allergy: Allergy to coconut is rare, but can nevertheless cause severe allergic reactions (including anaphylaxis) among sensitive persons. A small number of people who are allergic to nuts react likewise to coconut.
Coeliac disease: Gluten is the mixture of proteins found in some cereals, including wheat, rye and barley. Gluten intolerance, or coeliac disease is a lifelong disease caused by sensitivity to gluten, in case of which the lining of the small intestine, hindering the body from absorbing nutrients, will get damaged causing diarrhoea and eventually malnutrition. Coeliac disease can run in families. This disease, if untreated, can lead to anaemia and bone disorders. It can also cause growth problems concerning children. People with diabetes Type 1, thyroid problems, ulcerative colitis and certain neurological disorders, f.i. epilepsy are liable to suffer from coeliac disease. There is no cure for coeliac disease. The only way to avoid its symptoms is not to eat foods containing gluten, such as wheat, rye, barley, malt, malt extract, malt flavouring, beer and lager. Processed food can contain hidden gluten. Coeliac disease is often diagnosed after weaning, when cereals are introduced into the diet, but it can also be diagnosed at a later age.
Egg allergy
Like most food allergies, egg allergy is more common in childhood and about half of the children who have it will grow out of it by the age of three. In some cases, egg allergy can cause anaphylaxis. Egg allergy is caused mainly by three proteins of egg white, named ovomucoid, ovalbumin and conalbumin. Cooking can destroy some of these allergens, but not all of them. Thus some people might badly react to cooked eggs, as well as raw eggs. Occasionally some people with allergy to chicken, quail or turkey meat, or to bird feathers may also badly react to eggs. This phenomenon is termed bird-egg syndrome.
Fish allergy: Fish allergy can often cause various severe reactions, including anaphylaxis. Adults are more liable to have allergic reaction to fish and shellfish than children, probably because they eat these foods more often. People who are allergic to one kind of fish, such as cod, often react to other kinds of fish such as hake, haddock, mackerel and whiting as well, as the allergens of these fishes are quite similar. Cooking doesn't destroy fish allergens. Some people with fish allergy can be allergic only to cooked but not to raw fish.
Fruit and vegetable allergy: Allergic reactions to fruits and vegetables are usually mild, often affecting but the mouth, causing itching or a rash where the food touches the lips and mouth. This phenomenon is named oral allergy syndrome. A number of people who react

in this way to fruits or vegetables will also react to tree and weed pollens. Thus, people allergic to birch pollen are also likely to be allergic to apples. Cooking can destroy a number of the allergens in fruits and vegetables, so that cooked fruit often does not cause any reaction in people with allergy to fruit. For the same reason, pasteurized fruit juice will not cause any allergic reactions. However, the allergens in some vegetables, such as celery, will not be destroyed by cooking.

Milk allergy: Allergy to cows' milk is the most common food allergy in childhood. It's more common in babies with atopic dermatitis. Reaction against it can be triggered by offering even small amounts, either by passing to the baby through the mother's breast milk from dairy products she has eaten, or by feeding cows' milk to the baby. Most of the children usually grow out of milk allergy by the age of three, but about a fifth of them remains always allergic to it. The symptoms of milk allergy are often mild and can affect any part of the body. They can include rashes, diarrhoea, vomiting, stomach cramps and difficulty in breathing. In but very few cases, milk allergy can cause anaphylaxis. Cows' milk allergy is caused by a reaction to a number of allergens present in cows' milk, such as casein and whey.

Milk from other mammals (such as goats and sheep), and hydrolyzed milk and soy formulas, are sometimes used as a substitute for babies who are at risk of developing cows' milk allergy.

Milk protein intolerance: Intolerance to cows' milk protein is common among babies and children, the symptoms of which start from the time when cows' milk is first introduced into their diet. There is no cure for it and the only way to stop the symptoms is to avoid eating cows' milk products. Cows' milk protein intolerance is different from lactose intolerance and milk allergy.

Lactose intolerance: Lactose is a sugar found in milk. It's important to distinguish between lactose intolerance and milk allergy, because milk allergy can cause severe reactions. Lactose intolerance is caused by a shortage of the enzyme lactase, needed to break down lactose so that it can be absorbed into the bloodstream. If someone doesn't have enough of this enzyme, lactose isn't absorbed properly from the gut, causing symptoms like bloating and diarrhoea.

Lactose intolerance can be caused by many a factor. In humans, after the age of two the body produces less lactase. Digestive diseases or injuries of the small intestine can sometimes cause lactose intolerance by reducing the amount of lactase enzime. Milk from mammals f.i. cows, goats, sheep and humans do contain lactose, meaning that goats' milk and sheep milk are equally unsuitable as cows' milk for people who are intolerant to lactose. There is no medical treatment for lactose intolerance, but symptoms can be avoided by controlling the amount of lactose in the diet. Adults with lactose intolerance can often drink some milk without getting any symptoms.

Latex-food syndrome: Latex allergy is caused by a reaction to a number of allergens found in natural rubber or latex. Recently, the number of people with latex allergy has increased, particularly among healthcare workers and people with spina bifida, coming into contact with a lot of latex products. Latex contains many allergens similar to the allergens of some foods. Thus, people allergic to latex might also badly react to foods such as banana, mango, kiwi, chestnut, paprika, celery, apple, carrot, cherry, coconut, strawberry and avocado. This reaction is termed latex-food syndrome.

Lupin allergy: Lupins are common garden plants, related to legumes such as peas, lentils and beans. Many a type of lupin seed is poisonous, as they contain bitter-tasting toxins. Sweet lupins do not contain these toxins, they can be eaten by humans or livestock. Sweet lupin seeds are being used more and more in order to replace cereal grain in many food products, as flour and pasta.

Maize allergy: For people, who are sensitive to maize, it is most difficult to avoid eating it, as maize is commonly used in a wide variety of food products.

Meat allergy: People with meat allergy might badly react to beef, mutton, pork or chicken. Those, who are allergic to one type of meat or poultry might sometimes also badly react to some other kinds. Cooking destroys some of the allergens in meat, some people nevertheless will still badly react to cooked meat. Processed meat, such as frankfurter, luncheon meat and pâtés, sometimes contain other ingredients, particularly milk products, as emulsifiers or flavour enhancers, which all have to be taken into account.
Nut allergy: Allergy to nuts of trees remains usually lifelong. Nuts causing allergic reactions are most likely walnuts, hazelnuts, almonds, pecans, Brazil nuts, pine nuts, macadamia nuts and cashew nuts. Rarely do all these nuts cause anaphylaxis in people who are sensitive to them. Sometimes people with an allergy to one type of nut will also react in the same way to other nuts.
Peanut allergy: Allergy to peanut remains often lifelong. Peanuts are one of the most common causes of food allergy and can cause severe reactions, including anaphylaxis. They contain a number of allergens not destroyable by cooking or roasting. Peanut allergy can be so severe that very small amounts can cause bad reactions. Owing to this, even coming into contact with traces of peanut can cause a bad reaction in people who are sensitive to it. Somebody, for example can come into contact with traces of peanuts taken from unrefined oil, or when food is served using utensils formerly containing peanuts, or even if being close to someone eating peanuts. Refined peanut oil is thought to be harmless for people with peanut allergy, because the proteins causing allergic reactions got removed during the manufacturing process. However, cold-pressed, or unrefined/unprocessed (crude) peanut oil can contain small amounts of peanut allergens, which can cause bad reactions in people who are sensitive to them.
Pine nut allergy: Pine nuts can cause severe allergic reactions, such as anaphylaxis, in people who are sensitive to them. People who are allergic to pine nuts might also react to peanuts and nuts such as almonds.
Rice allergy: People allergic to rice can react by eating it or when inhaling its pollen. Rice can cause hayfever symptoms in areas where it's grown in great amounts. People who are allergic to rice can sometimes badly react to a number of other foods from the same botanical family, such as barley, maize, wheat, oats and rye, as well as other foods such as peach and apple.
Sesame allergy: Sesame allergy can be severe, causing even anaphylaxis. People with sesame allergy might also react to poppy seeds, kiwi fruit, hazelnuts and rye grain.
Shellfish allergy: Allergy to shellfish is rather common, and there exists a number of different types of shellfish, for example shrimps, prawns, lobster, crab, crayfish, oysters, scallops, mussels and clams, all able to cause reactions. People who are allergic to one type of shellfish often react to other types, too. Shellfish allergy can often cause severe reactions, some even react to the vapours when cooking shellfish.
Soya allergy: Soya allergy is a common childhood allergy. Most people grow out of it by the age of two, but sometimes even adults are allergic to soya. The symptoms of soya allergy are similar to milk allergy, f.i. rashes, diarrhoea, vomiting, stomach cramps and breathing difficulties. Some people with soya allergy might also badly react to milk. Very rarely, soya can even cause anaphylaxis.Soya is used as an ingredient in about two-thirds of all manufactured food products, including bakery goods, sweets, drinks, breakfast cereals, ice cream, margarine, pasta, processed meats and seasoned foods.
Spice allergy: Allergic reactions to spices are rare and usually mild, severe reactions can happen occasionally. Some people badly react to mustard, coriander, caraway, fennel, paprika or saffron and, less frequently, to onions, garlic or chives. Reactions to mustard have been reported to cause anaphylaxis, particularly in Europe, where mustard is commonly used. The allergens in spices are similar to those in pollens and vegetables, and people who are allergic to mugwort and birch are more likely to be sensitive to spices.

Vegetable oil allergy: Vegetable oil is usually a blend of oils. The refining process removes proteins from the oil. Since it are the proteins in oils that can cause allergic reactions, sensitive people probably will not react to refined oils. Cetain special oils, such as sesame and walnut, are not refined oils, so it is best to avoid them by people who are sensitive to nuts or seeds they are made from.
Wheat allergy: Wheat allergy is particularly common among babies. One of the main allergens in wheat is a protein called gliadin, which is contained in gluten. Owing to this, people with wheat allergy are told to eat a gluten-free diet.
Diagnosis: by allergy testing methods, IgE level measuring, looking for eosinophilia, by testing microbiological triggers (f.i. mycoplasmas, HTLV), and by examining the pathological and friendly flora of the bowels.
Differential diagnosis of food allergy:
1. Lactose intolerance is developing generally later in life though in severe cases it can even be present in young patients. This is caused by a lactase enzyme deficiency and not by allergy.
2. Celiac disease is an autoimmune disorder triggered by gluten proteins such as gliadin. It is a non-IgE mediated food allergy by definition.
3. Irritable bowel syndrome.
4. C1 esterase inhibitor deficiency.
Treatment: by eliminating the allergens, by administering antihistamines, corticosteroids, etc.
RFR method can detect HTLV and other pathologic microorganisms.
The most frequent resonances are: 297-299, 311-312, 314-319, 321, 330, 339, 359, 370-374, 442-451, 453-455, 459-464, 482, 485-490, 493-496, 574 kHz

13.11. Flatulence

Flatulence is the term of the anal expulsion of a mixture of gases, which are byproducts of the digestion process of the affected person. Flatus is brought to the rectum by the same peristaltic movements which cause feces to descend from the large intestines. Some of these gases, f.i. methane and hydrogen are flammable, some others, f.i. nitrogen, air, carbondioxide are unflammable.
The gas released by a flatus event has usually an unpleasant odor. For many years, this was thought to be due to skatole and indole, which are byproducts of the digestion of meat and sulfur-containing compounds, such as methanethiol, hydrogen sulfide and dimethyl sulfide. In contrast, the flatulence-producing foods (f.i. beans, lentils, dairy products, onions, garlic, scallions, leeks, turnips, rutabagas, radishes, sweet potatoes, potatoes, cashews, etc.) are typically high in certain polysaccharides and oligosaccharides such as inulin. In case of beans, endogenous gases can to arise from complex oligosaccharides due to methane-producing bacteria *Methanobrevibacter smithii* inhabiting the digestive tract. In case of people suffering from lactose intolerance, the intestinal bacteria feeding on lactose can give rise to an excessive gas production if milk or lactose-containing substances are being consumed. Excessive amount of intestinal gases can develop in case of aerophagia or due to certain intestinal bacteria. The latter case can be associated with malabsorption syndromes or significant constipation but is more frequently a consequence of eating foods such as broccoli, and cabbage which have a high content of nondigestible polysaccharides. The oligosaccharides stachyose and raffinose, isolated from beans, are particularly effective substrates for fermentation into carbon dioxide, hydrogen and methane by the colonic flora. The treatment of flatulence is generally undertaken in order to reduce embarassment and the patient should decrease his aerophagia and has to avoid eating food causing excessive amounts of gases. The noises commonly associated with flatulence are caused by the vibration of the anal sphincter and occasionally by the closed buttocks. Nodus hemorrhoidales can modify these noises as well. Malabsorption is the clinical term

encompassing the defects occurring during digestion and absorption of food nutrients. The digestion or absorption of a single nutrient component may be impaired, as it is in case of lactose intolerance caused by lactase deficiency. In case of complex intestinal disorders, such as celiac disease or Crohn's disease, the absorption of almost all nutrients is impaired. The resulted symptomes, such as diarrhea and weight loss are common, the specific causes of malabsorption are usually established based on physiological evaluations. The treatment depends often on the definitive etiology of malabsorption.
Pathological bacteria, such as certain *E. coli species, Clostridium difficile, Salmonella enteritidis, Pseudomonas aeruginosa, etc* can cause flatulence in the intestines.
Flatulence can be one of the symptoms of intestinal protozoal infections (f.i. amebic or balantidial colitis, giardiasis, spore-forming protozoal infections, dientamoebiasis and blastocystosis) as well.
The overgrowth of *Candida albicans* in the intestines frequently causes flatulence.
Treatment: by eliminating the pathogen gas-producing bacteria.
Acidophilus and Bifidus species are friendly bacteria inhabiting the large intestines, and can help to hold the growth of gas-producing bacteria in check. The replenishing these bacteria will often relieve flatulence, bloating and other digestive complaints. Activated charcoal can absorb gas in the intestine and can help to reduce the odor.
RFR method: can help to identify and eliminate the gas-producing microbiological agents.
The most frequent resonances are: 323-327, 355-357, 372, 392, 384-390, 396, 402, 410, 443-450, 572-586 kHz
As to the frequencies of intestinal protozoa, see their special Chapters.
As to the frequencies of Candida albicans, see its special Chapter.

13.12. The Role of the Bowel and the Peyer's Patches in Immune-Autoimmune Responses and in the Carcinogenesis

The pathological bacterium flora of the bowel has an important role in the development of autoimmune, allergic and cancerous processes. The bowel flora plays an important role concerning our ability to fight down infectious diseases, provides a front line in our immune defense and a passive mechanism to prevent infections and produces many vitamins. Acid-producing lactobacilli and bifidobacteria increase the bioavailability of minerals (calcium, copper, iron, magnesium, manganese), which require acid for their absorption. With no healthy colony of bowel flora, we cannot expect to gain robust health and wellbeing.
The gastro intestinal tract (GIT) provides distinct niches for colonization by commensal bacteria, as indicated by qualitative and quantitative differences in the bacterial flora throughout the GIT. The building up of the GIT in different ways reflects its functional role in the digestion, in the immune, autoimmune and allergic responses as well as in case of intestinal cancer. The lymphoid tissue throughout the GIT plays an important role in the localized immune responses to bacteria. The development of a strong localized gastrointestinal mucosa-associated immune system does not only depend on a healthy mucosal lymphatic tissue but also on the presence of the bacterial microflora. The gut flora is an integral part of our immune system. If the bowel flora colonies become dysbiotic, autoimmune or allergic conditions such as inflammatory bowel diseases, allergic skin processes and different autoimmune processes might arise. Dysbiosis is the name for the abnormal microbial colonization of the intestine, where the changes in quality and/or quantity are pathological.
Peyer's patches are localized mucosal lymphoid structures with different densities usually found in the lowest portion of the small intestine ileum in case of humans thus differentiating the ileum from the duodenum and the jejunum. In case of adults, B

lymphocytes predominate in the germinal centers of the follicles. T lymphocytes are found in the zones between the follicles. The gastrointestinal tract is exposed to the external environment, a great part of it is populated with potentially pathogenic microorganisms such as *Escherichia coli, Salmonella thyphimurium, S. paratyphimurium, Shigella flexneri, S. sonnei, S. dysenteri, Candida albicans* and others. Peyer's patches establish thus their importance in the immune surveillance of the intestinal lumen and in the immune response within the mucosa. Pathogenic microorganisms and other antigens entering the intestinal tract encounter macrophages, dendritic cells, B-lymphocytes and T-lymphocytes found in Peyer's patches and in other gut-associated lymphoid tissues (GALT). The pathological, GIT bacteria-induced intracellular signaling events are involved in the development of cancer. The development of cancer is characterized by the accumulation of gene modulations, including various gene mutations. These infections enhance the aberrant DNA methylation in the human cells which can participate in gastric carcinogenesis by silencing the tumor suppressor genes. These infections cause a chronic bowel inflammation as well as the atrophy of the mucosal tissue and the Peyer's patches. Peyer's patches have an important role in the adsorption from the gut and may hence be a likely target organ for lymphoid carcinogenesis beginning due to an oral exposure of carcinogenic substances.

Some bowel bacteria can produce certain bacterial factors able to develop cell mutation as well as to develop a cancerous process, if the human cell is infected with *HPV* and *Mycoplasma fermentans.*

As the gut flora has an important role in the development of the illnesses mentioned , the treatment of which can only be made effective by eliminating the pathological flora and by restoring the friendly flora in the GIT.

Parasitic diseases are defined as being caused by protozoa or helminths. To understand their life cycle it is essential to explain the pathophysiology of the diseases caused by these organisms. The diarrhea caused by intestinal protozoa, their ability to invade into the tissues, and/or their effects on the intestinal epithelium are associated with direct cytotoxic effects. Intestinal protozoa may provoke allergic processes. IgA immuneglobulins and T-cell responses are important in case of *giardiasis* and in case of infections caused by *spore-forming protozoa.*

Candidiasis may also be considered as a causative factor of allergic processes. A pathological bacterial or fungal flora of the bowel may develop if the content of the bile is abnormal. A complete bile acide structure is needed to maintain a healthy bowel flora. A healthy bowel flora can protect the person against allergic or autoimmune responses and against carcinogenesis.

14. DISORDERS OF THE LIVER AND OTHER DIGESTIVE ORGANS ASSOCIATED WITH INFECTIONS

Diseases associated with liver damage can appear in many a different form, such as jaundice, cholestasis, liver enlargement, portal hypertension, ascites, liver encephalopathy and liver failure. The bile, produced by the liver, is stored in the gallbladder until utilized by the digestive system. The bile duct system can be easily infected by pathogen bacteria, viruses, fungi and macro parasites, which microorganisms may get into the liver. Not every liver disease is caused by an infection, and not all pathogens infecting the liver originate from the bile system, f.i. Viruses causing hepatitis infect the liver via the blood.

14.1. Hepatitis

Hepatitis is an inflammation of the liver due to any cause whatever. This inflammation may be caused by noninfectious agents and by various pathogens such as viruses, bacteria, fungi and parasites, and in case of a genetic predisposition certain infections together can lead to an autoimmune hepatitis.

Medicaments and toxins may cause noninfectious hepatitis. The most common noninfecting agents causing hepatitis are toxins: f.i. alcohol, toxins of mushrooms (i.e. amatoxin, fallotoxin, virotoxin and giromitrin), certain herbaceous plants (f.i. asafetida) and carbon tetrachloride.

Liver damages can occur due to drugs, f.i. paracetamol, amoxicillin, antituberculosis medicines, minocycline, methyl-dopa, nitrofurantoin, isoniazide, ketoconazole etc. Ischemia and other circulatory insufficiencies also can cause hepatitis. The most common causes of hepatitis are the alcohol and drugs, but nevertheless, there are often viral components also involved in its pathology.

There are *various pathogens*, infecting the liver. Five types of the *hepatotropic viruses* cause the 95% of the clinical cases of viral hepatitis. These are *Hepatitis A virus (HAV)*, *Hepatitis B Virus, Hepatitis C Virus, Hepatitis D virus (HDV)*, and *Hepatitis E* virus *(HEV). Herpes simplex virus, Cytomegalovirus, Epstein-Barr Virus, yellow fever virus* and *adenoviruses* can also cause an inflammation of the liver.

The *non viral infections* of the liver can be caused by *toxoplasma, mycoplasma, leptospira, treponema pallidum, Borrelia B.s.l., Coxiella burnetii* (Q-fever), and *Rickettsia* (rocky mountain spotted fever).

Hepatitis is often present in auto immune conditions (f.i. Systemic Lupus Erythematosus) and in metabolic diseases (f.i. Wilson's disease) as well as in certain hereditary dieseases f.i. alpha 1-antitrypsin deficiency.

14.1.1. Acute Viral Hepatitis Infections

Acute viral hepatitis is an inflammation of the liver caused by infection with one of five hepatitis viruses. This inflammation begins in most cases suddenly and lasts but for a few weeks. An acute viral hepatitis is a systemic infection affecting predominantly the liver.

Hepatitis A viruses (causing infectious hepatitis, short time incubation hepatitis or MS-1 hepatitis) spread primarily from the stool of an infected person to the mouth of another. Such transmission is usually the result of poor hygiene. Waterborne and food borne epidemics also are common, especially in developing countries. Isolated cases arising from person-to-person contact can also come about. The infections with Hepatitis A virus are usually symptomless and remaining unrecognized.

Hepatitis B infection is also referred to as serum hepatitis, long time incubation hepatitis, MS-2 hepatitis, or Hepatitis B surface antigen (HbsAg) positive hepatitis. This acute viral hepatitis can cause symptoms of a minor flu like illness, and even those of a fatal liver failure as well. In general, Hepatitis B is more serious than Hepatitis A, being occasionally fatal, especially among elderly people and infants. The acute illness is usually mild, though the liver function may improve but later on can get repeatedly worse for several months.
Hepatitis C viruses often cause chronic hepatitis; about 80 percent of the acute Hepatitis C cases will become chronic. The hepatitis cases arising from blood transfusions are mostly caused by this Hepatitis C virus, and so are many scattered cases of acute hepatitis as well. Chronic hepatitis, cirrhosis and liver cancer can all be complications caused by this virus.
Hepatitis D viruses can cause less common diseases and only together with Hepatitis B viruses. These co-infections are usually more severe, prolonged, causing chronic liver damages and/or even cancer.
Hepatitis E viruses are responsible for occasional epidemics similar to those caused by Hepatitis A viruses. These infections are mild, and do not get chronic.
Researchers suspect that other viral agents (not completely characterized or identified) may cause a small percentage of the cases of acute hepatitis. These agents are referred to as **Hepatitis F** (HFV), **Hepatitis G** (HGV), or **other non-ABC viruses**. Less commonly, hepatitis results from other viral infections, such as *Epstein-Barr Virus*, *cytomegalovirus* and *yellow fever* infection.
The **symptoms** of acute viral hepatitis usually begin suddenly, are various and systemic such as nausea, vomiting, fatigue, malaise, arthralgias, myalgias, headache, loss of apetite, diarrhea and photophobia. With the onset of clinical jaundice these prodromal symptoms usually are diminished, though in some cases a mild weight loss and a yellowish skin together with general itching can occur during the icteric phase.
An acute hepatitis is usually mild, though the liver functions may worsen repeatedly for several months. A person with an acute viral hepatitis can become a chronic carrier of the virus. A carrier person has no symptoms but is still infected. This state can only occur in case of infections of Hepatitis B and C viruses.
Diagnosis: symptomatically, by the increase of the serum transaminase levels (ie. aminotransferases; SGOT and SGPT) during the prodromal phase of the acute viral hepatitis preceding the rise of bilirubin level. By biopsy, serology etc.
Differential diagnosis: by distinguishing it from other viral diseases f.i. caused by EBV, CMV, HSV and Coxsackie virus and from toxoplasmosis, etc.
Treatment: symptomatically. By administering iv. Immuneglobulin, cytokine therapies etc. In case of Hepatitis B and C the prognosis is bed, as the treatment is not able to kill the virus.
RFR method: detects and may eliminate the virus!
The most frequent resonant frequencies of Hepatitis A Virus are: 285-295, 320-330, 340-356, 361, 366, 403, 420-436, 449, 487-488, 498, 570-590 kHz
The most frequent resonant frequencies of Hepatitis B Virus are: 293, 340, 372, 384, 392-420, 444-450, 488 kHz
The most frequent resonant frequencies of Hepatitis C Virus are: 324-339, 350-352, 370-374, 396, 400-402, 450-456, 475-482, 540-541, 559-563 kHz
The most frequent resonant frequencies of the Hepatitis D and Hepatitis E viruses are: 348, 375, 386, 410, 432, 450, 468, 471, 490, 532, 535-548, 550-563, 580 kHz
The most frequent resonant frequencies of Cytomegalovirus are: 305, 349, 406-412, 512, 530-536, 548-550 kHz
The most frequent resonant frequencies of Epstein-Barr Virus are: 337-339, 342-347, 352, 372-382, 397-398, 422-424, 491, 516-519, 528, 560 kHz

14.1.2. Chronic Hepatitis

Chronic hepatitis results usually from infections, most often of viral origin, such as caused by Hepatitis B with or without Hepatitis D viruses and Hepatitis C. (Hepatitis A and E do not lead to chronic disease). Chronic hepatitis forms with other etiology are the alcoholic or non-alcoholic steatohepatitis (the latter caused mostly by drugs), the autoimmune hepatitis and the hereditary diseases affecting the liver.

Symptoms: The majority of patients remain asymptomatic or mildly symptomatic, abnormal blood tests being the only signs of the illness. These features are related to the extent of the liver damage and the cause of the hepatitis. The return of symptoms related to acute hepatitis can be experienced, Jaundice is usually a late feature indicating extensive damages. A feeling of abdominal fullness results from the enlarged liver or spleen, low grade fever and ascites can also come about. Extensive damages and scarring of the liver can lead to cirrhosis, weight loss, easy bruising and bleeding tendencies.

Hepatic abscesses can develop among patients suffering chronic hepatitis most commonly caused by Entamoeba histolytica in developing countries and by gram-positive aerobic cocci, which cause pyogenic abscesses affecting those at the extremes of age), f.i. in case of neonates sepsis and catheterization of the umbilical vein may result in hepatic abscesses.

Hepatomas, being primary liver cancers, are the most common types of cancer originating fom the liver, There is a high prevalence of chronic infections with Hepatitis B virus, increasing the risk of hepatomas. Chronic infections with Hepatitis C virus also increase the risk of hepatomas. Finally, Hepatitis D can cause hepatocellular carcinoma while Hepatitis E can induce carcinogenesis. Alcohol and certain fungal toxins f.i. aflatoxin increase the risk of getting hepatoma. A preexisting alcoholic cirrhosis infected by either Hepatitis D virus or Hepatitis E can lead to hepatoma.

Diagnosis: by lab studies of liver enzyme panel, total bilirubin may be elevated in case of infectious hepatitis. Bilirubin levels higher than 30 mg/dl indicate a more severe disease. Alkaline phosphatase usually is in the reference range but may be elevated to no higher than twice the normal level. If alkaline phosphatase is elevated significantly this will consider an abscess or a biliary obstruction. The presence of HBsAg in the serum for 6 months or longer is indicative of chronic infection.

Ultrasound examination, computed tomography is of value by the differential diagnosis of a gallbladder disease, biliary obstruction or liver abscess.

Treatment: concerning chronic viral Hepatitis B infection usually symptomatic and supportive. (A long-term therapy with Adefovir Dipivoxil for HBeAg-negative chronic Hepatitis B cases brings only temporary benefits). In case of Hepatitis C infections by administering preparations of Interferon alfa in order to eliminate detectable viral RNA in the blood. A pyogenic liver abscess requires intravenous antibiotic therapy directed toward the most likely pathogens and a consultation for possible surgical or percutaneous drainage. Concerning Autoimmune hepatitis by administering Corticosteroids, iv. Immune serum globulin. This treatment can not kill the virus, the prognosis is bed.

RFR method: can measure the resonances of the viral and other microbiological components and eliminate these pathological agents.

The most frequently found resonant frequencies are as follows:

Hepatitis A virus: 285-295, 320-330, 340-356, 361, 366, 403, 420-436, 449, 487-488, 498, 570-590 kHz

Hepatitis B virus: 293, 340, 372, 384, 392-420, 444-450, 488 kHz

Hepatitis C virus: 324-339, 350-352, 370-374, 396, 400-402, 450-456, 475-482, 540-541, 559-563 kHz

Hepatitis D and E viruses: 348, 375, 386, 410, 432, 450, 468, 471, 490, 532, 535-548, 550-563, 580 kHz

Cytomegalovirus: 305, 349, 406-412, 512, 534, 548-550 kHz

Epstein-Barr Virus: 337-339, 342-347, 352, 372-382, 397-398, 422-424, 491, 516-518, 528, 560 kHz
The general frequencies of the Herpes Simplex Virus are: 290-294, 344-346, 352-365, 413, 425 kHz the other frequencies, see Chapter 5.2.4.1.
Yellow fever virus: 303, 374-379, 398-400, 420-422, 471-473, 510-516 kHz
Adenoviruses: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz
Toxoplasma species: 390-400, 436-444 kHz
Leptospira species: 312-316, 339-341, 390-401 kHz
Q-fever: 310, 323, 347-353, 364-371, 384, 410-412, 422, 441, 460-461, 476-482, 528-534, 543, 562 kHz
Rocky mountain spotted fever: 314, 390, 403, 441, 478-482 kHz
Entamoeba histolytica: 300, 314, 322, 336, 381-387, 398-402 kHz
In case of a chronic or/and autoimmune hepatitis the most frequent pathogens are Mycoplasma fermentans (442-451, 491-495 kHz) and HTLV (370-376 kHz)

14.1.3. Autoimmune Hepatitis

In case of an autoimmune hepatitis the patient's immune system attacks its own liver cells. Chronic autoimmune hepatitis is characterized by continuing hepatic necrosis caused by an active autoimmune inflammation, conditioned and controlled by genetic factors. This genetic susceptibility is associated with the complement allele C4AQO and with the HLA haplotypes B8, B14, DR3, DR4, and Dw3. These C4A gene deletions are associated with the development of autoimmune hepatitis concerning younger patients. HLA DR3-positive young patients are likely to have more aggressive diseases less responsive to medical therapy. HLA DR4-positive patients often develop extrahepatic manifestations of their disease.
Autoimmune hepatitis is classified as either type I or II.
Type I is the most common form in North America and Europe. It occurs at any age and is more common among women than men. About half of those with type I have other autoimmune disorders, too, such as type 1 diabetes, proliferative glomerulonephritis, thyroiditis, Graves' disease, Sjögren's Syndrome, autoimmune anemia and ulcerative colitis, owing usually in case of these illnesses the same pathogens i.e. mycoplasma, HTLV, etc.
Type II autoimmune hepatitis is less common, typically affecting girls from ages 2 to 14, though adults can suffer it too.
The iniciating agent of this illness can be an *acute or chronic viral* infection, alcohol abuse, exposure to hepatotoxic medications and chemicals. This condition can coexist with other liver diseases such as chronic *viral hepatitis*, may be triggered also by other, mostly viral infections, f.i. *Human T-cell Lymphotropic Virus*, *Cytomegalovirus*, *Epstein-Barr Virus*, *Mycoplasma fermentans*. The cell-mediated immunological attack occurs against the genetically predisposed hepatocytes expressing aberrant MHC II complexes on their surfaces. The in that way stimulated antigen-processing cells together with the autoantigen-sensitized cytotoxic T lymphocytes lead to the destroying of the liver cells. This autoimmune pathogenesis is characterized also by plasma cells and circulating autoantibodies including antinuclear antibodies, anti–liver-kidney microsomal antibodies, antibodies against soluble liver antigens, antibodies to liver-specific asialoglycoprotein, antimitochondrial antibodies, antiphospholipid antibodies, etc. Hypergammaglobulinemia is common, particularly concerning patients with extensive plasma cell infiltration of the liver.
The symptoms of this disease is similar to those of other chronic liver diseases but there accompany usually certain systemic manifestations, extrahepatic features and seroimmunological abnormalities, which may dominate the clinical presentation.

Depending on the dominant sign or manifestation there are various terms to be used to describe the disorder, such as autoimmune hepatitis, lupoid hepatitis, acute juvenile cirrhosis, plasma cell hepatitis, subacute hepatitis and chronic liver disease of young women. The use of corticosteroids, believed to be effective in a variety of immunological and autoimmune disorders, is often beneficial in the treatment of severe chronic immune hepatitis.

Symptoms: can include abdominal discomfort, nausea, vomiting, hepatomegaly, diarrhea, loss of weight, jaundice, dark urine, ascites, edema, fatigue, myalgia, arthalgia, mild pruritus, skin rashes, erythema, itching, hirsutism, amenorrhea and anorexia.

A non adequately treated chronic immune hepatitis often progress into cirrhosis.

Diagnosis: by the examination of autoantibodies, serum protein electroforesis, aminotransferase examination, other clinical laboratory tests, ultrasound and liver biopsy. (A routine blood test for liver enzymes can help reveal a pattern typical of hepatitis, but further tests, especially for autoantibodies, are needed to diagnose autoimmune hepatitis.) Antibodies are proteins usually produced by the immune system to fight off bacteria and viruses. In case of autoimmune hepatitis, the immune system produces antinuclear antibodies (ANA), antibodies against smooth muscle cells (SMA), or liver and kidney microsomes (anti-LKM). The pattern and level of these antibodies help define the type of autoimmune hepatitis (type I or type II).

Differential diagnosis: by distinguishing it from chronic immune hepatitis, chronic persistent hepatitis and Laennec's cirrhosis.

Treatment: by administering corticosteroids and immunosuppressive drugs.

RFR method: detects and may eliminate the pathogens.

The most frequently present pathogen microorganisms are:

Hepatitis A virus: 285-295, 320-330, 340-356, 361, 366, 403, 420-436, 449, 487-488, 498, 570-590 kHz

Hepatitis B virus: 293, 340, 372, 384, 392-420, 444-450, 488 kHz

Epstein-Barr Virus: 280, 337-339, 342-347, 352, 372-382, 397-398, 422-424, 491, 516-518, 528, 560 kHz

Cytomegalovirus: 305, 349, 406-412, 512, 534, 548-550 kHz

Coxsackie viruses A9, B3-4: 334, 360-364, 430, 444, 552-554 kHz

ECHO viruses 2, 3, 6-9, 11-14, 18-19, 22-24: 308-321, 369, 391, 403, 472, 526 kHz

Human T-cell Lymphotropic Viruses 1, 2, 3, 6: 370-376, 382 kHz

Mycoplasma fermentans: 442-444, 447-451, 493-495 kHz

The resonant frequencies concerning autoimmune hepatitis are various, the other frequencies are still to be found. The efficacy of RFR method is optimal solely together with conventional medical treatment. The RFR elimination of these pathogens may cause a temporary acute, strong inflammative process.

14.2. Cirrhosis in General

Cirrhosis is a general term including all of the end stage forms of chronic diffuse liver diseases in which a significant loss of liver cells, collapse and fibrosis of the supporting reticulin network, distortion of the vascular bed and nodular regeneration of the remaining liver cell masses are present. The damages of the hepatic circulation develop secondarily. Chirrhosis causes symptoms of the brain and death. Chronic viral hepatitis, autoimmune hepatitis and other chronic progressive liver diseases can all lead to cirrhosis.

14.3. Biliary Cirrhosis

Patients suffering from biliary cirrhosis show clinical and chemical signs of a chronic impairment of the bile excretion and give morphologic evidence of a progressive liver destruction centering around the intrahepatic bile ducts. Most forms of the biliary cirrhosis

result from the chronic inflammatory lesions of the periportal liver-cells, liver-ductules and interlobular ducts. This biliary chirrosis represents the late and often nonspecific phase of a genuin cirrhosis with calcinosis. Biliary cirrhosis can be classified either as primary, where the process relates to chronic intrahepatic cholestasis, or as secondary, where the disease is caused by the obstruction of the common bile duct or its large branches. The cause of **primary biliary cirrhosis (PBC)** is multifactorial and associated with a number of specific and nonspecific immunological features: such as elevated serum levels of IgM; circulating antimitochondrial antibodies(AMAs), periductal lymphocytes in the liver producing IgM immunoglobulins. All these raise the possibility of a self-perpetuating immune process and the derangement of the delayed immune responses leading to an autoimmune disease, but their mechanisms involved are yet unclear, the role and the identity of *viral causative agents* are likewise unknown.
The earliest recognizable lesion of primary biliary cirrhosis might be termed a chronic nonsuppurative necrotizing cholangitis, which inflammatory process is centered around the portal triads.
Major clinical **Symptoms** of the impaired bile excretion in the early stage are itching; fatigue and sicca syndrome, the late stage is characterized by progressive and prolonged jaundice; steatorrhea; hepatomegaly, digestive problems, urinary tract infections, hyperpigmentation ascites, swollen feet and the development of cutaneous xanthelasmas and xanthomas i.e. lipid deposits in the skin. Marked elevations of serum alkaline phosphatase, cholesterol and other lipid fractions can be found by laboratory examination, which all lead to a slow but progressive decline in health. The later complications of the disease can be enlarged veins in the stomach and esophagus, portal hypertension, vitamine deficiencies, osteoporosis, the development of cirrhosis and even of liver cancer. Autoimmune diseases often occur together with this illness.
Diagnosis: symptomatically, by detecting AMAs in the patient's serum and by laboratory evidences of a protracted obstruction to bile flow. By cholangiography and liver biopsy.
Treatment: there is no specific therapy. By diet, by administering ursodeoxycholic acid, (even together with methotrexate) symptomatically, administering cholestyramine, vitamins K, linol- linoleic acid, opioid-antagonists, by treating and preventing other complications, liver transplantation, etc.
RFR method: detects the hepatic viruses (see Chapter 14.1.) and eliminates them.
Other, frequently found resonant frequencies are: 319-320, 346-370, 390, 395-400, 442-451, 526, 580-585 kHz

14.4. Budd-Chiari Syndrome

Budd-Chiari syndrome (BCS) is caused by the occlusion of the hepatic vein carriing blood from the liver or of the inferior vena cava. The classical triad of abdominal pain, ascites and hepatomegaly are characteristic. The syndrome can be fulminant, acute, chronic or even asymptomatic.
Four main clinical variants are known: i.e. acute liver disease, subacute liver disease, fulminant liver disease and liver failure. The subacute liver disease is complicated by portal hypertension and by different degrees of liver decompensation.
The acute and subacute forms are characterized by the rapid development of abdominal pain, ascites, hepatomegaly, jaundice and renal failure.
The chronic form is the most common form with progressive ascites. Jaundice is absent but approximately 50% of patients have also a renal impairment.
The fulminant or subfulminant hepatic failure form is is characterized by ascites, a tender hepatomegaly, jaundice, renal failure and an early developing encephalopathy.
The Budd-Chiari syndrome is multicausal, can be caused by

Chronic infections: f.i. *aspergillosis*, *amebic* abscess, *syphilis*, *hepatitis*, *herpex simplex*, *nanobacterium*, *brucellosis*, *schistosomiasis*, *clonorchiasis*, *toxoplasma* infection, *neonatal cytomegalovirus infection*, *tuberculosis*, etc.
Chronic inflammatory diseases: f.i. Behçet disease, inflammatory bowel diseases, systemic lupus erythematosus, Sjögren's Syndrome, other connective-tissue diseases and autoimmune diseases. These diseases can also be of infectious origin, see the special Chapters!
Hematologic disorders: f.i. polycythemia rubra vera, paroxysmal nocturnal hemoglobinuria, unspecified myeloproliferative disorders, antiphospholipid antibody syndrome and essential thrombocytosis. These diseases may also be caused by infections.
Inherited thrombotic diathesis: such as protein C deficiency, protein S deficiency, antithrombin III deficiency, factor V Leiden deficiency associated with pregnancy and postpartum.
Inherited diseases may predispose to BCS. The most common cause of BCS is the primary (75%) thrombosis of the hepatic vena, completely or partially blocking the large veins draining the liver. The symptoms may appear rapidly, leading to death within weeks due to acute liver failures, or can be indolent, lasting for several years.
Diagnosis: symptomatically, by measuring liver enzyme levels and other organ markers (creatinine, urea, electrolytes, LDH). By using ultrasound techniques, abdominal and retrograde angiography. By CT and MRI.
Treatment: by administering anticoagulants, fibrinolytic agents, tissue plasminogen activators, streptokinase; urokinase, activase, etc.
RFR method: can support the medical treatment. Detects and eliminates the pathogen microorganisms, but should only be used after the beginning of the medical treatment.
The thrombosis can occur due to polycythemia rubra vera, or hypernephroma invading the inferior vena cava.
RFR method concerning Polycythemia rubra vera, Paroxysmal nocturnal hemoglobinuria, unspecified myeloproliferative disorders, Antiphospholipid antibody syndrome, see their special Chapters.
RFR method concerning Behçet disease, inflammatory bowel diseases, systemic lupus erythematosus, Sjögren's Syndrome, connective-tissue diseases and other autoimmune diseases, see their special Chapters.
RFR method concerning *Aspergillosis*, *amebic* abscess, *syphilis*, *hepatitis*, *herpes simplex*, *nanobacterium*, *brucellosis*, *schistosomiasis*, *clonorchiasis*, *toxoplasma* infection, neonatal *cytomegalovirus* and *tuberculosis*, see their special Chapters.

14.5. Functional Liver Disorders Associated with Jaundice

Gilbert-Meulengracht syndrome, Crigler-Najjar syndrome and Dubin Johnson syndrome belong to the functional liver disorders associated with jaundice. Hereditary hyperbilirubinemia forms can be divided into conjugated and unconjugated ones. Gilbert-Meulengracht syndrome and Crigler-Najjar syndrome are examples of the unconjugated hyperbilirubinemia form, while Dubin-Johnson syndrome and Rotor syndrome represent 2 familial conjugated hyperbilirubinemia forms.

14.5.1. Gilbert-Meulengracht Syndrome

The Gilbert-Meulengracht syndrome is the most common hereditary form of liver disorders with an increased bilirubin level in the serum. This autosomal recessive condition is characterized by intermittent jaundice in the absence of hemolysis or of an underlying liver disease. Its main symptom is an otherwise harmless jaundice, which does not require any treatment and is caused by elevated levels of unconjugated bilirubin in the bloodstream

(hyperbilirubinemia). Conjugation renders the bilirubin molecules to be water-soluble, and the conjugated bilirubin can then be excreted in the bile into the duodenum.Hyperbilirubinemia in this syndrome is the result of the reduced activity of glucuronyltransferase, an enzyme conjugating bilirubin and some other lipophilic molecules and located primarily in the endoplasmic reticulum of hepatocytes. The progress of the syndrome can be precipitated by dehydration, fasting, menstrual periods, by stress and by intercurrent illnesses caused by silent viruses. Patients may feel vague abdominal discomfort and general fatigue, though this condition is otherwise usually asymptomatic. Affected adult people can develop brain damages caused by jaundice (cornicterus).
Genetical predisposition: the gene expressing bilirubin-UGT has a complex structure and is located on chromosome 2. The basic factor of the etiology of Gilbert-Meulengracht syndrome is the abnormality of this gene.
The other important factor for its manifestation is **a combined infection** caused f.i. by *Hepatitis viruses, EBV, CMV, HTLV* and *Mycoplasma*. These infections can be clinically asymptomatic. (Viral hepatitis can be excluded by blood samples which are negative concerning the antigens specific to the different hepatitis viruses).
Prevention: genetic counseling is recommended for future parents with a family history of Gilbert-Meulengracht syndrome. Blood testing can identify people who carry the gene.
Diagnosis: by excluding hemolysis, by measuring bilirubin and bilirubin-UGT levels, haptoglobin, lactate dehydrogenase levels, by the absence of reticulocytosis; by gene abnormality examinations; by viral antibody examinations.
Differential diagnosis: by distinguishing it from the Crigler-Najjar syndrome, the Dubin-Johnson syndrome and the Rotor syndrome.
Treatment: symptomatically, by phototherapy and liver transplantation.
RFR method: can detect the viral infections triggering the disorder and can eliminate them.
The most frequent resonances are: 293, 324, 328, 336, 341, 354, 356, 384, 392, 398, 408-410, 414-420, 422, 444-450, 454-456, 475-479, 488, 561 kHz

14.5.2. Crigler-Najjar Syndrome

Crigler-Najjar syndrome is a rare autosomal recessive disorder of bilirubin metabolism. This disorder is an inherited form of non-hemolytic jaundice, leading often to brain damages of affected infants. The syndrome has two types.
In case of **Crigler-Najjar syndrome type 1** an intense jaundice appears on the first days of life of the patient and persists permanently. Most patients belonging to type 1A show a mutation in one of the common exons (2 to 5), and have difficulties in conjugating several additional substrates (certain drugs and xenobiotics). A smaller percentage of patients belonging to type 1B have mutations limited to the bilirubin-specific A1 exon; in case of which the conjugation defect is mostly restricted to bilirubin. This Crigler-Najjar syndrome is associated with an almost complete absence of the enzyme, resulting thus in very high levels of unconjugated bilirubin at birth.
In case of **Crigler-Najjar syndrome type 2**: (named also Arias syndrome) UGT1A1 is present at reduced but detectable levels caused by single base pair mutations.
Lower levels of serum bilirubin and markedly depressed activity of hepatic UGT are characteristic of this type 2. A treatment with phenobarbital can induce the expression of UGT in case of patients suffering from this syndrome type 2, decreasing thus the serum bilirubin level. Bilirubin encephalopathy can but rarely occur in this case, usually only then, if the patients suffer from a superimposed infection or stress, too.
Both types of the Crigler-Najjar syndrome are autosomal recessive inherited disorders. Alterations in the coding sequence of UGT1 gene result in no UGT activity or in a reduced UGT activity together with a marked impairment of the bilirubin conjugation.

Symptoms: confusion, disturbancies in thinking; jaundice and yellow coloured sclera (icterus) beginning a few days after birth and getting worse over time.
Diagnosis: by liver function tests concerning conjugated bilirubin, total bilirubin, unconjugated (unbound) bilirubin in the blood and liver biopsy with enzyme assay.
Treatment: by regularly repeated phototherapy, by liver transplantation, by blood transfusions in order to control the amount of bilirubin in the serum. By administering phenobarbital in case of Arias syndrome (type 2).
RFR method can detect and eliminate all present pathogens.
The most frequent resonances are: 324, 336, 370-372, 384, 392, 398, 414-420, 442-451, 454-456, 475-479, 488, 541, 561 kHz

14.5.3. Dubin-Johnson Syndrome

Dubin-Johnson syndrome is an autosomal recessive disorder causing increased conjugated bilirubin levels of the serum without elevating the liver enzymes ALT and AST. This condition is also associated with a certain defect of the hepatocytes concerning their ability to secrete conjugated bilirubin into the bile. This syndrome is caused by the mutation in the gene responsible for the human canalicular multispecific organic anion transporter (cMOAT) protein, which is also named multidrug resistance protein 2 (MRP2). This protein mediates an adenosine triphosphate (ATP)-dependent transport of certain organic anions across the canalicular membrane of the hepatocytes. The Dubin-Johnson syndrome has been described in all countries and ethnic groups. Its prevalence is highest among Iranian Jews.
Genetical predisposition: The human MRP2 gene is localized to band 10q23-10q24. Defects in the cMOAT (MRP2) protein result in impaired hepatobiliary transport of non–bile salt organic anions and are thought to be responsible for the conjugated hyperbilirubinemia and for the accumulation of the hepatocellular pigment. Several different mutations in the MRP2 gene are identified in patients with Dubin-Johnson syndrome. MRP2 plays an important role in the detoxification of many drugs by transporting a wide range of compounds, especially the conjugates of glutathione, glucuronate and sulfate, collectively known as phase II products of biotransformation. Besides hepatocytes, MRP2 can be found located in renal proximal tubular cells, enterocytes and syncytiotrophoblasts of the placenta, too. Energy derived from ATP is important for the secretory function of MRP2.
Patients with Dubin-Johnson syndrome tend to develop nonpruritic jaundice in their teenager years. Associated findings are the presence of a hepatitis B virus (HBV)-related chronic hepatitis, the history of tubercular lymphadenitis, chronic cholecystitis, coronary heart disease and its exacerbation during pregnancy.
Certain infective agents, f.i. *HBV, HTLV, CMV* and perhaps some *other gastroenteral viruses* can play a role in this syndrome.
Prognosis: Most patients are asymptomatic and have normal life spans. Some neonates might suffer from cholestasis. Hormonal contraceptives and pregnancy may lead to overt jaundice and icterus.
Diagnosis: by family history, by urine porphyrin analysis.
RFR method can detect HBV, HCV, HTLV, CMV, EBV and eliminate them.
The most frequent resonances of this syndrome are: 293, 324, 336, 370-372, 384, 392, 398, 414-420, 442-451, 454-456, 475-479, 488, 541, 561 kHz

14.6. Gallbladder Disorders

Gallbladder disorders are one of the most common gastrointestinal disorders, their spectrum ranging from asymptomatic cholelithiasis to gallbladder (or biliary) colic,

cholecystitis, choledocholithiasis and cholangitis. Their further complications can be gallstone pancreatitis, gallstone ileus, biliary cirrhosis and gallbladder cancer.
The occlusion of the bile ducts by sludge and stones leads to complications. Cholecystitis is often caused by cholelithiasis meaning the presence of choleliths (gallstones) in the gallbladder. Choleliths can block the cystic duct directly, leading to the thickening and the stasis of the bile, and to secondary infections caused by microorganisms, predominantly *E. coli and Bacteroides species* present in the gut.

14.6.1. Gallstones

Gallstones are collections of crystals developing in the gallbladder and the bile ducts.
In case bile gets concentrated in the gallbladder, it can become supersaturated with its substances, which then precipitate from the solution as microscopic crystals. The crystals are trapped in gallbladder mucus, producing gallbladder sludge.
The bile acids, lecithin and phospholipids help to maintain the cholesterol solubility in the bile. When the ratio of the cholesterol to bile acids or phospholipids is increased, the bile becomes supersaturated with cholesterol; crystallizes and forms a nidus for stone formation. Calcium and pigment as well may be incorporated in the stone. People with impaired gallbladder motility, biliary stasis and bile content are predisposed to develop gallstones. The 2 main substances involved in the gallstone formation are cholesterol and calcium bilirubinate.
Later on the crystals grow, aggregate and fuse forming macroscopic stones.
Cholesterol gallstones: liver cells secrete cholesterol into the bile along with phospholipid (lecithin) in form of small spherical membranous bubbles, termed unilamellar vesicles. Liver cells do also secrete bile salts, which are powerful detergents required for the digestion and absorption of dietary fats. Bile salts in bile dissolve the unilamellar vesicles to form soluble aggregates called mixed micelles. If the bile is supersaturated with cholesterol, cholesterol monohydrate crystals may be formed. Increased levels of estrogen as a result of pregnancy, hormone therapy, or the use of combined (estrogen-containing) forms of hormonal contraception may increase the cholesterol level in the bile and may decrease the movement of the gallbladder, resulting in gallstone formation.
If the gallstones are in the gallbladder, the condition is named cholelithiasis; if in the bile ducts, it is called choledocholithiasis. Gallstones may be asymptomatic for decades.
Calcium, bilirubin and pigment containing gallstones: Bilirubin, a yellow pigment derived from the breakdown of heme, is actively secreted by liver cells into the bile . Bilirubin in the bile is usually in form of glucuronide conjugates, which are quite water soluble and stable, though a small proportion remains in unconjugated form. Similarly to fatty acids, phosphate, carbonate and other anions, unconjugated bilirubin tends to form insoluble precipitates with calcium. Calcium enters the bile passively together with other electrolytes. In case of a high heme turnover, f.i. in case of chronic hemolysis or cirrhosis, unconjugated bilirubin will be present in the bile at higher concentrations. Calcium bilirubinate will crystallize and, eventually, form stones. Over time, the bilirubin precipitates will get a black color due to oxydations, the stones formed are termed black pigment stones.
The bile is normally sterile, but in certain unusual circumstances it may become colonized by bacteria, mostly by *E. coli, Klebsiella, Enterobacter, Salmonella, Pseudomonas and Bacteroides species*, which hydrolyze conjugated bilirubin resulting the increase of unconjugated bilirubin. This can lead to the precipitation of calcium bilirubinate crystals and grow with help of *Nanobacteria.* The bacterial hydrolysis of lecithin leads to the release of fatty acids, which make complex with calcium and precipitate. Calcium apatit crystals develop surrounding the Nanobacteria.
Mixed gallstones: Cholesterol gallstones can get colonized with bacteria and cause the inflammation of the gallbladder mucosa. The lytic enzymes of the bacteria and of the

leukocytes can hydrolyze the bilirubin conjugates and fatty acids, thus the cholesterol stones may be combined with calcium bilirubinate and other calcium salts producing mixed gallstones.

Pigment gallstones: People with erythropoetic protoporphyria are at increased risk to develop gallstones. Disorders causing hemolytic anemia (f.i. sickle cell anemia, hereditary spherocytosis, beta-thalassasemia, etc.) can cause pigment gallstones. Black pigment gallstones develop in case of high heme turnover, though, in most cases no risk factor can be found.

The risk factors for gallstone formation are old age, obesity, womanhood and the inflammation of the bowel system. Some studies suggest that genetical predispotition plays also a significant role in the development of gallstones. The prevalence rates of cholelithiasis are highest among western Caucasian, Hispanic and Native American populations. Eastern European, African American and Asian populations are less afflicted.

The migration of gallstones may lead to the occlusion of the biliary and pancreatic ducts, causing pain and acute complications, such as acute cholecystitis, ascending cholangitis or acute pancreatitis.

Symptoms: Gallstones are initially asymptomatic, and are called "silent stones". They usually start to cause symptoms once they reach a certain size (5-10 mm).

Its main symptom is commonly referred to as a gallstone "attack", also known as biliary colic, in which case patients experience an intense pain in the upper abdominal region steadily increasing for about thirty minutes to several hours. Pain in the back, mostly between the shoulder blades, or under the right shoulder is often present. Pain in the lower region of the abdomen is less common. Nausea and vomiting can occur. Attacks occur mostly after a fatty meal and at night. Abdominal bloating, belching, gas, indigestion and intolerance of fatty foods can often be experienced. Chills, lowgrade fever, yellowing of the skin or eyes, and/or clay-colored stool are signs of complications.

The most frequent resonancies of the Gallstones: 323-327, 331-335, 355-357, 374-381, 392-393, 397-401, 414-421, 560-568 kHz

14.6.2. Acute cholecystitis

Acute cholecystitis is the inflammation of the gallbladder wall. In case of an acute cholecystitis there is often no previous sign of a gallbladder disease when the person experiences a sudden, excruciating pain in the upper abdomen, which instead of resolving spontaneously, persists and worsens. Acute cholecystitis is associated with a gallstone impacted in the cystic duct in 90 percent of cases. The in this way occuring sudden distention of the gallbladder damages its blood supply and its lymphatic drainage, and the normally present commensal bacteria will proliferate.

Less often this inflammation is associated with bacterial bowel infections, commonly occuring among people with inflammatory bowel diseases, especially with irritable bowel syndrome and Crohn's disease.

The acute inflammation may sometimes subside with conservative measures, such as hydration and antibiotics.

Complications: The complications of an acute cholecystitis can be very severe and can lead to *gangrene*, *pericholecystic abscess* and to the *perforation* of the gallbladder. In this case an immediate surgery is necessary to remove the severily inflamed gallbladder. A bile duct inflammation may spread to the liver.

14.6.3. Primary cholangitis

Primary cholangitis is the inflammation and obstruction of the bile ducts inside and outside the liver. The disease affects mostly young men and develops due to abnormal immune responses occuring in case of inflammatory bowel diseases, especially of ulcerative colitis.

14.6.4. Chronic cholecystitis

Chronic cholecystitis is a long standing inflammation of the gallbladder characterized by repeated attacks of severe, sharp abdominal pain. A damaged gallbladder musculature can but slowly contract, the gallbladder wall is thick, consisting mostly of fibrous materials. The lining of the gallbladder may get ulcerated and scarred, the gallbladder contains sludge or gallstones which often obstruct the cystic duct. This condition is probably caused by the damage and then by repeated episodes of acute inflammations, caused often by gallstones. Protective colloids, inhibiting the crystal separation from the bile, play an important role in the hindering of the development of gallstones. Bacterial infections and inflammations inhibit the production of these protective colloids. The administering of cholesterol-lowering drugs should be reserved for cases in which the natural excretion cannot be regained. *E. coli* and other intestinal bacteria, doing no harm if present only in moderate numbers, can overgrow in a few hours and cause bloating, gas and pain. Only bacteria can produce gas. If the right side abdominal pain is accompanied by bloating and gas, one has a digestive problem, and if this digestive problem arises from a congested liver, the pain is felt directly under it or over it, the feces is light coloured and the cholesterol levels are high. Not every patient has all the symptoms.

Complications: Occasionally, a large stone may erode the wall of the gallbladder producing a *cholecystoenteric fistula* connecting the duodenum. The overgrowth of colonizing bacteria in the gallbladder often occurs. The accumulation of pus in the gallbladder is termed *gallbladder empyema*. If sufficiently large, a gallstone may gradually erode the gallbladder wall and enter the small or large intestine (usually the ileum), where it can cause an intestinal obstruction, termed *gallstone ileus*.

More typically, gallstones pass from the gallbladder into the bile ducts. They may pass through these ducts and into the small intestine without any harm, or may remain in the ducts without obstructing the flow of the bile or causing symptoms. If a gallstone partially or transiently obstructs a bile duct, pain will be experienced.

This pain typically rises slowly to a plateau and is falling then gradually. The pain may extend up to the right shoulder blade. *Bile duct inflammations* may cause loss of appetite, nausea, vomiting and fever. The blockage of the bile may produce symptoms of cholestasis. *Cholestasis* is the term of the reduction or stoppage of the bile flow. The bilirubin will then be deposited in the skin, causing jaundice and then pass into the urine. The bile stasis can cause even a general toxicosis.

Not only gallstones and tumors can cause bile duct obstructions. An injury during gallbladder surgery may occasionally also cause obstruction, or an obstuction can be present if the duct passing through a chronically diseased pancreas get narrowed. Obstructions can rarely develop due to infections of parasites, such as of *Ascaris lumbricoides*, *Clonorchis sinensis, Sheep liver fluke, Pancreatic fluke* and *Human Liver fluke*.

A chronic stone disease, bacterial and viral (HPV) infections can lead to *gallbladder cancer*, which often invades the adjacent liver region and the common bile duct, causing jaundice. The prognosis is poor, unless the cancer is localized to the gallbladder, in which case cholecystectomy may be curative.

Prevetion: RFR method can detect and eliminate bacteria, fungi and viruses

Diagnosis: by x-ray, ultrasound, CT, MRI, PETscan, hepatobiliary scintigraphy, endoscopic retrograde cholangiopancreatography, bacteriological culturing, feces examination for worms, etc.

Treatment: usually by administering antibiotics, ursodeoxycholic acid, antihelmintics and by surgery (ERS, ERCP, cholecystectomy etc).

RFR method: is not able to diagnose these diseases. After administering antibiotics or antihelmintics one can try to detect and eliminate the pathological microorganisms.

The most frequent resonant frequencies of acute and chronic cholecystitis are: 296, 317, 320-330, 345-350, 352-356, 380-394, 396, 399, 403-409, 430, 442, 474, 581, 592 kHz

Nota bene: The RFR way of treating an acute cholecystitis is very dangerous.

The most frequent resonant frequencies of Escherichia coli are: 280-285, 288-290, 317, 322-328, 337, 352-358, 390-397, 408, 410-412, 422, 426, 435-443, 478, 489 kHz

The most frequent resonant frequencies of Pseudomonas are: 324-325, 330-335, 339, 364-367, 372-374, 377-380, 388-397, 401, 414, 428, 492 kHz

The most frequent resonant frequencies of the Salmonella group are: 329-339, 354, 360-370, 380-386, 390-395, 428, 452, 497, 558 kHz

The most frequent resonant frequencies of the Shigella group are: 310, 313, 315-321, 369, 388-398, 403-410, 423-425, 496, 499, 506 kHz

The most frequent resonant frequencies of the Enterobacter group are: 351, 373-375, 418 kHz

The most frequent resonant frequencies of the Proteus group are: 320-329, 333-339, 345-352, 408-416, 426, 516, 522-529, 535 kHz

The most frequent resonances in case of gallstones are: 323-327, 331-334, 346, 355-357, 361, 372-374, 380, 392-393, 397-402, 409-421, 442-461, 474 kHz

The most frequent resonances in case of calcium gallstones are: 264, 280-284, 296-298, 317-318, 324-325, 332-334, 375-381, 430-435, 486, 554 kHz

14.7. Parasitic Infections of the Liver

The obstruction of the bile way system is often caused by parasitic infections caused by f.i. *Ascaris lumbricoides, Clonorchis sinensis, Sheep fluke, Pancreatic fluke, Fasciolopsis buskii and Fasciola hepatica.* These worms live in the intestines or in the bowels. Flukes, flatworms and roundworms have a complex life cycle with many stages such as miracidia, redia, cercaria and metacercaria. Though sheep, cattle, pigs, cats, dogs and human beings can be natural hosts to the adult parasites, their other stages come to pass outdoors and in secondary hosts. Some worms live in pets (dogs, cats, horses) while others live in wild animals and domestic ones. Eggs and larvae of these worms can infect the human organism. These worms often spread through the bile way into the liver causing there occasional obstructions.

Diagnosis: by examination of parasites in the stool.

Treatment: by administering antihelminthic drugs such as Mebendazole, Thiabendazole, Prasiquantel, Niclosamide, Yomesan, etc.

RFR method: should only be done after repeated antihelminthic treatments. Don't make diagnosis with RFR examination, since not only one, but more pathogens can resonate on one certain radiofrequency!

As regards the resonant frequencies of the worms, see Chapter 9.

14.8. Pancreatitis

Pancreatitis is the inflammation of the pancreas. The pancreas is a large gland behind the stomach and close to the duodenum and secretes digestive enzymes into the small intestine through a tube named the pancreatic duct. These enzymes help to digest fats, proteins and carbohydrate components of food. The pancreas releases also insulin and glucagon into the bloodstream. These hormones help the body to utilize the glucose taken from food for getting energy.

Acute pancreatitis is an inflammatory process in which the pancreatic enzymes may autodigest the pancreas gland itself. This inflammation can easily be spread, as the pancreas is located in the retroperitoneal space having no capsule. Parenchymal edema and peripancreatic fat necrosis are the first alterations caused by acute pancreatitis. This

process is also known as acute edematous pancreatitis. Acute pancreatitis occurs suddenly, lasts for a short period of time and then usually recuperates. If the necrosis involves the parenchyma, and is accompanied by hemorrhages and dysfunctions of the gland, the inflammation evolves into hemorrhagic or necrotizing pancreatitis. There can pseudocysts and pancreatic abscesses be developed in case of necrotizing pancreatitis, as the enzymes get walled off by granulation tissues or by a bacterial infection of the pancreatic and peripancreatic tissue.

Due to the presence of bradykinins and phospholipase A the inflammatory process has systemic effects as well. These mediators may lead to vasodilatation, to the increase in vascular permeability, to pain, and to leukocyte accumulations in the vessel walls. Fat necrosis can cause hypocalcemia. Pancreatic B-cell injury may lead to hyperglycemia.

Patients frequently own a history of previous biliary colic and biliary stone disease, as well as of a **long-lasting alcohol consumption**, these being the major cause of acute pancreatitis. Ethanol leads to intracellular accumulations of he digestive enzymes and to their premature activation and release. Ethanol increases the permeability of the ducts, allowing the enzymes to get into the parenchyma, which results in pancreatic tissue damages. Ethanol increases the protein content of the pancreatic juice and decreases the bicarbonate levels and the concentration of the trypsin inhibitors, leading to the formation of protein plugs which block the pancreatic outflow causing obstructions. The other common cause of acute pancreatitis is the **biliary stone disease** (f.i. cholelithiasis, choledocholithiasis). A biliary stone may get lodged in the pancreatic duct or the ampulla of Vater obstructing the pancreatic duct, leading to the extravasation of pancreatic enzymes into the parenchyma. The biliary stone disease is caused by *nanobacteria* (see biomineralization) which produces stones. If the stone blocks the pancreatic duct, an acute pancreatitis will develop.

Less common causes of acute pancreatitis are *viral infections*, including Hepatitis viruses, mumps, Coxsackie virus, Cytomegalovirus (CMV), Epstein-Barr Virus (EBV), HTLV and rubella and/or *bacterial infections*, f.i. Mycoplasma, Enterobacteriaceae and coccal infections. *Intestinal parasites*, f.i. *Ascaris lumbricoides* blocking the pancreatic outflow can cause obstructive pancreatitis. *Medicaments*, f.i. azathioprine, corticosteroids, sulfonamides, thiazides, furosemide, NSAIDs, mercaptopurine, methyldopa and tetracyclines can also be causative agents of this illness. If a *Carcinoma of the pancreas*, leads to a pancreatic outflow obstruction an acute pancreatitis can come about.

Symptoms: The main presentation of acute pancreatitis is epigastric pain and right upper quadrant pain radiating to the back, nausea and/or vomiting and fever. Acute respiratory distress syndrome (ARDS), acute renal failure, cardiac depression, hemorrhage and hypotensive shock may all be systemic complications of a most severe acute pancreatitis.

Though acute pancreatitis must surely be noted, chronic pancreatitis has a more severe symptomatology as the episodes recur. Chronic pancreatitis does not heal by itself it rather results in the slow destruction of the pancreas.

Chronic pancreatitis, a continuous chronic inflammatory process of the pancreas, is characterized by irreversible morphological changes. This chronic inflammation can lead to chronic abdominal pains and/or to the impairment of endocrine and exocrine functions of the pancreas. Chronic pancreatitis can result in an atrophic fibrotic gland with dilated ducts and calcifications. However, the findings got by conventional diagnostic studies may prove to be normal in the early stages of a chronic pancreatitis, the inflammatory changes can only be seen by histologic examinations.

Chronic pancreatitis differs from the acute pancreatitis in many a way. In case of an acutely inflamed pancreas (neutrophils and edema) the serum levels of the pancreatic enzymes (amylase and lipase) are significantly elevated. A full recovery can be observed by most patients, suffering from acute pancreatitis, whereas in case of a chronic pancreatitis, the process is chronic, characterized by an irreversible inflammation

(monocytes and lymphocytes) leading to fibrosis with calcification. The patient suffering from a chronic pancreatitis has chronic abdominal pain, normal or mildly elevated pancreatic enzyme levels; and if the pancreas loses its endocrine and exocrine functions, the patient will suffer from diabetes mellitus and steatorrhea.
The symptoms of a chronic pancreatitis may include tachycardia, tachypnea, hypotension, fever, abdominal tenderness, distension, guarding, mild jaundice, diminished or absent bowel sounds.
Hereditary pancreatitis is an autosomal dominant disorder with 80% penetrance, which accounts for about 1% of the cases. Regarding families with hereditary pancreatitis researches have led to the identification of several mutations in the cationic trypsinogen gene on chromosome 7.
Cystic fibrosis, an autosomal recessive disorder is one of the most common genetic abnormalities, accounting for a small percent of patients suffering from chronic pancreatitis. The cystic fibrosis transmembrane regulator (*CFTR*) gene, which transcribes a protein regulating chloride transport across the cellular membranes, can have several hundred mutations in case of this disease. The clinical manifestation of a given mutation depends on the degree of the potency of this protein to regulate chloride transport. Clinical manifestations range from severe chronic pancreatitis associated with classic pulmonary disease to chronic pancreatitis associated with relatively normal respiratory functions.
Hyperlipidemia (usually type I and type V) may also cause chronic pancreatitis; however, it usually causes repeated attacks of acute pancreatitis.
Autoimmune pancreatitis is uncommon and accounts probably for less than 1% of cases of chronic pancreatitis. Its clinical characteristics include symptomatic or asymptomatic diffuse enlargement of the pancreas, diffuse and irregular narrowing of the main pancreatic duct, increased levels of circulating gamma globulin (elevated level of IgG4), the presence of autoantibodies and a possible association with other autoimmune diseases. Fibrosis with lymphocytic infiltration can be experienced by pathologic examinations. The secondary forms of autoimmune chronic pancreatitis are associated with primary biliary cirrhosis, primary sclerosing cholangitis and Sjögren's Syndrome.
Mycoplasma, HTLV, Epstein-Barr Virus and Cytomegalovirus can cause autoimmune pancreatitis among genetically predisposed patients.
Diagnosis: symptomatically, by laboratory examinations of BUN, creatine, electrolytes (Na, K, Cl, CO_2, P, Mg), amylase P levels, (which levels if more than 3 times higher than normal, strongly suggest the diagnosis of an acute pancreatitis.); lipase levels. The lipase levels of patients suffering from chronic pancreatitis (**usually caused by alcohol abuse**), may be elevated even in case of a normal serum amylase level. By liver function tests, utrasonography, CT.
Treatment: by parenteral nutrition if the prognosis is poor, by administering analgesics to relieve pain, f.i. meperidine. Antibiotics are used in severe cases associated with septic shock or if the CT scan indicates a phlegmon of the pancreas. Pancreatitis caused by biliary stones and associated with cholangitis also need antibiotic treatment. The preferred antibiotics are those secreted by the biliary system, f.i. ampicillin, third-generation cephalosporins, etc. In case of ascariasis antihelminthic therapy, surgery. In case of autoimmune pancreatitis by administering corticosteroids and immunsuppressive drugs. Surgical care, etc.
RFR method: detects and may eliminate the pathogens, which are usually viruses. Nota bene: Ascaris should not be influenced with RFR method!
The most frequent resonances of the Hepatitis A virus are: 285-295, 320-330, 340-356, 361, 366, 403, 420-436, 449, 487-488, 498, 570-590 kHz
The most frequent resonances of the Hepatitis B virus are: 293, 341, 384, 392, 398, 414-420, 444-450, 454, 488 kHz

The most frequent resonances of the Hepatitis C virus are: 324-339, 350-352, 370-374, 396, 400-402, 450-456, 475-482, 540-541, 559-563 kHz
The most frequent resonances of the Epstein-Barr Virus are: 372-383 kHz
The most frequent resonances of the Cytomegalovirus are: 408-410, 530-536 kHz
The most frequent resonances of the Mumps are: 299, 308, 318, 328, 344, 363-364, 372-384, 472-492, 498, 512 kHz
The most frequent resonances of the Coxsackie viruses are: 291-292, 300-304, 331-346, 360-368, 370-376, 388-396, 416, 426-434, 443-446, 471-472, 553-554 kHz
The most frequent resonances of the Rubella virus are: 372, 402, 440, 450-451, 468, 520-530 kHz
The most frequent resonances of the Human T-cell Lympotropic virus-1 are: 370-376 kHz
The most frequent resonances of the Mycoplasma are: 307-308, 321-324, 327-329, 342-351, 491-495 kHz
The most frequent resonances of the Nanobacterium are: 324-325, 375-381, 560-568 kHz
The resonant frequencies of other microorganisms found, see the special Chapters.

14.9. Chronic Congestive Splenomegaly

This disorder is characterized by portal hypertension, splenomegaly, pancytopenia and gastrointestinal bleedings. It is usually secondary to cirrhosis of the liver or, less commonly, to portal vein thrombosis. Portal hypertension can develop either due to intrahepatic or to extrahepatic pathologic conditions. Intrahepatic obstruction is seen in case of Laennec's cirrhosis and schistosomiasis. Extrahepatic obstruction can be caused by thrombosis of the portal and splenic vein as well as by compression of the splenic vein occuring due to a pancreatic tumor or fibrosis. The hematologic changes are those of hypersplenism and reflect destruction of blood cells in the spleen. Progressive fibrosis of the reticulum, the trabeculi and the capsule can be observed. The examination of bone marrow show myeloid or erythroid hyperplasia.
This splenomegaly may be expressed and accompanied by gastrointestinal complaints such as flatulence, indigestion, or pain.
The prognosis is determined largely by the underlying causes of the chronic congestive splenomegaly. Hepatic failures are indicators of a bad prognosis.
Diagnosis: requires detectable splenomegaly, changes consistent with portal hypertension and pancytopenia together with a normocytic, normochromic or iron deficiency anemia.
Differential diagnosis: by distinguishing it from intrahepatic or other extrahepatic obstructions, schistosomiasis or other pathogen infections.
Conditions, most often associated with splenomegaly may be:
Infections, such as bacterial endocarditis, brucellosis, tuberculosis, infectious mononucleosis, cytomegalovirus, syphilis, histoplasmosis, malaria, kala azar, schistosomyasis, gram negative sepsis.
Connective tissue diseases, such as rheumatoid arthritis, Felty's syndrome, lupus erythematosus.
Hematological disorders, such as lymphomas, histiocytosis, myeloproliferative syndromes, chronic lymphatic leukemia, acute leukemia, hemolytic anomalies, hereditary spherocytosis, autoimmune hemolytic anemia, polycythemia rubra vera.
Metabolic diseases: Gaucher's disease, Niemann-Pick's disease, Amyloidosis.
Treatment: symptomatically or/and surgery.
RFR method: detects and may eliminate the pathogen microorganisms.
As regards *Schistosomiasis* (see Chapter 9.3.1.), *Brucellosis* (see Chapter 6.2.2.), *Mononucleosis* (see Chapter 5.2.4.3.1.), *Cytomegalovirus* (see Chapter 5.2.4.4.), *Syphilis*

(see Chapter 6.19.2.), *Histoplasmosis* (see Chapter 7.1.4.), *Malaria* (see Chapter 8.3.), *Hepatitis* (see Chapter 5.1.7.3.3. and 5.2.5.).
Resonant frequency values frequently found are: 370-374, 442-451 kHz

14.10. Banti's Syndrome

Chronic congestive splenomegaly developed due to portal hypertension is the cause of the Banti's syndrome, which is characterized by gastrointestinal bleeding and pancytopenia. It occurs usually secondarily to the cirrhosis of the liver or, less commonly, to portal vein thrombosis. The spleen is not palpable as concerns normal persons beyond the age of twenty, so that patients with a palpable spleen but without any other signs or symptoms should be examined by spleen scan and by complete blood count. Splenomegaly is often accompanied by the syndrome of hypersplenism, consisting of a large spleen, anemia, leukopenia or thrombocytopenia and hyperactivity of the bone marrow. This syndrome can often be reversed by splenectomy. Hypersplenism can be present in most splenomegalic states. Its severity does not correlate with the degree of the enlargement of the spleen. The splenomegaly, however, does not always cause hypersplenism. The development of this disease is iniciated by a viral infection.
Diagnosis: according to the symptoms.
Differential diagnosis: by distinguishing it from chronic myelogenous or lymphatic leukemia and from chronic viral infections.
Treatment: according to the symptoms.
RFR method: can inhibit the development of this disease.
The most often found resonant frequencies in case of this disease are: 324-325, 336, 341, 375-381, 384, 392-400, 408-413, 416-420, 442-451, 454, 475-479, 488, 541, 561-568 kHz

14.11. Familial Paroxysmal Polyserositis (Mediterranian Fever)

The familial Paroxysmal polyserositis (FPS) is an illness caused by *Brucella* bacteria affecting only genetically predisposed persons. Brucellosis can be contracted by direct contact with the secretions and excretions of infected animals; by drinking unpasteurized milk of cows, sheep and goats, or by eating dairy products containing live *Brucella* bacteria. Transmission from person to person is rare. This disease is characterized by brief, recurrent episodes of peritonitis, pleuritis and arthritis, usually associated with fever.
The disease occurs in families, originating from around the Mediterranean See, Sephardi and Ashkenazi Jews, Armenian people, people from Lebanon, Turkey and the Arabian countries. The mutations of MEFV gene appear to cause the disease, which gene normally produces pyrin, an inhibitor of chemotactic factor (C5a) and perhaps that of interleukin 8, too. Patients with FPS lack this inhibitor, resulting in an uninhibited activity of the chemotactic factor, so that episodes of inflammation (with associated fever) in the peritoneum, pleura, joints and other loci will come about. These inflammatory episodes can lead to an excessive production of amyloid A protein coming from the acute phase reaction, which, if deposited in the kidneys, may cause nephritic syndrome; though amyloidosis develops solely among patients with specific MEFV haplotypes.
After the initial phase, the **symptoms** can be severe constipation, loss of appetite, loss of weight, abdominal pain, joint pain, fever, headache, backache, weakness, irritability, insomnia, depression, erythematous skin and emotional instability. Later on, the lymph nodes, the spleen, and the liver may get enlarged. Complications may include infection of the heart, the brain and the lining of the brain, as well as inflammations of the nerves, the testes, the gallbladder, the liver and the bones. Usually this acute episode lasts from 24 to 48 hours, but may last evend for 7 to 10 days. The frequency of such attacks range from

twice weekly to once a year, the most common intervals, however, are from 2 to 4 weeks. There may be a decrease in the severity and the frequency of the attacks when persons get older or due to the development of amyloidosis. Approximately one third of female patients are infertile, and 20-30% of the pregnancies results in a fetal loss.
I think, that also an immune trigger has an important role in the pathogenesis of this disease. The immune response will be triggered by *Mycoplasma fermentans* or/and the *HTLV*.
FPS is a hypersensitive process, with no proved evidence of an autoimmune etiology. MEFV gene determine the development of FPS in case of a Brucella infection.
Diagnosis: symptomatically, by bacterial culturing, blood tests.
Treatment: by administering antibiotics, f.i. Doxycycline. Colchicine is effective in the preventing attacks of FPS and in the preventing the development of amyloidosis.
RFR method: detects and may eliminate the pathological microorganism, but should only be used together with antibiotic therapy.
The most frequent resonances of Brucella are: 329, 355, 364-365, 382 kHz
The most frequent resonances of Mycoplasma fermentans are: 442-444, 447-451, 518-519 kHz
Concerning the Human T-cell Lymphotropic Virus, see Chapter 5.1.10.1.

14.12. Ascites and Peritonitis

14.12.1. Ascites

Ascites (AS) is the term for every accumulation of fluid in the peritoneal cavity. The accumulation of ascitic fluid points to an extremely enhanced total-body sodium and water excess. Three theories for ascites formation have been proposed, that is underfilling, overflow and peripheral arterial vasodilation.
The theory of underfilling suggests a primary abnormality, i.e. an inappropriate accumulation of fluid within the splanchnic vascular bed due to portal hypertension and a consequent decrease in the effectively circulating blood volume. This phenomenon activates the production of plasma renin and aldosterone as well as the sympathetic nervous system, resulting in the retention of renal sodium and water.
The theory of overflow suggests that the primary abnormality is the inappropriate renal retention of sodium and water without any volume decrease. This theory had been developed in accordance with the observation that patients with cirrhosis show intravascular hypervolemia rather than hypovolemia.
The most recent theory, **the peripheral arterial vasodilation hypothesis**, shows components of the other two theories, suggesting that the portal hypertension leads to vasodilation, decreasing thus the effective arterial blood volume. As the disease progresses, the neurohumoral excitation will increase, more renal sodium will be retained, expanding thus the plasma volume. This process leads to the overflow of fluid into the peritoneal cavity. The vasodilation theory proposes that in case of cirrhosis underfilling occurs in the early phase, while overflow but later on.
Although the sequence of events occuring between the development of portal hypertension and renal sodium retention is not entirely clear, portal hypertension will lead apparently to an increase in the nitric oxide levels. Nitric oxide mediates the splanchnic and peripheral vasodilation. The nitric oxide synthetase activity of the hepatic artery of patients with ascites is higher than of those having no ascites.
Regardless of the initiating event, quite a number of factors contribute to the accumulation of fluid in the abdominal cavity. Elevated levels of epinephrine and norepinephrine are well-documented factors. Hypoalbuminemia and a reduced plasma oncotic pressure favor the extravasation of fluid from the plasma to the peritoneal fluid, so that, unless both portal

hypertension and hypoalbuminemia are present, ascites is not frequently developing in patients with cirrhosis. Jaundice, palmar erythema and spider angiomas are physical signs suggesting liver disease. An elevated jugular venous pressure can suggest an ascites of cardiac origin. A firm nodule in the umbilicus, the so-called Sister Mary Joseph nodule suggests a peritoneal carcinomatosis originating from a primary gastric, pancreatic or hepatic tumor.
A pathologic, left-side supraclavicular node, the so-called Virchow node points to the presence of an upper abdominal malignancy.
A transudative ascites can develop caused by infectious hepatic cirrhosis, alcoholic cirrhosis, heart failure and portal vein thrombosis.
An exudative ascites can develop due to peritoneal carcinomatosis, inflammation of the pancreas or the biliary system, nephrotic syndrome, peritonitis, ischemic or obstructed bowel diseases.
In case of ascites the peritoneum can be intact or damaged:
An intact peritoneum can be observed
in case of portal hypertension (serum-ascites albumin gradient [SAAG] >1.1 g/dL)
hepatic congestion, congestive heart failure, constrictive pericarditis, tricuspid insufficiency, Budd-Chiari syndrome,
liver disease, cirrhosis, alcoholic hepatitis, fulminant hepatic failure, massive hepatic metastases
Hypoalbuminemia (SAAG <1.1 g/dL)
Nephrotic syndrome
Protein-losing enteropathy
Severe malnutrition with anasarca
Miscellaneous conditions (SAAG <1.1 g/dL)
Pancreatic ascites
Bile ascites
Nephrogenic ascites
Urine ascites
Ovarian disease
A damaged peritoneum can be observed (SAAG <1.1 g/dL)
in case of certain infections:
Bacterial peritonitis
Tuberculous peritonitis
Fungal peritonitis
Human immunodeficiency virus (HIV)-associated peritonitis
in case of malignant conditions:
Peritoneal carcinomatosis
Primary mesothelioma
Pseudomyxoma peritonei
Hepatocellular carcinoma
in case of vasculitis
Granulomatous peritonitis
Eosinophilic peritonitis.

14.12.2. Peritonitis

Peritonitis is the inflammation of the serosal membrane that lines the abdominal cavity and the organs contained therein. The infection of the ascitic fluid with no intra-abdominal infection occurs usually among patients with chronic liver diseases due to the translocation of enteric bacteria. Peritonitis is often caused by an infection owing to the perforation of the bowel, f.i. owing to a ruptured appendix or colonic diverticulum.A secondary bacterial

peritonitis is characterized by an infected ascitic fluid associated with an intra-abdominal infection.
Concerning women, localized peritonitis does most often occur due to an infected fallopian tube or due to a ruptured ovarian cyst.
The most frequent causative bacteria are mostly Gram negative, such as *Escherichia coli, Klebsiella pneumoniae, Enterococci, Pseudomonas species*, though *Streptococcus or/and Staphylococcus species* are Gram positive. *Enteroviruses* can but rarely cause infectious peritonitis. Anaerob bacteria, such as *Bacteroides fragilis and other Bacteroides species, Eubacterium, Clostridium, Anaerobic Streptococcus species and Candida albicans* can sometimes also cause infectious peritonitis.
The disease may also be caused by chemically irritating materials, such as gastric acid induced by a perforated ulcer or the bile coming from a perforated gall bladder or a lacerated part of the liver.
The symptoms of peritonitis are: abdominal pain, vomiting and tenderness on palpation, rigidity of the abdominal wall muscle and systemic signs of inflammation. Patients may show acute intensive symptoms or unrecognizable ones, and can thus develop a limited and mild disease, or a systemic and severe one with septic shock.
Patients with peritonitis are often feeling unwell and distressed. Fever is usually present, though patients with sepsis may suffer from hypothermia as well.
Diagnosis: by blood analysis, basic metabolic profile, liver enzimes and coagulation examinations. By ascites analysis (color, protein content, cell counting, bacterium culturing, etc.) By cytopathology.
Treatment: by restricting salt and water, by administering diuretics, antibiotics. By paracentesis, by liver transplantation.
RFR method: can detect and eliminate the pathogens present.
The most frequent viruses present in case of ascites are: Hepatitis viruses, Cytomegalovirus, HTLV and HPV. As to their frequencies, see their special chapters.

15. GENITOURINARY TRACT DISORDERS ASSOCIATED WITH INFECTIONS

The inflammations of the urogenital tract are not always caused by a present urinary tract infection. In case of a urinary tract infection there are a significant number of pathogens (bacteria, viruses and fungi) present in the urine. Glomerulonephritis is f.i. a disease which may or may not result from the immediate or late effects of urinary tract infections. Just as glomerulonephritis cannot be assumed to exist if there is a urinary tract infection, the latter may not be present in case of a glomerulonephritis.

15.1. Inflammations of the Kidneys

Nephritis is the common name of the inflammations of the kidneys. Inflammations, showing sometimes similar symptoms, though they develop and show histopathology in many a different way, are the following:

15.1.1. Acute Nephritic Syndrome

This acute nephritic syndrome is usually caused by an acute postinfectious glomerulonephritis being an inflammation of the glomeruli, and is characterized by the sudden appearance of blood in the urine with clumps of red blood cells, variable amounts of protein in the urine and by hypertonia. This most often occuring acute postinfectious glomerulonephritis form causing acute nephritic syndromes is **immune-complex mediated**, caused by chronic persistent infections or other diseases with a prolonged antigenic stimulation. In these glomerulopathies, mediated by a III. type hypersensitivity reaction, the immune complexes are formed in the circulation and then will get deposited in the glomerular basement membranes. The antigenic component of these heterologous immune complexes is often found to be of *viral* origin, f.i. *chickenpox, Hepatitis B and Hepatitis C viruses, CMV, EBV and HIV.* Also systemic symptoms may accompany this glomerulonephritis form, as the vascular membranes may be affected by immune-complex depositions in various loci other than the glomeruli.

An acute nephritic syndrome can often follow a *streptococcal* infection, in which case, the disease is named **post-streptococcal glomerulonephritis.** The pathogenesis of this disease may occur after a streptococcal pharyngitis, tonsillitis or a streptococcal pyodermal skin infection. The latent period between the beginning of the streptococcal infection and the development of the clinical glomerulonephritis averages from one to three weeks. In case of this illness the glomeruli are damaged by the accumulation of antigens of the destroyed streptococci clumped together with the antibodies neutralizing them and by the inflammation caused by complement activation. These immune complexes coating the membranes of the glomeruli can interfere with their filtering function. Though there may be microscopic hematuria with pharyngitis per se be present, in case of a developing nephritis, this microscopic hematuria will then disappear and only reoccur 1 to 3 weeks later.

Symptoms: A developing post-streptococcal glomerulonephritis results in a small daily urine volume, with an appearence resembling cola beverage or dilute coffee. A persistent fever suggests either a continued streptococcal infection or a systemic disease associated with acute glomerulonephritis, f.i. SLE or PAN. Facial edema is very common, among older patients a pedal edema may be present even if the face does not get swollen. In severe cases the hypertension may be associated with encephalopathy and retinopathy

including hemorrhages, exudates and papilledema. Severe headaches, somnolence and even convulsions may occur as the manifestations of encephalopathy.
Postinfectious glomerulonephritis with the symptoms of an acute nephritic syndrome can be caused by other bacterial infections, too, f.i. by *syphilis, Streptococcus species, Staphylococcus aureus, Mycoplasma species etc.* and even by *malaria.*

15.1.2. Rapidly Progressive Nephritic Syndrome

This rapidly progressive nephritic syndrome, caused by the socalled crescentic glomerulonephritis, is a rare disorder in which most of the glomeruli are partly destroyed, and the syndrome results in abruptly developing severe kidney failures with azotemia, edema, with protein blob, red blood cell clumps in the urine causing macroscopic haematuria, proteinuria and anemia. This syndrome is present in case of Goodpasture's syndrome, an autoimmune illness, caused by antibodies against the kidney and the lungs. These antibodies attack the basement membrains of the glomeruli. The production of other damaging antibodies can be related to *viral* infections and to other autoimmune disorders, f.i. *SLE* A progressive nephritic syndrome may develop also in case of vasculitic disorders, such as *Wegener's granulomatosis* and *PAN*, in which cases there are no immune deposits found when staining, but the blood tests are positive for ANCA antibodies (see Chapters 11.16. and 12.13.).

15.1.3. Nephrotic Syndrome

This syndrome can be caused by many a disease affecting the kidneys and resulting in a severe, prolonged loss of small sized protein (albumin, immunoglobulins and antithrombin) in the urine. Having this syndrome the serum level of albumin is decreased, the level of lipids in the serum is increased (hyperlipidaemia and hypercholesterinaemia), lipiduria and the retention of a great amount of salt and water in the body are characteristic, though the renal functions remain normal.
The characteristic **renal symptoms** of the syndrome are: edema around the eyes and over the legs, pleural effusion and ascites. In very serious cases hypertonia, azothemia and haematuria can also occur.
Based on etiology this illness can develop either in primary illnesses (i.e. if the exact cause of the disease is idiopathic) or in secondary ones (i.e. if the syndrom is associated with more defined diseases affecting not only the kidneys but other organs as well). Based on its histologic findings the primary forms of the disease can be sorted into three groups, such as *minimal change disease, focal segmental glomerulosclerosis* and *membranous nephropathy.* Examined light-microscopically, in most cases the glomeruli appear to be normal (minimal change disease, otherwise: nil disease or lipoid nephrosis) and the corticosteroid therapy proves to be effective, but corticosteroids are generally ineffective concerning patients suffering from a nephrotic syndrome secondary to a membranous glomerulopathy or to a proliferative glomerulonephritis.
The nephrotic syndrome can be present in case of certain autoimmune diseases such as SLE, rheumatoid arthritis and polyarteritis nodosa, can be associated with malignancies (f.i. leukemia, lymphoma, Wilms tumor, pheochromocytoma) and can be caused by toxins and medications (f.i. penicillamin and aspirin) as well. An other origin of this syndrome used to be postinfectious (f.i. bacteria such as *group A beta-hemolytic streptococci, Haemophilus influenzae, lues, Mycobacterium tbc, Toxoplasma, Staphylococcus aureus, Escherichia coli, Mycoplasma fermentans* and viruses such as *Varicella, Rubella, hepatitis B, HIV type 1, HTLV-1, Epstein-Barr Virus, Cytomegalovirus,* as well as *malaria*, etc.). In postinfectious cases the different bacterial and viral antigens adsorbed to the glomerular basement membranes provoke pathological immune responses of the host, though the

precise mechanism of these processes are as yet not exactly defined. There do exist evidences that also genetic factors may be involved in the pathogenesis.
The group of secondary connatal nephrotic syndromes include cases caused by intrauterine infections (eg. *toxoplasmosis, cytomegalovirus, rubella, syphilis*), cases associated with gonadal dysgenesis, nail-patella syndrome and Lowe syndrome.
Diagnosis of kidney infections: is based on the symptoms, on blood and urine tests and by imaging of the kidneys, by biopsy of the kidneys, etc.
Histological analysis of the biopsy samples by light microscopic examination:
Minimal-change nephrotic syndrome (MCNS) indicates a normal or a minimally altered glomerular morphology. There may be found minimal mesangial alterations, but immunoglobulins and deposits are absent observed by electron microscopy. The only significant change seen by electron microscopic examination is the flattening and fusion of the epithelial cell podocytes.
Focal global glomerulosclerosis (FGGS) describes a more than 5% globally sclerotic glomeruli occurring in focal areas together with glomeruli remaining normal.
Focal segmental glomerulosclerosis (FSGS) describes a lesion in which some glomeruli are involved with segmental sclerosis (one lobule or section within a glomerulus), together with the remaining glomeruli being normal. As this lesion is focal and often confined to the juxtamedullary nephrons, it can easily be overlooked at renal biopsy examinations.
Immunofluorescent microscopy can yield various alterations. In some cases, all classes of immunoglobulins and complement components are trapped in the sclerotic area; while in other cases, distinct immune-complex deposits can be found, particularly those of IgM type.
The **Mesangial proliferative glomerulonephritis (MPN)** but recently distinguished from MCNS shows by light microscopy minimal to moderate proliferations of the mesangial cells with a certain mesangial expansion. The most striking change is observed with immunofluorescent microscopy, where IgM, IgG, and C3 deposits are often to be seen.
Membranoproliferative glomerulonephritis (MPGN), named also mesangiocapillary glomerulonephritis, shows a distinct histologic picture; where all glomeruli are involved.
Treatment: is very complex as the etiology of this disease is likewise very complex.
Specific therapy: by observation of infections: A child with nephrotic syndrome is a prime candidate for infection, and the chance for dissemination increases if steroids are administered indiscriminately. Therefore, a child who is febrile or has evidence of an infection should be closely observed for a brief period while appropriate studies are performed. A child from an environment conducive to tuberculosis should be tested. Infections should be actively treated, but a prophylactic therapy is usually not indicated.
Nonspecific therapy: by administering glucocorticoids to stop the false immune response and the immune inflammation, by diuretic therapy, antihypertensive therapy, etc.
There is solely the **RFR method** capable to detect and eliminate viruses and antibiotic resistant bacteria. In case of a postinfectious nephrotic syndrome there are usually many different frequency resonances to be detected. Having measured these resonances, one need to eliminate everyone of the pathogen microorganisms resonating on these frequencies. Though certain antibiotics may damage the renal function and RFR method is to be prefered, the supporting of RFR method with antibiotics can be thus expedient.
The most frequent resonances of nephrotic syndromes are:
In case of **primary nephrotic syndromes:**
Minimal-change nephrotic syndrome (MCNS): 320-326, 327-339, 345-352, 408-416 kHz
Focal global glomerulosclerosis (FGGS): 320-326, 358-376, 380-387, 442-451 kHz
Membranous nephropathies: 326-339, 358-376, 380-387, 442-451, 493-495 kHz
Hereditary nephropathies: 320-326, 327-339, 345-352, 358-376, 380-387, 408-416, 442-451, 493-495 kHz

As to the most frequent resonances of **secondary nephrotic syndromes**, see the special Chapters of the causative diseases:
In case of Membranous nephropathy: Viral infections (f.i. HBV, HCV), Syphilis, Sjögren's syndrome, SLE, Diabetes mellitus, Sarcoidosis and Malignancies (cancers)
In case of Focal segmental glomerulosclerosis: Hypertonia, HIV, Diabetes mellitus, Obesity
In case of Minimal change nephrotic syndrome: Malignancies

15.1.4. Chronic Nephritic Syndrome

This slowly progressive syndrome can occur associated with several diseases, in case of which the glomeruli are damaged and the kidney function worsens over years. This syndrome may be caused by *viral* infections. The occurrence of hematuria and the red blood cell casts generally indicate the presence of either a diffuse or a focal proliferative glomerulonephritis. This proliferation may involve the mesangium, the endothelial or the epithelial capillary cells and their combinations as well. The infiltration of the glomeruli with polymorphonuclear leucocytes is termed exudative glomerulonephritis. A number of patients with glomerular diseases may have none of the manifestations of an acute nephritic syndrome, are symptomless, but have urinary abnormalities found by routine laboratory examinations.

15.1.5. Acute Tubulointerstitial Nephritis

This illness is characterized by a suddenly beginning kidney failure, caused by damages to the kidney tubules and to its surrounding interstitium. These damages may be *toxic reactions caused by drugs*, f.i. amphotericin B and aminoglycosides, or *allergic reactions to drugs* f.i. to penicillin and aspirin. Other causes include *bacterial infections* of the kidneys, and *malignancies,* such as leukemia and lymphoma.

15.1.6. Chronic Tubulointerstitial Nephritis

Chronic tubulointerstitial nehritis is a chronic kidney disease in which the damages of the tubules and their surrounding tissue are more severe than the damages of the glomeruli and the blood vessels. In case of certain types of this chronic tubulointerstitial nephritis kidney stones can be formed. This disease can be caused by certain viral infections and can develop due to chronic bacterial toxicosis, too.
Diagnosis of kidney diseases: symptomatically and by urinary analysis, culturing bacteria and viruses in the blood, by biopsy, ultrasound, x-ray, immunological tests, etc.
Treatment is depending on the cause of the illness. By administering effective antibiotics for a long time treating the causative infection. In case of a rapid progressive nephritic syndrome high dose corticosteroids are usually given intravenously for about a week and then by mouth. Immunosuppressants, such as Cyclophosphamide and Azathioprine can be of great value.
RFR method: detects the viruses and bacteria producing toxins or antigens! Eliminates the causative viruses and bacteria! RFR method is not a diagnostic method! Various different bacteria and viruses may have the same frequencies.
The most frequent resonances in nephritis are: 317, 325, 332-340, 352-356, 372, 378, 381, 392-396, 402, 451, 476, 545-560 kHz
The resonant frequencies of Streptococcus are: 307-310, 312-321, 324, 337-340, 345, 351-353, 363-376, 381-385, 391, 397-413, 418, 426, 432, 440-447, 450-453, 466-468, 478, 486, 498, 508, 511-516, 542 kHz
The resonant frequencies of Escherichia coli are: 317-319, 323-328, 337, 352-358, 390-397, 408, 410-412, 422-426, 435, 478, 489, 560, 576-579 kHz

The resonant frequencies of Pseudomonas are: 324-325, 330-335, 339, 364, 372-374, 377-380, 388-397, 401, 414, 428, 496, 579 kHz
The resonant frequencies of the Salmonella group are: 318, 329-339, 354, 360-370, 380-386, 390-395, 428, 452, 558 kHz
The resonant frequencies of the Proteus group are: 320-329, 333-339, 345-352, 408-416, 426, 516, 522-529, 535 kHz
The resonant frequencies of Shigella group are: 310, 313, 315-321, 369, 388-398, 403-410, 423-425, 496, 499, 506 kHz
The resonant frequencies of Adenovirus are: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz
The resonant frequencies of the Herpes Virus group are: 290-294, 301-307, 310, 328, 331-340, 344-346, 348, 352-365, 380, 397-402, 413, 425, 431-433, 449-450, 458-459, 474, 478, 483-486, 533, 540 kHz
The resonant frequencies of the Coxsackie virus group are: 287-295, 297-301, 307-308, 313, 331, 333-336, 340-345, 350, 360-376, 289, 392, 396, 407-416, 419-426, 430, 443-445, 472, 475, 498, 533-544, 546, 552-557, 564 kHz
The resonant frequencies of Cytomegalovirus are: 305, 349, 407-412, 512, 530-536, 543-548 kHz
The resonant frequencies of Epstein-Barr Virus are: 291, 337-339, 342-347, 352, 360, 370-385, 397-398, 403, 422, 451, 491, 516-518, 582 kHz
The resonant frequencies of Candida albicans are: 297, 308, 338, 345, 352-362, 372, 380-390, 403, 410, 420-424, 448-453, 460, 474, 520-523, 544, 580-590 kHz
The resonant frequencies of the Chlamydia group are: 317-319, 376-390, 429, 440-444, 480-482, 566 kHz
The most fequent resonant frequencies of the Mycoplasma group are: 307-308, 321-324, 337-350, 442-451, 493-495 kHz

15.2. Goodpasture's Disease

Goodpasture's disease (also named anti-Glomerular Basement Membrane (GBM) disease or Goodpasture's syndrome) is a rare immune illness characterized by autoantibodies to the glomerular basement membranes, by a rapid destruction of the kidneys (due to an acute glomerulonephritis) and the haemorrhaging of the lungs. In case of this illness the patient's immune system attacks the cells presenting Goodpasture's antigens, found in the kidneys (GBM) and the lungs (alveoli capillaries). This type II hypersensitivity reaction causes damages to these organs triggering a local destructive inflammation. The occuring glomerulonephritis is usually rapidly progressing, but the damages of the lungs are less serious, characterized by a dry cough and a minor breathlessness which may last for many years before the more severe damages of the developing autoimmune disease come to pass. Patients with anti-GBM disease may have symptoms ranging from pulmonary hemorrhages with minimal or no renal involvement to full-blown renal failure with a limited or no pulmonary involvement. In case of children there is a limited experience concerning this disease. Lung damage may cause a severe impairment of oxygenization so that intensive care should be required. This lung hemorrhage causing a coughing up of blood and leading to anemia occurs most often among smokers and those with damages caused by mycoplasmal or viral lung infections and those exposed to fumes. The antigens of the microorganisms adsorbed to the basement membranes start an immune-autoimmune process usually among *genetically predisposed* individuals. HLA-DR2 is expressed among 80-90% of patients with anti-GBM disease compared to 20-30% of a control group of blood donors. The simultaneous expression of HLA-B8 and HLA-DR2 is associated with a worse prognosis because of the tendency to form glomerular crescents. The production of anti-GBM antibody is strongly associated with HLA-DR15 and HLA-DR4 alleles, and in

contrast, HLA-DR7 and HLA-DR1 being highly protective, have strong negative associations to this disease.

The role of infections (f.i. *Influenza type A2*) in regard to etiology can be supposed, as an upper respiratory tract infection or a flu-like illness does often occur before the onset of this anti-GBM disease. The RFR examination usually shows many different pathogen resonances indicating infections caused by microorganisms such as *Mycoplasma, Hemophilus influenzae, HTLV, Cytomegalovirus, EBV, influenza virus and other viruses.*

The symptoms of this Goodpasture's disease usually are the coughing up of blood and a burning sensation when urinating. Its other signs may be vague, such as fatigue, nausea, dyspnea and pallor. These signs are first followed by small amounts of blood in the urine, protein in the urine and then their worsening, too. Goodpasture's syndrome may last only for a few weeks but sometimes even for 2 years. The bleeding in the lungs can be very serious in some cases, but does not lead usually to permanent lung damages. The damage to the kidneys, however, may be long-lasting.

Diagnosis: Because of the vagueness of the early symptoms and the rapid progression of the disease, the diagnosis is often established only very late in the course of the disease. Kidney (and pulmonal) biopsy can be the fastest way to ensure the diagnosis and to gain information about the extent of the disease and likely about the effectivity of the treatment. Tests for anti-GBM antibodies may also be useful, combined with tests for ANCAs, which can also be present in this illness.

Treatment: Symptomatically, administering corticosteroids and immunosuppressants (cyclophosphamide) to dampen the body's immune response. A serious side effect of this kind of therapy is that the patient will be more susceptible to infections. The concentration of anti-GBM antibodies in the blood can be reduced by apheresis. Antibiotic treatment of lung infections.

RFR method: detects and may eliminate the pathogen microorganisms.

The most frequently found resonances are: 308-321, 367-387, 442-451, 493-497 kHz

RFR method can inhibit the development of this immune disease by eliminating everyone of the pathogen microorganisms.

15.3. Renal Corticomedullary and Perinephric Abscess

Renal cortical abscess results from hematogenous spread of bacteria from a primary extrarenal focus of infection. The most frequent etiologic agent of cortical abscess cases are *Staphylococcus aureus, Klebsiella, Enterobacter, Serratia, Pseudomonas, Streptococcus fecalis, Proteus groups bacteria and E. coli.* Other causes include fungi, especially Candida species, *Mycoplasmal species* and Mycobacterium tuberculosis. In contrast, a **renal corticomedullary abscess** develops as an ascending infection caused by microorganisms already isolated from the urine. These abscesses are usually associated with an underlying urinary tract abnormality, f.i. with a vesicoureteral reflux. Renal corticomedullary abscesses occur most often in individuals with diabetes mellitus with or without urinary tract obstruction. Renal corticomedullary infections include most of the acute and chronic infectious processes of the kidneys below to be mentioned.

A severe renal parenchymal damage in combination with a corticomedullary abscess is more likely to extend to the renal capsule and, perforating, it forms a **perinephric abscess.** Perinephric abscesses are located between the capsule of the kidney and the Gerota fascia. The abscesses remain there confined owing to this fascia. Perinephric abscesses usually occur due to disruption of a corticomedullary intranephric renal abscess, recurrent pyelonephritis, xanthogranulomatous pyelonephritis, or an obstructing renal pelvic stone causing pyelonephrosis. Approximately 30-40% of the cases are caused by a hematogenous dissemination of microorganisms from the loci of infection, f.i. as wound infection, furuncles or pulmonary infections. Abscesses can also be caused by ascending urinary tract infections, by infections of the intestine, pancreas, liver, gall bladder, prostate

and pleural cavity, by osteomyelitis of the adjacent ribs or vertebrae. Due to a superimposed infection, a perirenal hematoma can sometimes develop into a perinephric abscess.

Acute focal bacterial nephritis usually causes interstitial inflammation within a focal area of the kidney. Its histologic characteristics are a marked infiltration with polymorphonuclear leukocytes at the apex of the medulla with distortion of the glomeruli and renal tubules. An other form is the **emphysematous pyelonephritis**, an uncommon, but severe necrotizing form of acute multifocal bacterial nephritis. Abdominal radiography shows a characteristic intraparenchymal gas formation. The gas is located within the renal parenchyma rather than within the renal collecting system, suggesting an infection with gas-forming anaerobic or facultative anaerobic pathogens.

Xanthogranulomatous pyelonephritis is a chronic renal infection often associated with renal calculus. As a result of a long-standing infection, the kidney gets enlarged and fixed to the retroperitoneum by a perirenal fibrosis and extension of the granulomatous inflammation. Xanthogranulomatous pyelonephritis is occasionally associated with renal cell carcinoma, transitional cell carcinoma and squamous cell carcinoma.

The most common **symptoms** are fever, abdominal pain, chills, dysuria, weight loss, lethargy and gastrointestinal symptoms. Pleuritic pain may occur due to diaphragmatic irritation. If the abscess is pressing the nearby nerves, the pain caused may be felt in the groin, thighs, or knees.

Diagnosis: by its symptoms, x-ray, CT scanning, MRI and PETscan.

Treatment: by administering antibiotics. In case of paranephric abscesses by optimalizing these individual medical conditions prior to surgery.

RFR method detects and can eliminate the pathogen microorganisms.

The most frequent resonances are: 313-322, 320-329, 331-335, 345-352, 355-357, 360-375, 376-381, 384-390, 392-393, 388-403, 408-415, 416-421, 429-436, 442-451, 470-480, 393-395 kHz

15.4. Kidney Cyst and Polycystic Kidney Disease

These renal cystic diseases have various etiologies, such as

1. Developmental – f.i. Multicystic dysplastic kidney (MCDK)
2. Genetic – f.i. the Autosomal recessive polycystic kidney disease (ARPKD)
3. Autosomal dominant polycystic kidney disease (ADPKD),
4. Juvenile nephronophthisis (JNPHP), medullary cystic kidney disease (MCKD),
5. Glomerulocystic kidney disease (GCKD),
6. Cysts associated with systemic diseases – Von Hippel-Lindau Disease (VHL),
7. Tuberous sclerosis
8. Acquired - Simple cysts, acquired cystic renal disease, medullary sponge kidney (MSK),
9. Malignancy - Cystic renal cell carcinoma (RCC)

The most common larger cysts include acquired cysts, simple cysts and cysts associated with the ADPKD. Smaller cysts are present in case of ARPKD, JNPHP, MCKD and MSK. Among adults, renal angiomyolipomas and RCC may also have cystic components.

Cysts usually develop from renal tubule segments and most of them will be detached from the parent-tubule after becoming a few millimeters in size. The development of cysts is generally attributed to an increased proliferation of the tubular epithelium, to abnormalities of the tubular cilia, and to an excessive fluid secretion. In case of polycystic renal diseases hypertension and a progressive renal failure will develop usually after the patient's third decade of life. The **autosomal dominant polycystic kidney disease (abbreviated as ADPKD)** is an uncommonly occuring disease concerning children and sometimes neonates as well.

This ADPKD is an inherited condition comprising at least 3 phenotypically indistinguishable but genetically distinct entities, depending on which of the 3 genes–

PKD1, PKD2, or PKD3-becomes mutated. The disorder can be transmitted as an autosomal dominant trait. Cysts vary in size from barely visible to several centimeters in diameter. In contrast to fluid from simple renal cysts, which is biochemically similar to plasma, the biochemical features of the fluid content of ADPKD cysts are closer to those of urine. These cysts can become infected, the aspiration of its fluid may reveal purulent contents. The incidence of renal cell carcinoma is only slightly increased concerning patients with ADPKD; while a greater rise in incidence is associated with cystic disease and dialysis (infection with *HPV*). The stromal changes are nonspecific, dystrophic calcifications are common.
The risk of the development of a renal cell carcinoma is greater if this disease is associated with von Hippel–Lindau disease or with tuberous sclerosis.
Getting older than 60 years, most patients with ADPKD will get a renal failure combined with hypertension. More easily occuring infections, hemorrhages, cyst rupture and renal calculus disease may be complications of ADPKD. Fever, dysuria and leukocytosis are often present caused by urinary tract infections. Calculi caused renal and ureteric colic can often come to pass. Hemorrhages, intracystic or retroperitoneal, can cause hematuria, abdominal pain, and sometimes a massive hemorrhagic shock and anemia as well. Polycythemia is a rare, but well known association secondary to increased erythropoietin production.
Diagnosis: by ultrasonography, urography, nephrotomography, sonography, MRI, CT scanning, angiography, etc. Caused by a *nanobacterial* infection the cysts may be calcified. The presence of renal calculi may signify urinary tract infection. By urinary tests regarding pathogen bacteria and their culturing.
Treatment: in case of hereditary kidney diseases there is no specific therapy known. In case of an originally infectious abscess or cystic disease the elimination of the pathogens by administering antibiotics. Surgically.
RFR method: detects every one of the pathogen microorganisms and eliminates them.
The most frequent resonances found in regard to these diseases are: 312-329, 332-339, 345-352, 408-416, 502-503, 556-558 kHz
There can be many a different pathogen present in everyone of these cases.

15.5. Urolithiasis

Urolithiasis is a process of stone formation in the urinary tract (the kidneys, the bladder and the urethra). These stones are formed if the urine is oversaturated with salt and minerals such as calcium oxalate, struvite(magnesium ammonium phosphate), uric acid and cystine or if the urine lacks the normal inhibitors of stone formation. Stones can vary considerably in size from small gravel like stones, to large stag horn calculi. The calculi may stay in the position in which they are formed initial, or migrate down the urinary tract producing symptoms along the way. Recent studies suggest that the factor involved in the formation of a stone may be the presence of *nanobacteria* forming a calcium phosphate shell. These small intracellular bacteria are present at the center of more than 95% of all stones. Renal stones are associated with nanobacteria or with a combined infection of nanobacteria and other pathogens, and with a progressive damage of the renal functions. Stones coming from above are rarely retained in the bladder unless obstruction and residual urine are present.
There are several *risk factors* known to increase the potency of a susceptible individual to form stones, including: anatomical anomalies, chronic dehydratation, diseases f.i. hypertension, gout, sarcoid, hyperparathyroidism, bone diseases and bone metastases causing hypercalciuria. Metabolic disorders, which increase the excretion of calcium in the urine, f.i. excessive ingestion of milk, alkali and vitamin D could also be predisposing; just like every other problems connected with renal tubular acidosis; metabolic acidosis, hypercalciuria, hyperuricosuria, cystinuria, glycinuria, caused f.i. by inherited illnesses.

Chronic dehydration, an important cause of stone formation, might probably be responsible for the high incidence of nephrolithiasis of patients living in tropical climates and those with chronic diarrhea. Urinary obstruction may be favourable to stone formation in case of a nanobacterial infection together with other urinary infections. Most calculi originate from the kidney and proceed distally, creating urinary obstructions of various degrees when they become lodged in narrow areas, including the ureteropelvic junction, the pelvic brim and the ureterovesical junction. The location and the quality of the pain depend on the position of the stone in the urinary tract. The severity of the pain depends on the degree of the obstruction, the presence of ureteral spasm and the presence of any associated infections.

Symptoms: The hard, stone-like masses can be formed anywhere within the urinary tract and may cause pain, bleeding, obstruction of the urine flow and lead to infections. They may be formed and passed by urination even for years with no deleterious effect on the renal function and causing no discomfort except for occasional renal colic. Tiny stones may not cause any symptoms. Stones impacted within the ureter cause abrupt, severe, colicky pains in the flank and lower abdomen, radiating to the testicles or the vulvar area. Intense nausea, with or without vomiting usually comes about. Stones lodged at the ureterovesical junction also may cause irritative voiding symptoms, dysuria and frequent urination. Stones obstructing the ureter, the renal pelvis, or any of its drainage tubes may cause back pain or severe, colicky pains. Other symptoms include abdominal distension, chills and blood in the urine. Calculi that have entered the bladder are usually asymptomatic and will pass relatively easily while urinating.

The classic patient with renal colic is writhing in pain, pacing about, unable to lie still. Fever, if present, suspects infected hydronephrosis, pyonephrosis or perinephric abscess.

The most common finding in case of ureterolithiasis is a flank tenderness due to the dilatation and spasm of the ureter coming from the transient obstruction as the stone passes from the kidney to the bladder.

Calcium stones (75%): can be composed by calcium oxalate, calcium phosphate and calcium urate and be associated with the following disorders: hyperparathyroidism, increased gut absorption of calcium; the most common identifiable cause of hypercalciuria, and can be treated with calcium binders or thiazides plus potassium citrate. The most important organisms playing role in the stone formation are the *nanobacteria.*

Struvite (magnesium ammonium phosphate) stones (15%) stones are only formed secondarily to chronic urinary tract infections with gram-negative rods capable of splitting urea into ammonium, which will combine with phosphates and magnesium. These bacteria are *Proteus, Pseudomonas* and *Klebsiella* species. Escherichia coli is not capable of splitting urea and is, therefore, not associated with struvite stones. In case of these stones the urine pH is typically greater than 7.

Uric acid stones are associated with urine pH less than 5.5 and with high purine intake.

Cystine stones can be formed due to an intrinsic metabolic defect resulting in failure of the renal tubular reabsorption of cystine, ornithine, lysine and arginine. The urine becomes supersaturated with cystine with resultant crystal deposition. This form of illness can be treated with low-methionine diet, with binders such as penicillamine, a-mercaptopropionylglycine, with large urinary volumes and alkalinizing agents.

Drug-induced stone disease: a number of medications or their metabolites can precipitate in urine causing stone formation. These include indinavir; guaifenesin; triamterene; silicate (the overuse of antacids containing magnesium silicate); and sulfa drugs including sulfasalazine, sulfadiazine, acetylsulfamethoxazole, acetylsulfasoxazole and acetylsulfaguanidine.

Diagnosis: by kidney, ureter, and bladder radiography, urinanalysis, CT, MRI, gadolinium-enhanced 3-D FLASH MR urography, x-ray, ultrasonography and bacterium culturing. Urine pH higher than 7 suggests the presence of urea-splitting organisms, such

as *Proteus, Pseudomonas,* or *Klebsiella* species and struvite stones, a urine pH less than 5 suggests uric acid stones.
Prevention: In case of renal calcium leak, by administering thiazide diuretics, in case of renal phosphate leak by administering phosphate supplements. Hyperuricosuria should be treated with allopurinol, low purine diet and with alkalinizing agents, such as potassium citrate. Hyperoxaluria can be treated with dietary modification, oxalate binders, vitamin B-6 and orthophosphates. Hypocitraturia can be treated with potassium citrates and hypomagnesuria with magnesium supplements. Drinking large amounts of fluids is also helpful.
Treatment: symptomatically: by administering analgesics, narcotics, Alpha-1-adrenergic receptor antagonists f.i. nifedipine, hydratation therapy. Antibiotics are necessary in case of secondary bacterial infections. Surgery is frequently a percutaneous nephrolithotomy, which might be followed by ultrasound treatment. Kidney stones can sometimes be broken up by sound waves. Small stones being in the lower part of the ureter may be removed endoscopically.
RFR method: detects and may eliminate the nanobacteria and all the other pathogen microorganisms. Use together with some other types of treatment which depends on the composition of the existing stone formations.
The most frequently found pathogen microorganisms are:
Nanobacteria: 294-298, 305-310, 317-318, 324-325, 332-334, 340-344, 375-386, 424-426, 430-435, 438, 466-476, 482-486, 552-560 kHz
Proteus group: 320-329, 333-339, 345-352, 408-416, 426, 516, 522-529, 535 kHz
Klebsiella: 381, 392, 397-404, 416-422, 429 kHz
Pseudomonas: 324-326, 331-334, 364, 401, 414, 496, 579 kHz

15.6. Sassoon Hospital Syndrome

This syndrome, a curious disorder attributed to the nephrotoxin of *Rhizopus nigricans*, is characterized by an epidemic, excessive polyuria and polydipsia, anorexia, weakness and fatigue. This phycomycetous black mold lives usually on millet grains and breads. In case of this syndrome the fungus can be found in the urinary system and/or in the bowel canal. Cerebral and pulmonary problems may but seldom come about.
Diagnosis: by finding the fungus in biopsy materials and cultures
Treatment: with amphotericin B and other effective fungicidal drugs.
The most frequent resonances are: 342-350, 368-369, 391, 427-429, 442-451, 507-509 kHz

15.7. Urinary Tract Infections in General

Urinary tract infections (UTIs) may be caused by viruses, bacteria, fungi and parasites.
Viral illnesses of the lower urinary tract caused by viruses (f.i. *BK virus, adenovirus* and *CMV*) are very uncommon and occur mostly among immunocompromised patients to cause hemorrhagic cystitis. *Herpes Simplex Virus-2 (HSV2)* often causes infections of the urethra and on the penis of men as well as of the vulva, the perineum, the buttocks, the cervix, or the vagina of women. Sexually transmitted other viral infections such as *HIV, HPV, CMV* may also infect the lower urinary tract, causing but seldom any symptoms, but being potencial co-factors of malformation.
Bacterial infections of the lower urinary tract are very common. Urinary tract infections are often caused by bacteria coming from the person's own intestines or reproductive organs. Bacterial urinary tract infections occur most often caused by *E. coli, Proteus, Streptococcus, Staphylococcus, Pseudomonas, Shigella, Neisseria gonorrhoeae, Chlamydia, Mycoplasma* species.

Fungal infections of the urinary tract are most often caused by Candida species. In case of dissemination, blastomycosis and coccidiomycosis can also affect the urinary tract. Fungi and bacteria often infect the kidneys simultaneously.
Parasitic infections: a number of parasites, including worms, can cause UTIs as well as genital infections. Malaria causes acute kidney failure. Trichomoniasis, a protozoan, sexually-transmitted disease is characterized by a copious, greenish-yellow, frothy discharge from the vagina. In men it usually causes inflammation of the prostates. Schistosomiasis, a worm infection can cause severe kidney failures and can affect the urethers and the bladder, too. Filariasis, a threadworm infection, obstructing the lymphatic vessels, can cause a lymphatic fluid accumulation in the urine.

15.8. Urethritis

Urethritis, an infection of the urethra can be caused by many a pathogen and, based on its cause, is sorted into two groups: i.e. gonorrheal and non-gonorrheal urethritis (NGU). The pathogens most often causing non gonorrheal urethritis are *Adenoviruses, Chlamydia trachomatis, Escherichia coli, Proteus, Herpes simplex virus, Mycoplasma genitalium* and *Trichomonas species*. Its symptoms include a frequent, urgent need to urinate and pain during urination. Pain during urination can occur also in case of vaginal infections, as the acidic urine passes over the inflamed labia.
Diagnosis of urethritis is usually apparent from the symptoms alone. Laboratory tests and bacteria cultures are used in order to confirm the diagnosis.
Treatment: by administering antibiotic/antifungal/antihelminthic drugs. Herpes simplex viral as well as other viral infections may be treated with an antiviral drugs, f.i. Acyclovir.
RFR method: is to be used concurrently with the conventional treatment to detect and eliminate the pathogen microorganisms; it is especially advantageous in case of viral and antibiotic-resistant bacterial infections.
The most frequent resonances are: 291-293, 307-308, 320-339, 342-350, 371, 379-383, 387, 391-394, 408-416 kHz

15.9. Cystitis

Cystitis is an infection of the bladder. Some women suffer from recurring bladder infections. Bacteria present in the vagina may get to the urethra and into the bladder as well. Women may get bladder infections following a sexual intercourse.
Bladder infections are less common in men, and generally begin with an infection of the urethra that ascends into the prostate and then into the bladder.
Bladder infection usually produces a frequent, urgent need to urinate and a burning or painful sensation during urination. Pain is usually felt above the pubic bone and often in the lower part of the back as well. Frequent urination does usually not belong to its symptoms. The pathogens are just the same as those regarding UTIs mentioned above.
Diagnosis: symptomatically and by urine bacterial count. The samples have to be cultured to identify the type of the bacteria.
Treatment: by administering antiviral, antibiotic, antifungal, and antihelminthic drugs.
RFR method: must be used concurrently with the conventional treatment in order to detect and eliminate the pathogen microorganisms; it is especially advantageous in case of viral or antibiotic-resistant bacterial infections.
The most frequent resonances are: 290-294, 307-308, 320-329, 332-352, 354-363, 370-374, 376, 396, 402, 410, 440-452, 476, 500-502 kHz

15.10. Prostatitis

Prostatitis is an inflammation of the prostate glands. It may result from bacterial, fungal, viral, protozoal and worm infections. The infection of the prostate causes pain in the groin,

in the area between the penis and the anus (perineum), and in the lower back; swelling, redness, chills and fever, and an extreme pain occurs, if this area is touched.
Diagnosis: symptomatically, the evidence can be supported with microbiological examinations.
Treatment: by administering antiviral, antibiotic, antifungal, antihelminthic drugs.
RFR method: must be used together with the conventional treatment in order to detect and eliminate the pathogen microorganisms; it is especially advantageous in case of viral or antibiotic-resistant bacterial infections.
The most frequent resonances are: 291-293, 307-308, 320-326, 331-335, 342-352, 355-363, 378-388, 402-410, 442-451 kHz

15.11. Orchitis and Epididymitis

Mumps is complicated by isolated orchitis in 20-35 percent regarding postpubertal males. Testicular involvements usually appear seven to ten days after the onset of parotitis, though the former may precede the latter or they may appear simultaneously. Occasionally, orchitis occurs even in the absence of parotitis. Orchitis is heralded by the recrudescence of malaise and the appearance of chilly sensations, headache, nausea and vomiting. Shaking chills and high fevers follow. The testicles become greatly swollen and acutely painful. The epididymis often becomes palpable, like a swollen, tender cord. Occasionally there may exist epididymitis without orchitis. Mumps orchitis is followed by progressive atrophy of the testicle in one-half of the cases. Though even after a bilateral orchitis sterility is unusual (provided that no significant atrophy came to pass), if a bilateral testicular atrophy has occured secondary to mumps, sterility or subnormal sperm counts are quite common.
Orchitis can only very seldom be caused by other viruses (f.i. *Coxsackie virus, ECHO virus, EBV* and *varicella* virus). In case of sexually active men orchitis caused by a bacterial infection (f.i. *E. coli, Klebsiella, Streptococcus, Staphylococcus, Pseudomonas, Neisseria gonorrhoeae, Chlamydia, Mycoplasma* species) occurs usually secondary, spreading from an epididymitis. Other bacterial infections can occur very rarely and only among immunocompromised patients caused by f.i. *Mycobacterium avium complex, Cryptococcus neoformans, Toxoplasma gondii, Haemophilus parainfluenzae and Candida albicans.*
Diagnosis: symptomatically. Laboratory tests and bacteria cultures are used in order to confirm the diagnosis.
RFR method: can detect and eliminate the mumps virus and also the other causative agents (see their special Chapters).

15.12. Pelvic Inflammatory Disease

Pelvic inflammatory disease is an inflammation of the uterus, the fallopian tubes and/or the ovaries and is caused by an infection, e.g. of candidal, chlamydial, trichomonal, viral, etc. origin. An inflammation of the fallopian tubes occurs mainly among sexually active women, mostly among those who use intrauterine devices (IUDs). Infections occur mostly via the vagina, getting into the uterus, and then into the fallopian tubes and the ovaries, and are acquired usually due to sexual intercourses. Less common causes of inflammations include *actinomycosis, schistosomiasis* and *tuberculosis,* that may cause this inflammatory disorder hematogenously. This type of infection can spread into the abdominal cavity, causing peritonitis.
Symptoms include pain in the lower abdomen, nausea and/or vomiting, a low fever, irregular bleeding and a scant vaginal discharge. Salpingitis may cause adhesions in the fallopian tubes. Abscesses may develop in the tubes, the ovaries and even in the pelvis. These infections may spread into the bloodstream causing sepsis, which can be fatal. A perforated abscess requires emergency surgery.

Diagnosis: symptomatically and by routine laboratory examinations, such as white blood cell counts, by bacteria culture, etc.
Treatment: in case of
Candidal infections: by administering f.i. miconazole, clotrimazole, butoconazole, terconazole, fluconazole and ketoconazole.
RFR support: in order to detect and eliminate the **Candida with resonant frequencies: 338, 383-389, 379 kHz.** (Regarding other candida frequencies, see Chapter 7.3.1.)
Bacterial infections: by administering f.i. metronidazole, clindamycin, ceftriaxone, doxycyclin, etc.
RFR support: should be used in accordance with the found causative bacteria (Regarding their frequencies, see Chapter 6.)
Actinomycosis infection: by administering f.i. doxycyclin, iv. penicillin, clindamycin etc.
RFR support: in order to detect and eliminate the pathogens with resonant frequencies: **372, 395-399, 402, 418-422 kHz**
Chlamydial infections: by administering f.i. doxycyclin, azitromycin, etc.
RFR support: happens with resonant frequencies **317-320, 379-383, 429, 566 kHz** (Regarding its other frequencies, see Chapter 6.13.1.2.)
Trichomonal infection: by administering f.i. metronidazole, tinidazole.
RFR support: in order to detect and eliminate the pathogens with resonant frequencies **312, 354, 378-385, 500-503 kHz**
Viral infections: in case of Herpes viral infections by administering acyclovir, etc.
RFR method: detects and may eliminate the viruses (see Chapter 5.)
The frequencies of HPV or genital warts are: 402-410 kHz
Parasitic infection, schistosomiasis: by administering Praziquantel, Oxamniquine, Metrifonate, etc.

15.13. Dysmenorrhea

Dysmenorrhea is an abdominal pain, caused by uterine cramps during the menstrual period. It is generally accepted that strong or abnormal uterine contractions are significant etiologic factors in the feeling of discomfort experienced by the patient. **Primary dysmenorrhea** is defined as menstrual pain not associated with macroscopic pelvic pathology. It typically occurs in the first few years after menarche and affects up to 50% of postpubescent females.
Its pathogenesis is influenced by prostaglandin F2alpha (PGF2alpha), a potent myometrial stimulant and vasoconstrictor, present in the secretory endometrium. The response to prostaglandin inhibitors in patients with dysmenorrhea supports the assertion that dysmenorrhea is prostaglandin mediated. Substantial evidences attribute dysmenorrhea to prolonged uterine contractions and decreased blood flow to the myometrium. The elevated prostaglandin levels in the endometrial fluid of women with dysmenorrhea correlate with the degree of their pain. Leukotrienes are known to heighten the sensitivity of the pain-fibers in the uterus., The posterior pituitary hormone vasopressin can also have a role in the myometrial hypersensitivity, in the reduced uterine blood flow, and in the pain felt.
Secondary dysmenorrhea is defined as a menstrual pain resulting from anatomic and/or macroscopic pelvic pathology, f.i. as in case of women with endometriosis or chronic pelvic inflammatory diseasee.
It can be caused by several different factors, such as viral, bacterial and fungal infections, endometriosis, pelvic inflammatory disease, ovarian cysts, adenomyosis, tumors, uterine myomas, uterine polyps, inflamed intrauterine adhesions, various connatal uterus damages, intrauterine contraceptive devices, Allen-Masters syndrome, Pelvic congestion syndrome and others.
Dysmenorrhea can disrupt a person's life, is a significant public health problem associated with a substantial economic loss related to work absences. The patient's history is

important in establishing the diagnosis of dysmenorrhea and should include an assessment of the onset, duration, type, and severity of her pain. A thorough menstrual history is also essential and should include the patient's age at the time of her menarche, data concerning her cycle regularity, cycle length, last menstrual period and duration and the amount of her menstrual flow as well as the factors exacerbating or ameliorating the symptoms.
Symptoms associated with dysmenorrhea are usually nausea, headache, vomiting, bloating, diarrhea, an urge to urinate frequently, fatigue, depression and severe cramps. In extreme cases, dizziness, nervousness, hysterical reactions may also occur. One of the most common causes of secondary dysmenorrhea is endometriosis. (See infections of endometriosis in Chapter 24.9.)
Diagnosis: symptomatically, by laboratory examinations and ultrasound.
Treatment: symptomatically, and depending on its cause, f.i. by administering low dose oral contraceptives containing estrogen, progestin, or long-acting medroxyprogesterone, or antibiotics etc.
RFR method: detects and can eliminate all pathogen microorganisms.
The most frequent resonances are: 307, 337, 364, 396, 406-410-411, 440-451 kHz
The most frequent resonances of ovarial cysts are: 289-293, 363-370 kHz
The most frequent other infections associated with secondary dysmenorrhea can be caused by: Gonococci, Staphylococci, Sreptococci, Pseudomonas, Klebsiella, E. coli, Proteus species, as well as by Candida species and Trichomonas vaginalis.
The most frequent other viral infections can be caused by: Coxsackie viruses A1, 2, 5, 8, 9, 16 and B1-5, CMV, HTLV and HSV1 and 2.
The most frequent resonances of myoma uteri are: 370-376, 418-426, 442-451, 459-464, 516-521 kHz
The most frequent resonances of adenomyosis are: 426-434, 442-444 kHz
The most frequent resonances of womb polyps are: 318-319, 332-340, 343-348, 352-354, 367-368, 402-409, 476-479, 513, 534-538, 543-545, 552-555 kHz
The most frequent resonances of colpoxerosis are: 291-293, 344-350, 352-363, 384, 402, 443-450, 572-586 kHz
See the other mentioned frequencies of bacteria, microparasites, fungi and viruses in their special Chapters.

15.14. Infertility Caused by Infections

Fertility is defined as the capacity to reproduce and the state of being fertile. This term should be differentiated from fecundability, meaning the probability of achieving pregnancy each month, and fecundity, which is the ability to achieve a live birth within one menstrual cycle.
The most accepted definition of the term **infertility** is the lack of pregnancy (regardless of its cause) within and after 1 year of unprotected intercourse. Infertility affects approximately 15% of couples of reproductive age. Its prevalence remained stable during the past 50 years, though a change in its etiology and in the age of the patients has occurred. Infectious infertility can be caused by different viral, bacterial and rarely by fungal infections in the tract of the reproductive organs, all impeding normal fertility.
The reproductive process requires the interaction and integrity of the female and male reproductive tracts, allowing the release of a normal preovulatory oocyte, the production of adequate spermatozoa and the normal transport of the gametes to the ampullary portion of the fallopian tubes, where the fertilization will occur, as well as the subsequent transport of the cleaving embryo up to the endometrial cavity, where its normal implantation and further development will happen.
The etiologic factors of an increased risk of infertility include pelvic infectious inflammatory diseases (PID); endometriosis (caused by a coinfection with *ECHO* and *HPVs* with *Mycoplasma genitalium* or *Mycoplasma fermentans*); as well as environmental

and occupational factors; the toxic effects of tobacco, marijuana and other drugs, exercise, inadequate diet leading to extreme weight loss or gain, as well as advanced age.

Pelvic inflammatory diseases were caused by *Neisseria gonorrhoeae* and other bacterial and viral infections for more than a century. While gonorrhoea still plays an important role in causing tubal diseases, it is surpassed by infections of *Chlamydia* and *Mycoplasma*. In many instances, the patient never recalls having had an acute PID episode; but years later, the incidental finding of tubal obstruction detected by hysterosalpingogram (HSG) or by laparoscopy may be the only indication of having had previously a PID.

Endometriosis (see also Chapter 16.12. and 24.9.) is an enigmatic pathologic disease affecting women in their reproductive years. Its incidence increases with the patient's age and often affects women of the middle and high socioeconomic classes. Though the associated gene-defect of endometriosis has not yet been identified, its genetic link seems probably based on observed chromosomal defects in the endometriotic tissue and on the observed 7-fold increased risk of endometriosis regarding patients with a family history of the disease. The lesions of endometriosis vary from microscopic to macroscopic ones. Classic endometriosis appears as black pigments affecting the surface of the peritoneum of the bladder, the oyary, the fallopian tubes, the cul-de-sac and the bowels. Nonclassic endometriosis appears as nonpigmental, i.e. red, tan and white lesions or vesicles. Minimal and mild infectious endometriosis may reduce the fertility.

Endometriosis can be associated with ovulatory disorders, such as luteal phase deficiency (LPD), oligo-ovulation and luteinized unruptured follicle (LUF) syndrome.

The role of many other factors (excessive heat exposures, microwave radiation, ultrasonography and other health hazards) **in inducing infertility** is doubtful. Excessive radiation damages the germinal cells. Exposure to lead, or other heavy metals and pesticides may also be associated with male infertility.

Smoking is associated with the infertility of males as well as females. Marijuana and its metabolite, delta-9-tetrahydrocannabino, inhibit the secretion of the luteinizing (LH) and follicle-stimulating hormones (FSH), inducing thus ovulatory disorders and LPD in women. The use of Marijuana affects males by decreasing the sperm-count and lowering the quality of the sperm.

Chronic alcoholism is related to ovulatory disorders, interfering therefore with fertility. Alcohol abuse by males interferes with the synthesis of testosterone and has also an impact on the sperm concentration. Alcoholism may delay the sexual response and may lead to impotence.

Obesity has an impact on infertility only if the female patient's weight is extremely high. Loss of weight associated with **anorexia nervosa** or **bulimia** induces hypothalamic amenorrhea, a low FSH level and low LH secretion, while weight gain is less harmful. The disruption of the hypothalamic-pituitary-ovarian axis is affected if associated with other endocrine disorders such as in case of polycystic ovarian disease (PCOD with **E vitamine deficit**), adrenal hyperplasia and hypothyroidism.

Aging does also influence the fertility of men, it decreases the testosterone level, increases the gonadotropin level, the sperm concentration, changes the semen volume, and decreases the libido. Moreover, the incidence of birth defects increases. Concerning the history of previous infertility evaluation and treatment, specific questions should be put about the frequency of intercourse, use of lubricants (eg, K-Y gel) that could be spermicidal, use of vaginal douches after intercourse and the presence of any **sexual dysfunction** such as anorgasmia and dyspareunia.

Female patients should be questioned about their menstrual frequency and patterns beginning from their menarche. A history of weight changes, hirsutism, frontal balding and acne should also be questioned.

Ask male patients about their previous spermiogram results, their history of impotence, premature ejaculation, change in libido, testicular trauma, previous relationships, any

previous pregnancy of their partners and the existence of offsprings got from previous partners.
Thyroid gland should be carefully examined in order to exclude gland enlargements and thyroid nodules.
By inspecting the vaginal mucosa one may find signs of the deficiency of estrogens or the presence of an infection.
The evaluation of the cervix should include a Papanicolaou test (Pap smear) and culturing for *N. gonorrhoeae, Chlamydia and Ureaplasma urealyticum*, as well as testing for *ECHO and Human Papilloma Viruses*.
Bimanual examinations should be performed to establish the direction of the cervix, the size and position of the uterus in order to exclude the presence of uterine fibroids, adnexal masses, tenderness, pelvic nodules, indicative of infection or endometriosis.
Gynecologists should perform a pelvic ultrasonographic scan. By this the physician is able to establish an early diagnosis of adnexal masses; to determine the size and aspect of the ovaries; to detect the presence of endometrial polyps, human papilloma viral infections, submucous fibroids and hydrosalpinx.
The size of the testis, the presence of an urethral stenosis or a varicocele must also be determined.
The history of a previous inguinal hernia repair can indicate an accidental ligation of the spermatic artery.
Diagnostic tests should progress from the most simple (eg. postcoital test [PCT], endometrial biopsy) to the more complex ones, or to those implying a major risk for the patients (eg, laparoscopy). The couple will be stressed by their need to seek medical intervention; so that in order to relieve their anxiety, one must emphasize that a complete infertility evaluation has to be performed according to the woman's menstrual cycle and that it may take 2 menstrual cycles before the factor(s) causing the infertility problem is found.
The male partner must give a semen sample for a comprehensive semen analysis. Previous paternity does not guarantee that his reproductive system has not been affected since the birth of his previous offspring. The comprehensive semen analysis must be performed in a certified andrology laboratory, and the semen sample should be collected preferably at the same andrology laboratory that will conduct the test.
Specific biochemistry analyses relevant to the accessory sex gland functions can be performed using the semen sample. These include fructose from the seminal vesicles, zinc and acid phosphatase from the prostate gland, alpha-glucosidases and carnitine from the epididymis.
Sperm agglutination indirectly indicates the presence of **sperm antibodies**. The immunobead test can be performed either directly on the sperm or indirectly on the sperm and blood. Surface antibodies against immunoglobulin A (IgA) and immunoglobulin G (IgG) may be present.
Several congenital and acquired conditions can affect the female reproductive function. These conditions alter either the anatomy or the normal physiology of reproduction and may impair the transport of the gametes or embryo(s) and/or interfere with the implantation and the embryo/fetal development.

Congenital defects

The normal development of the müllerian ducts accounts for the normal anatomic configuration of the uterus, the fallopian tubes, cervix and the upper vagina. The full spectrum of congenital/müllerian abnormalities varies from the total absence of the uterus and vagina (**Rokitansky-Küster-Hauser syndrome**) to minor defects such as arcuate uterus and vaginal septa (transversal or longitudinal).
LPD is associated with ovulatory dysfunction related to the following 3 factors: The first one is, if at the time of the ovulation, the size of the follicle is small. The second one is if

the preovulatory LH level is high enough to induce resumption of myosis of the oocyte, but might not be enough to induce a follicular rupture and a normal corpus luteum function. The third factor is the normal development of the follicle size.
The fallopian tubes play an important role in the reproduction. After ovulation, the fimbriae pick up the oocyte from the peritoneal fluid accumulated in the cul-de-sac. The epithelial cilia transport the oocyte up to the ampulla. The capacitated spermatozoa are transported from the endometrium through the cornual section and advance through the fallopian tube down into the ampulla, where the fertilization occurs. The embryo initiates its early cleaving stages and is propelled upward to arrive at the endometrial cavity its blastocyst stage (96-120 hours after ovulation).
Abnormalities or damages of the fallopian tube interfere with fertility and are responsible for an abnormal implantation.
PID, peritoneal adhesions secondary to a previous pelvic surgery, infectious inflammation, endometriosis and ovarian cyst ruptures all compromise the motility in the fallopian tubes or block the fimbriae and develop hydrosalpinx. Large myomas, pelvic masses, and the blockage of the cul-de-sac interfere with the accumulation of the peritoneal fluid as well as with the normal oocyte pickup mechanism. Periovarian adhesions, encapsulating the ovary, interfere with the normal oocyte release at the time of ovulation, becoming thus a mechanical factor for infertility.
Oogenesis occurs in the ovary from the first trimester of embryonic life and is completed in the 28-30th week of gestation. By then, approximately 7 million oocytes are present. They are arrested at the prophase stage of the first meiosis division. Subsequently, the number of oocytes decreases due to the continuous process of atresia. At birth, the pool of oocytes is reduced to approximately 2 million. At the time of menarche, approximately 500,000 oocytes are present. These oocytes are used throughout the reproductive years until the menopause.
The ovulatory process is initiated when the hypothalamus-pituitary-ovarian axis matures and under the regulation of the gonadotropin-releasing hormone (GnRH) the FSH and LH acquire their normal secretory patterns. Ovulatory dysfunction is defined as an alteration in the frequency and duration of the menstrual cycle. A normal menstrual cycle lasts for 25-35 days, in average 28 days. Failure to ovulate is the most common ovulatory problem. Absence of ovulation can be caused by primary amenorrhea or premature menopause, indicating the depletion of oocytes or the absence of the ovaries.
A Primary amenorrhea occurs if at the age of 16 years, or if after 3 years of pubarche and telarche the spontaneous menstrual period is absent. A primary amenorrhea is generally related to the failures of the gonadal development, such as in case of Turner syndrome, in which the karyotype 45,X indicates an absence of the X chromosome. These patients present with sexual infantilism associated with short stature, webbed neck and cubitus valgus. Other chromosome abnormalities include 46,XX, which is associated with partial deletions of the short or long arm of one of the X chromosomes, as well as mosaicism (eg, X/XXX; X/XX/XXX; pure gonadal dysgenesis; 46,XX; 46,XY).
Oligomenorrhea is characterized by the dysfunction of the hypothalamus-pituitary-ovarian axis and is the most common ovulatory disorder associated with infertility. Patients with this disorder present a history of irregular menstrual cycles fluctuating from 35 days to 2-5 months, sometimes associated with a history of dysfunctional uterine bleeding or prolonged periods of breakthrough bleeding. These patients often have symptoms of hyperandrogenism, acne, hirsutism, immune dysfunction and baldness. Obesity is frequently associated, aggravating the prognosis of this disorder. Though these patients are not sterile, their fertility is nevertheless decreased, and their obstetrical outcome is poor because of an increased history of pregnancy losses.
The treatment of infertility depends on the state of the present pathological process.

An abnormal PCT-(postcoital test) result, attributable to chronic cervicitis, may be treated with antibiotics. The reduced secretion of cervical mucus due to the destruction of the endocervical glands by previous cervical conization, freezing, or laser vaporization, responds but poorly to low-dose estrogen therapy.
If necessary, endometrial polyps can be removed by operative hysteroscopy associated with dilatation and curettage, the virus infection will nevertheless remain.
Small and asymptomatic myomas do generally not require any treatment, though the patient should be periodically monitored. If the fibroids are associated with hypermenorrhea or menometrorrhagia or if they cause infertility, they have to be treated. Three modalities are used to treat myomas: medical treatment, surgical treatment and embolization. Surgical treatment of myomas is indicated in case of hypermenorrhea, menometrorrhagia and if the myoma is implicated in recurrent miscarriages, or interferes with embryo implantation.
Endometriosis treatment depends on the severity of the disease and the patient's need. Four alternatives are currently in use to treat endometriosis: expectant therapy, surgical intervention, medical treatment and combined therapy.
Infectious inhibitors of fertility
Toxoplasmosis is not responsible for miscarriage during its acute episodes. Nevertheless, spontaneous abortion has been described in pregnant patients with seroconversion of *Toxoplasma gondii* antibody. Chronic toxoplasmosis is not associated with habitual abortion.
Listeria monocytogenes is responsible for recurrent abortions in cattle and is an established cause of human pregnancy loss during its second and third trimester. The pregnancy loss results from the hematogenous spread of enterically acquired infection caused by consuming unpasteurized dairy products. Listerial colonization of the genital tract is unlikely, many studies have failed to demonstrate a chronic infection.
Ureoplasma urealyticum plays a role in miscarriages of animals, but its role is controversial concerning people. According to many reports, *U. urealyticum* has been cultured from the endocervix and from the products of conception. Patients with positive culture findings have usually fever after miscarriage. Even lacking information, it is a customary practice to treat patients prophylactically with doxycycline.
Cytomegalovirus
Although CMV has been isolated from placentas and fetuses, most of its pathology is associated with premature rupture of membranes, preterm delivery, and neonatal infections.
The most frequent and most important infectious inhibitor factors are *HPV* and *Mycoplasmas*.
Infections caused by ECHO viruses and Human Papilloma Viruses can not be treated in a conventional medicinal way.
Mycoplasma genitalium or/and M. fermentans can be treated with certain macrolids, though it is less effective.
The RFR method detects and may eliminate all the pathogen microorganisms.
In most cases there can be found *Mycoplasma genitalium* or/and *M. fermentans* and *Human Papilloma Viruses* (HPVs).
The most frequent resonances of Mycoplasma genitalium are: 307-308, 342-350 kHz
The most frequent resonances of Mycoplasma fermentans are: 442-451, 493-495 kHz
The most frequent resonances of HPVs are: 427-438 kHz
The most frequent resonances of ECHO viruses are: 317-319, 369, 397-405, 471-473, 526 kHz
As to the resonances of other, rarely found pathogen microorganisms see their special Chapters.

16. SEXUALLY TRANSMITTED DISEASES

Sexually transmitted diseases are infections that are often, if not always, passed from person to person through their sexual contact. Transmission of certain sexually transmitted diseases does not require genital penetration. Although a sexually transmitted infection usually results from having oral, vaginal, or anal sexual intercourse with an infected partner, occasionally it may be transmitted even by kissing or any other close body contact. Traditionally, five diseases are classified to be sexually transmitted, such as syphilis, gonorrhea, chancroid, lymphogranuloma venereum and granuloma inguinale. However, many other diseases are sexually transmitted, too, including genital herpes, Hepatitis B infection, molluscum contagiosum, HIV infection, chlamydial cervicitis, pediculosis pubis, genital candidiasis, genital warts, trichomonas infection, amebiasis, campylobacteriosis, EBV and CMV infection, giardiasis, salmonellosis, shigellosis, etc.

16.1. Syphilis

Syphilis is a chronic, in a venereal way acquired systemic human infection caused by *Treponema pallidum pallidum.* This spirochete bacterium is usually transmitted sexually, entering the body through the mucous membranes of f.i. the vagina or the mouth, or even through an injury of the skin. Within hours, the bacterium can reach the lymph nodes nearby, spreading then in the whole body via the lymphatic and blood system. Syphilis may also infect the fetus during pregnancy, causing birth defects and other problems.

The illness is characterized by an incubation period of three weeks; followed by a primary lesion associated with regional lymphadenopathy, a secondary bacteremic stage associated with generalized mucocutaneous lesions and generalized lymphadenopathy; a latent period of subclinical infection lasting for many years. In 60 percent of all untreated cases there will develop a tertiary stage characterized by progressive destructive mucocutaneous, musculoskeletal and parenchymal lesions, aortitis and central nervous system symptoms. Uncommon ways of transmission include non-sexual personal contact, or infection in utero or due to transfusing infected blood. Atypical primary lesions may occur. The most frequent genital lesions that must be distinguished from the primary syphilis include traumatic and superinfected lesions, genital lesions of Herpes progenitalis (caused by HSV2) and chancroid lesions. Secondary lesions are disseminated, occuring also on the palms, soles, face and scalp. The tiny papular, follicular syphilides involving the hair follicles may lead to patchy alopecia and to the loss of eyebrows or beard. On warm, moist, intertriginous loci of the body, including the perianal region, vulva, scrotum, inner thighs, axillas and the skin under the breasts, the syphilitic papules usually get enlarged and become eroded producing broad, moist, pink or gray-white, highly infectious lesions called condyloma latum. Superficial mucosal erosions occur among about a third of the affected patients, may involve the lips, the oral mucosa, the tongue, palate, pharynx, vulva, vagina, glans penis, or the inner part of the prepuce.

Syphilitic hepatitis is characterized by an unusually high serum alkaline phosphatase level and by a nonspecific histologic appearance which is unlike the viral hepatitis, and shows moderate inflammations with polymorphonuclear leucocytes and lymphocytes, some hepatocellular damages and no cholestasis. Renal involvement may occur and be associated with proteinuria, acute nephritic syndrome, or, rarely, a hemorrhagic glomerulonephritis, too.

The late stage of syphilis is characterized by the slowly progressing inflammatory damages of the aorta and of the central nervous system, starting during the latent stage of the illness. Meningovascular syphilis is associated with the inflammation of the pia mater and the arachnoid area, going together with the symptoms of a focal or a widespread cerebrovascular disease, causing often only reflex changes. In case of widespread

parenchymal damages a general paresis may develop, including abnormalities corresponding to this paresis, f.i. hyperactive reflexes, Argill-Robertson pupils of the eye, etc. In the late stage of syphilis, patients often suffer change in their personality, affectivity, intellect and speech, can have illusions, delusions, hallucinations of the sensorium, memoria disturbances, etc. In case of tabes dorsalis, demyelinization of the posterior columns, the dorsal roots and dorsal root ganglia will occur, causing ataxia, paresthesia, bladder disturbances, impotency, areflexia, deep pain, temperature sensations and joint degeneration as well. Lues may persist for decades, may cause heart damages, certain other forms of brain damages and can even lead to death.

Diagnosis: symptomatically, by laboratory tests: VDRL, RPR and FTA-ABS.

Treatment: by administering intramuscularly given retard penicillin preparates, using protocols for its definitive healing. A long lasting and more frequently given intravenous treatment may be needed. People allergic to penicillin can receive Doxycyclin orally for 3-4 weeks, their partners have to be screened with antibody tests and should also be treated if their tests are positive.

The most frequent resonances are: 307, 319, 332-338, 340-350, 360 kHz

RFR method: should not be used for diagnosing. The RFR method should only be used after beginning the treatment with antibiotics.

16.2. Gonorrhea

Gonorrhea is a sexually transmitted disease caused by the bacterium *Neisseria gonorrhoeae*, which can infect the inner lining of the urethra, the cervix, the anus, the throat, or the conjunctivae. Local complications of the illness may be bartholinitis, salpingitis, adnexitis, endometritis and peritonitis in case of women; phymosis, balanitis, periurethral abscess, prostatitis, epididymitis and sometimes even cystitis in case of men. Gonococcemia, i.e. the dissemination of the infection via the blood can cause arthritis, tenditis, erythema multiforme, erythema nodosum, keratosis gonorrhoica on the skin of the feet and the upper part of the hands, endocarditis, pericarditis, meningitis and can cause sepsis. The characteristic epidemiology of this disease is that gonorrhea is usually spread by carriers having no symptoms or ignored symptoms. Patients with symptomatic gonorrhea should always be interviewed to identify all their recent sexual partners, who, if proved to be infected, should be examined and treated.

Symptoms: The first leading symptom of the illness is a copious fluor accompanied by itchy, burning sensations. The infection, ascending the genital tract, causes pain during the sexual intercourse and pain of the affected areas (f.i. testis, prostatae, etc.). A deep pelvic pain, dysuria, frequent urination and reproductive problems (aspermia, sterility) can also be caused. The onset of gonococcemia is often characterized by fever, polyarthralgia and by papulous, petechial, pustular, or hemorrhagic skin lesions. Its spreading into the upper abdomen may lead to gonococcal perihepatitis causing right upper quadrant or bilateral upper abdominal pain and tenderness, occasionally with a hepatic friction rub named Fitz-Hugh-Curtis syndrome. Gonorrheal infection of children occurs mostly during their birth causing ophthalmoblenorrhoea, characterized by a greenish-yellowish discharge together with edema of the eyelids and, if untreated, panophthalmia and blindness might also develop.

Diagnosis: by microscopic and immunofluorescent staining examinations and bacterial culturing.

Differential diagnosis: by distinguishing it from other infections caused f.i. by Trichomonas vaginalis, Candida species, Chlamydia trachomatis, HSV2, etc.

Treatment: by administering effective antibiotica (f.i. im. ceftriaxone, other third generation cephalosporines, im spectinomycine, etc. Chlamydia infections are often associated with gonorrhea, are difficult to diagnose, patients can be treated by administering a week-long course of giving Doxycyclin together with post treatment

examinations. There are regional differences in the antibiotic resistance of *Neisseria gonorrhoeae*.
RFR method: is advised to be used in complicated cases and only after treatment with antibiotics.
The resonant frequencies of Neisseria are: 330-340, 364-367, 370 kHz
The resonant frequencies of Chlamydia are: 317-320, 370-386, 429, 444, 482, 566 kHz

16.3. Chancroid

Chancroid, caused by *Hemophilus ducreyi* bacteria, is an acute infection characterized by painful genital ulcerations usually associated with inflammatory, often suppurative, inguinal adenopathy. A presumptive diagnosis is supported by excluding syphilis, genital herpes and other specific causes of genital ulcerations, together with improvement following therapy.
Symptoms include small, painful blisters on the genitals and around the anus, which rapidly rupture to form shallow ulcers. These sores may get enlarged and be joined together. The lymph nodes in the groin may become tender, enlarged, and matted, forming an abscess. The skin over the abscess may become red and shiny and break up, so that pus will discharge.
Diagnosis of chancroid is based on its appearance and on the result of tests applied for diagnosing other diseases causing ulcer. A specific diagnosis is proved only if Hemophilus ducreyi is isolated from the lesion or the suppurative node.
Differential diagnosis: HSV, syphilis, and other sexually transmitted diseases.
Treatment: by administering ceftriaxone, erythromycin, etc.
RFR method: is to be used after treatment with antibiotics.
Its resonant frequencies are: 330-340 kHz
This list is not complete; as there are still other species with different frequencies.

16.4. Lymphogranuloma Venereum

Lymphogranuloma venereum is a sexually transmitted infection caused by invasive *serovars (L1-3) of Chlamydia trachomatis*, different from those causing inflammation of the urethra and cervix. The acute disease is characterized by a transient primary genital lesion followed by multilocular suppurative regional lymphadenopathy. Homosexual men may develop hemorrhagic proctocolitis with regional lymphadenitis. Acute lymphogranuloma venereum is almost always associated with nonspecific systemic symptoms, f.i. with fever and leukocytosis, and sometimes with systemic complications such as meningoencephalitis as well. After a latent period of years, late complications include genital elephantiasis, strictures and fistulas of the penis, urethra and rectum.
Symptoms: begin with a painless, fluid-filled blister, that usually develops on the penis or in the vagina. This blister typically ulcerates. Lymph nodes in the groin on one or both sides may become enlarged and tender. The skin covering the infected area becomes warm and red. Fever, a feeling of illness, headache, joint pain, poor appetite and vomiting can also come to pass. Due to prolonged or repeated episodes, the lymphatic vessels may become obstructed, causing lymphatic edema in the tissues. Rectal infection may cause scarring, causing narrowing of the rectum.
Diagnosis can be confirmed by blood tests identifying antibodies against these Chlamydia serovars.
Treatment: by administering Doxycycline for three weeks.
Its resonant frequencies are: 317-320, 370-386, 429, 440-444, 481-482, 561 kHz

16.5. Granuloma Inguinale

Granuloma inguinale is a sexually transmitted infection caused by the bacterium *Calymmatobacterium granulomatis* that leads to chronic inflammation of the genitals. Granuloma inguinale is a mildly contagious, chronic, indolent, progressive, autoinoculable, ulcerative disease involving the skin and lymphatics of the genital or perianal areas. In the infected tissues the disease is associated with the presence of an intracellular microorganism and is identified morphologically as the Donovan body. Donovan bodies have been recovered from lesions and pseudobuboes of granuloma inguinale.

Diagnosis: by microscopic examinations of specimens from the edge of the lumps, at which the presence of Donovan bodies can confirm the diagnosis.

Treatment: by administering Doxycyclin, Erythromycin, etc.

RFR method: to be used solely after antibiotic treatment, detects and eliminates the bacteria.

Its most frequent resonances are: 392-428 kHz

These frequencies do not represent the entire range of frequencies for Calymmatobacteria.

16.6 Trichomoniasis

Trichomoniasis is a sexually transmitted disease of the urethra and the vagina caused by *Trichomonas vaginalis*, a single-celled parasite with a whip-like tail. Of the many members of the genus Trichomonas, three are parasites of human beings: Trichomonas hominis in the intestines, Trichomonas tenax in the oral cavity, and Trichomonas vaginalis, the only one capable to cause disease in the vagina, urethra and prostate. All three exist only in the trophozoite stage and resemble each other morphologically. Trichomonas vaginalis can infect the genitourinary tract of either men or women, though symptoms are more common as regards women.

Symptoms: The disease usually starts with a greenish-yellow, frothy vaginal discharge accompanied by itching and burning. In severe cases, the vulva and its surrounding skin may become inflamed and the labia swollen. Pain when urinating and frequent urination may occur, resembling the symptoms of a bladder infection. Prostate and urethra are usually affected by this infection concerning men. Acute purulent urethritis may occur.

Diagnosis: by examining the urine with culturing and microscopically, as well as by serologic tests.

Treatment: by administering metronidazole or tinidazole.

The most frequent resonances are: 312, 321, 354, 375-388, 501 kHz

16.7. Genital Candidiasis

Genital candidiasis can be a yeast infection of the vagina or the penis caused by *Candida albicans*. Candida infections are usually transmitted sexually, but Candida normally resides on the skin and the intestines, and can spread from those areas to the genitals. Candidiasis is more common among pregnant or menstruating women, diabetics, and patients with a decreased immune function. The vulva may be reddish and swollen. The skin may be raw and may crack. The vaginal wall is usually covered with a white, cheese-like fungal material, though it may look also normal. Men often have no symptoms, but the end of the penis and the foreskin may be sore and irritated, especially after sexual intercourse.

Diagnosis: by Candida culturing

Treatment: by administering f.i. Clotrimazole, Miconazole, Butoconazole, Tioconazole, Tetraconazole, Ketoconazole, Fluconazole and Itraconazole.

Candida may be resistant to antifungal drugs.

RFR method: is to be used only after antifungal treatment. RFR method may stop the drug resistance of the Candida.

The most frequent resonances are: 297, 338, 345, 352-362, 372, 380-390, 403, 410, 420-425, 443, 448-454, 460, 474, 488, 504, 520-526, 570-580 kHz

16.8. Genital Infections Caused by Herpes Viruses

Genital herpes is a sexually transmitted disease of the genital area, the skin around the rectum and the adjacent areas caused by *Herpes Simplex Viruses* having two types i.e. *HSV1* and *HSV2*, which both can infect the genitals, the skin around the rectum and also other parts of the body (see Chapter 5.2.4.1.). Other herpesviruses, f.i. the *Epstein-Barr Virus* can also be transmitted sexually thus causing mononucleosis infectiosa (see Chapter 5.2.4.3.1.).

RFR method: detects and may eliminate these viruses.

Regarding the frequencies, see their special Chapters.

16.9. Genital Warts and Other Wart Infections

Genital warts (Condyloma acuminatum), are warts in or around the vagina, the penis, or rectum caused by sexually transmitted *Human Papilloma Viruses*. Genital warts are common and being unsightly cause concern, may become infected with bacteria, and may indicate an impaired immune system. There are many Human Papilloma Viruses and other wart viruses that cause infections; nowadays more then 160 types are known. These types of papilloma viruses may cause cervical intraepithelial neoplasms or cancer of the vagina, vulva, anus, penis, prostate, mouth, throat, esophagus, and of other places as well. The warts grow rapidly in case of pregnancy and people with an impaired immune system, AIDS, or those having undergone immunosuppressive treatment. Certain papilloma viruses can cause cancer over a prolonged period of time, e.g. getting malignant within 20-30 years.

Treatment: by surgery, cryosurgery, laser surgery, etc.

RFR method: detects and may eliminate the wart virus.

The most frequent resonances are: 324, 343-345, 389-392, 400-410, 418-422, 426-450, 460-470, 476, 564 kHz

This list is not complete, there are still other species with different frequencies.

There is a sure evidence indicating an important relationship between wart viruses and human tumor genesis.

16.10. Genital Chlamydia and Ureaplasma Urealyticum Infection

Genital chlamydia is a sexually transmitted disease of the genital area, the skin around the rectum, or adjacent areas caused by *Chlamydia trachomatis* and *Chlamydia pneumoniae*. Both Chlamydia types can infect the genitals, the skin around the rectum, the infection may spread to other parts of the body, including the heart, the blood vessels and the joints. An intense synovitis can be caused by this intracellular pathogen, and even a chronic inflammation can be present (reactive arthritis) the culture-positive phase well beyond.

Symptoms: As to women, the infection causes at first vulvovaginitis accompanied by an abnormal vaginal discharge. (A discharge is considered to be abnormal if it occurs in large amounts, has an offensive odor, or is accompanied by vaginal itching, soreness, or pain.) This chlamydial infection can eventually spread to the pelvis, causing a pelvic inflammatory disease and/or salpingitis. Salpingitis, an inflammation of the fallopian tubes usually develops several weeks after being chlamydia infected. Women using intrauterine devices are especially at risk. These infections rarely occur before the first menstrual period, after menopause, or during pregnancy. They are most commonly acquired by sexual intercourse.

As to men the disease causes a minor inflammation in the penis and eventually a chronic prostatitis. Infection of the prostate causes pain in the groin, the area between the penis and

the anus and the lower back, and may be accompanied by chills or low fever. Patients may need to urinate frequently and urgently, blood may appear in their urine. The Chlamydia infection may spread to the scrotum, causing intense discomfort, swelling, redness, and extreme pain when the area is touched. Eventually the infection spreads to other organs if left untreated.

Most of the remaining causes of urethritis not mentioned above, are caused by *Ureaplasma urealyticum*, a mycoplasma-like bacterium. Ureaplasmas are very small bacteria lacking a rigid cell wall but which can reproduce themselves outside of the host's cells. Ureaplasma infections are not diagnosed specifically in routine medical settings, its culturing being difficult and other techniques for diagnosis are expensive. Thus, the diagnosis of Ureoplasma infections often are presumed on the basis of their characteristic symptoms and the negative results of other urethritis testings.

Diagnosis: symptomatically and by bacterial culturing, serology, etc.

Treatment: by administering Doxycyclin or Azithromycin. (Though certain species of the Chlamydia group are resistant to most antibiotics).

RFR method: detects and eliminates the Chlamydia.

The most frequent resonances are: 316-319, 374-390, 429, 440-444, 480-482, 566-571 kHz

16.11. Genital Mycoplasma Infections

Genital mycoplasma infection is a sexually transmitted disease caused by *Mycoplama genitalium* infecting the inner lining of the urethra, the cervix, and/or the rectum and can possibly spread to other organs as well. Some patients feel weak and tired even after several weeks after being treated. Mycoplasma genitalium infections can cause immunodeficiency disorders. These mean diverse conditions in case of which the immune system does not function adequately, and thus infections are more common, recur more frequently, are unusually severe, and last longer than usual. These deficiencies of the immune system can also lead to rarely occuring cancers and to unusual viral, bacterial and fungal infections.

Diagnosis: by laboratory tests, serology.

Treatment: many of these Mycoplasma species are antibiotic-resistant. By administering Doxycyclin, Azithromycin, Clarithromycin, Erythromycin, Troleandomycin, etc.

RFR method: has to be used after antibiotic treatment, detects and eliminates the Mycoplasma, the eradication of which is very difficult.

The most frequent resonances are: 307-308, 316-319, 342-350, 374-390, 429, 440-444, 480-482, 566-571 kHz

16.12. Endometriosis

Endometriosis means the presence of functioning ectopic endometrial glands and stroma outside of the uterine cavity. It can occur in various pelvic areas or at distant loci, such as the vagina, cervix, abdominal wall, arms, legs, pleura, lungs, diaphragm, kidneys, spleen, gallbladder, nasal mucous membranes, spinal canal, stomach and breasts. Pelvic endometriosis occurs typically among women aged 25-30, while extrapelvic manifestations of this disorder occur among women between 35-40. As endometriosis is an estrogen-dependent disease, it usually affects women of reproductive age.

Endometriosis is often associated with pelvic pain and infertility, but it can be asymptomatic as well. This gynecologic disorder is frequently encountered among outpatients. Its exact cause and pathogenesis is yet unclear. Four main theories exist trying to explain this disease.

Previous theories suggest that endometriosis results from the transport of viable endometrial cells through retrograde menstruation. Retrograde menstruation, however, is a

common physiologic event during which viable endometrial cells are shed into the peritoneal cavity.
Another theory suggests that transtubal dissemination is the most common route of dissemination, though also other routes have been observed, including the lymphatic and vascular channels.
Iatrogenic deposition of endometrial tissue has been found in some cases following gynecologic procedures and Caesarean sections.
The fourth theory suggests that endometriosis may be caused by combined infections by *ECHO virus*, or by one of the *Human Papilloma Viruses*, and by a *Human T-cell Lymphotropic Virus*. ECHO viruses are RNA viruses of the genus Enterovirus and the family Picornaviridae. The papilloma virus may be the most important causal factor. Frequently, other viral and bacterial infections are associated with these primary infections, which can often be passed from person to person through sexual contact. Men, however, are not characteristically affected by this disease, as its manifestation is estrogen-dependent. If developed in men, its target is the prostate and the typical age of its manifestation is when men are 60 years of age.
The endometrium and the peritoneum are derivatives of the same coelomic wall epithelium. Peritoneal mesothelium can undergo metaplastic transformations into endometrial tissue. Such transformation may occur spontaneously or be facilitated by exposure to the chronic irritation of retrograde menstrual fluid.
It is supposed that the immune system plays also a role in the pathogenesis of endometriosis. Women with this disorder appear to exhibit increased humoral immune responsiveness and macrophage activation while showing diminished cell-mediated immunity with a decreased T-cell and Natural Killer Cell responsiveness. A patient with endometriosis usually has pelvic pain that often cycles with menstruation. It is associated with dysmenorrhea, dyspareunia and infertility. The pain can be a deep constant ache with bilateral patterns of distribution and can radiate to the buttock and perianal region. One third of women with endometriosis are asymptomatic.
Cyclic pain accompanies bleeding at the time of menstruation. This could involve the bladder (hematuria), bowels (hematochezia and painful defecation), or, rarely, bleeding at uncommon loci, such as at the umbilicus, the abdominal wall, or the perineum. The degree of the visible endometriosis has no correlation with the degree of pain or other symptomatic impairments.
Acute exacerbations occur due to peritonitis caused by the leakage of old blood originating from an endometriotic cyst. These painful lesions involve peripheral spinal nerves rather than autonomic ones.
Secondary dysmenorrhea often occurs among women suffering endometriosis. Pain does frequently commence prior to menses. In case of patients presenting significant dysmenorrhea, endometriosis has to be thought of and her empiric therapy should be started.
Deep dyspareunia may be caused by the uterosacral ligaments being scarred, by uterine retroversion, etc. All of these may cause chronic backache as well. Associated intrapelvic and intra-abdominal adhesions may be also important determinants of the degree of pain experienced.
By a contiguous spreading, endometriosis may rarely invade the rectovaginal septum, the anterior rectal wall and the sigmoid colon, so that surgical interventions may often be required. The ectopic endometrial tissue can undergo malignant transformations, so that their histologic evaluation is necessary.
Postmenopausal endometriosis may be encountered in women who are on an estrogen replacement therapy.

Peritubal and periovarian adhesions obstructing the way of the ovum transport can cause subfertility. Peritoneal endometriosis hindering the tubal motility, the folliculogenesis as well as the function of corpus luteum may also lead to subfertility.
Diagnosis: symptomatically, by ultrasonography, MRI, hysterosalpingography, by examining CA 125 levels, etc.
Treatment: by using hormonal therapy, by the surgical removal of the endometrial tissues.
RFR method: detects and may eliminate the pathogens.
The most frequent resonances are: 307-321, 330, 354-359, 369-376, 391-392, 396-397, 402-410, 427-437, 442-451, 470-472, 496, 500, 526-530 kHz
It is important first to eliminate the virus(es), and then the pathogen bacteria and/or fungi.

16.13. HIV Infections

The human immunodeficiency viral infection is caused by *HIV-1* and *HIV-2 retroviruses* which destroy progressively the CD4+ helper lymphocytes, can cause AIDS and other diseases associated with impaired immunity. Transmission of the virus requires contact with a body fluid that contains infected cells or virus particles; which fluids include blood, semen, vaginal secretions, sperms, breast milk, urine and saliva. The disease is primarily spead through having sexual relations with an infected person, during which the mucous membrane lining of the mouth, vagina, or rectum is exposed to contaminated body fluids (see Chapter 23.3. HIV Infection and AIDS).

16.14. Gardnerellosis (Bacterial vaginosis)

Gardnerella vaginalis (previously named *Haemophilus vaginalis, Corynebacterium vaginale)*, a facultatively anaerobic Gram-negative rod is one of the microorganisms responsible for bacterial vaginosis (BV), which is the most common cause of vaginitis. BV is a nonspecific vaginitis caused by a polymicrobic synergistic infection. In case of this vaginitis *Gardnerella vaginalis* is usually associated with some other bacteria including f.i. *Mobiluncus, Bacteroides, Peptostreptococcus, Fusobacterium, Veillonella,* and *Eubacterium species. Mycoplasma hominis, Ureaplasma urealyticum,* and *Streptococcus viridans* may also be associated with them.
The acidic pH of healthy vaginas is owing to the hydrogen peroxide–producing lactobacilli present in high concentrations. Lactobacilli inhibit other anaerobic microorganisms elaborating hydrogen peroxide. In case of BV, the lactobacillus population is greatly reduced, so that the local pH of the vagina and the population of various anaerobes and G. vaginalis will get increased.
Sexual activity can link to the development of this infection, though BV and that of the rectum may occur among virginal females and virginal boys as well. Therefore, BV is not considered to be a sexually transmitted disease.
Predisposing factors may be the following: recent antibiotic use, decreased estrogen production of females, the wearing an intrauterine devices (IUDs).
The **symptoms** of bacterial vaginosis are characterized by an increased vaginal discharge and a vaginal malodor caused by the change in the vaginal flora. This vaginal fishy odor can be smelled usually after a sexual intercourse caused by the release of volatile amines from the vaginal discharge. This vaginal discharge is usually a thin, gray, homogeneous fluid which is adherent to the vaginal mucosa. Untreated chronic BV may cause endometritis, salpingitis, pelvic inflammatory disease, complications of pregnancy, including premature rupture of the membranes and chorioamnionitis. Infections following postgynecologic procedures may also occur. Concomitant infections have to be suspected in case of patients whose symptoms still remain after treatment for BV.
Diagnosis and differential diagnosis: symptomatically, by vaginal (pH, lack of lactobacilli, predominantly coccobacilli present) and microscopic examinations. By

culturing in order to exclude other infectious etiologies (f.i. Trichomonas species, Chlamydia trachomatis and Neisseria gonorrhoeae).
Treatment: by administering clindamycin, metronidazole, etc.
RFR method: detects and may eliminate the pathogen microorganisms. The examination and treatment of the sexual partners is also necessary, to be followed by the replacement of friendly vaginal flora.
The most frequent resonances are: 336-342, 390, 400, 496, 500-509 kHz
This list is not complete yet.

17. SKIN DISORDERS ASSOCIATED WITH INFECTIONS

The skin provides a remarkably good physical, mechanical and chemical barrier against infections. The complexity of immune response-associated cells present in a normal human skin is named skin immune system (SIS), which covers all humoral and cellular components involved in the cutaneous immune reactions of people, including the innate as well as the acquired immunity elements. Within the epidermis of the skin there is an integrated network of Langerhans cells (LCs) trapping antigens that may have penetrated the physical barrier and these LCs migrate to the lymph nodes to induce an effective immune response. Though many microorganisms live on the skin, they are normally unable to cause infection. Infections of the skin and its underlying tissues caused by bacteria include pyoderma, cellulitis, necrotizing fasciitis, skin gangrene and skin abscesses. There are also many other types of skin infections, caused by viruses, fungi and parasites. The skin is involved in certain systemic diseases of infectious origin, causing f.i. exanthems and other alterations.

17.1. Common Skin Infections Caused by Viruses

Viruses that often invade the skin belong to three groups. Illnesses caused by two of them, i.e. warts and cold sores on the lips are familiar nuisances. Warts are caused by *papilloma viruses*, cold sores are caused by *Herpes Simplex Viruses*, just like shingles. The third group of viruses infecting the skin belongs to *poxviruses*. The most dangerous *poxvirus* is the smallpox virus, causing Variola vera. Molluscum contagiosum is also caused by a poxvirus.

17.1.1. Skin Infections Caused by HPVs

Human Papilloma Viruses can cause various different clinical forms of common warts and genital warts. Warts may persist and spread on the patient for several years and may recur even several years after a total remission. They are frequently seen on the skin of patients with primary and secondary immune deficiencies, as well as concerning those who undergo an immunosuppressive therapy. The overwhelming majority of warts are harmless.

17.1.1.1. Common Warts (Verruca vulgaris)

Common warts are usually seen on the hands, fingers, and even under and around the fingernails, but can affect the skin anywhere on the body. These lesions are horny papules varying in size with rough surfaces. Confluence of cluster lesions occurs frequently. The lesions are usually asymptomatic, may nevertheless be very painful, f.i. on the soles or under the fingernails.

Diagnosis: by macroscopic examinations.

Treatment: by administering local preparations, by surgery and laser surgery. In severe cases by administering antiviral immunomodulating drugs f.i. Isoprinosine.

RFR method: is rarely effective in these cases.

The most frequent resonances are: 329, 392, 403, 487 kHz

The resonant frequencies of plantar warts are: 402-408, 424, 442-451, 465-470 kHz

17.1.1.2. Moist Warts (Condyloma acuminatum)

Moist warts, known also as venereal or fig warts, are lesions usually occuring on the mucocutaneous parts of the skin of the genital, perianal areas and seldom on the

subaxillary and periaxillary areas as well. These small skin growths are caused by various *Human Papilloma Viruses*. The infection can rarely affect the skin around the areola of the nipple, the margins of the mouth, and the skin between the toes. These warts are pink to red in color, moist and soft and may be pedunculated or elongated. They may occur in great numbers and can become macerated and malodorous. These warts are most often experiencedextended among young adults, though children and adults can also be affected. The eruption of moist warts is frequent and can be extensive in pregnancy. Although the lesions can be transmitted by sexual partners, they are not always venereal in origin. The most common types do not become cancerous. However, certain rare types infecting the uterine cervix, the penis and the anal region can become cancerous. The more sessile condyloma latum of secondary syphilis may be confused with this viral, genital wart.
Diagnosis: by macroscopic examinations.
Treatment: by electrodesiccation, laser surgery, surgery, by locally applicating liquid nitrogen and other antiviral preparations.
RFR method: detects and may eliminate the virus.
The most frequent resonances are: 236-240, 400-408, 442-451, 464, 477 kHz

17.1.2. Erythema Infectiosum

Erythema infectiosum (also named Fifth disease) is a contagious mild illness caused by Parvovirus B19 (B19V), the only member of the Parvoviridae family known to be human pathogenic. The virus has a tropism for the rapidly dividing erythrocyte precursors, particularly pronormoblasts and normoblasts, wherein by replicating to high titers it destroys the infected cells. Thus no reticulocytes are present to replace the aged or damaged erythrocytes, the level of hemoglobin in the blood will decrease, especially in case of infected patients suffering from hemoglobinopathies or hemolytic anemias. The disease may occur worldwide mostly during the spring months and affects usually children and teenagers. This infection spreads mainly by breathing in small droplets of moisture exhaled by an infected person. It can be transmitted from mother to fetus during pregnancy, perhaps causing stillbirth and a severe anemia developing due to bone marrow damages of the fetus. B19V may be spread by blood products, too, such as intravenous immunoglobulin (IVIG), packed RBCs, platelets, and nonrecombinant clotting factors. B19V lacks an outer envelope, due to which it is extremely resistant to heat, cold and solvents. (See also Chapter 5.2.6.)
Symptoms: begin after an incubation period from five to ten days with low-grade fever, malaise, headache, myalgia, nausea, rhinorrhea and a confluent erythema on the cheeks, showing a „slapped face". This rash corresponds to the appearance of immunoglobulin M (IgM) in the serum. One or two days later a bilaterally symmetric rashes develop on the arms, legs and trunk, rarely on the palms or soles as well. These maculopapular lesions tend to be confluent, may disappear and reappear within hours at the same areas waxing and waning sometimes for several weeks.
B19V may cause a papular-pruritic **„gloves-and-socks" syndrome**, an erythematous exanthem of the hands and feet, with a distinct margin at the wrist and ankle joints, associated with pain and edema. This syndrome is exclusively caused by B19V infections, is uncommon among adults and rare among children.
This viral infection can decrease the reticulocyte count to less than 1% (usually to 0%) in case of patients with hemoglobinopathies or hemolytic anemias, it may precipitate an aplastic crisis characterized by profound **anemia** caused by the temporary stopping of the production of new erythrocytes. The reticuloendothelial system removes the abnormal erythrocytes from the circulating blood while the virus-caused interruption in the new erythrocyte production triggers a crisis. B19V is the sole infectious agent which can cause an aplastic crisis. This latter complication is primarily observed in patients with underlying hemolytic anemias (f.i. in case of sickle cell disease, thalassemia) or in patients in

immunodeficient state (f.i. leukemia, HIV). Patients immunocompromised due to chemotherapy, immunosuppressive drugs, or having congenital and acquired immune damages may suffer from a chronic anemia caused by chronic B19V infections. This anemia will persist until the normal immune function returns. Patients with severe anemia suffer pallor, fatigue and tachycardia. A **symmetric postinfectious arthritis** with pain and swelling of the small joints of hands and feet can also come to pass, mostly among adult women. Knees and elbows are rarely involved. This arthritis may persist for weeks and months, may mimic rheumatoid arthritis (RA); though, unlike RA, the B19V infection does not damage bones and joints.
Erythema infectiosum can affect persons of all ages but is most common among children of school age, and may occur in epidemic form, too. 20% of persons infected with the B19V are asymptomatic. B19V infection may cause neurologic symptoms in people with HIV and other HTLV infections. In case of people with mycoplasma infections, B19V infection can cause a serious chronic disease process.
Diagnosis: symptomatically, based on the characteristic appearance of rashes, though B19V infection may be indistinguishable from other viral illnesses in absence of the classic exanthem. By serology for parvovirus. If positive IgG results are together with negative IgM ones it will indicate past infections (with no risk to fetus). If positive IgG results are together with positiv IgM ones it will indicate infection within the last 7-120 days (with a possible risk to fetus). If negative IgG results are together with positive IgM ones, an acute infection is indicated (with high risk to fetus). By examinations of the low reticulocyte count (0-1%).
Differential diagnosis: rubella and certain enteroviral infections.
Treatment: symptomatically, as there is no specific treatment. IVIG has a limited role in treating of chronic parvovirus infections. Aplastic crisis requires packed RBC transfusions.
RFR method: though it can detect and eliminate the parvovirus, it is nevertheless forbidden during pregnancy because of the danger to damage the fetus. If used at an early stage in case of infected children and adults, it may inhibit the appearance of rashes and other symptoms.
The most frequent resonances are: 413-414 KHz
This frequency list is not complete; as there are other parvovirus subspecies having different resonant frequencies.

17.1.3. Hand-Foot-and-Mouth Disease

Hand-foot-and-mouth diseasee (HFM), a viral infection affecting infants and children is usually spreading via the fecal-oral or the oral-oral route, though its transmission by respiratory droplets may also occur. HFM can be caused by several members of the enterovirus family. This slightly contagious illness is most often caused by *Coxsackie virus A-16* and *Enterovirus 71 (HEV17),* less frequently by certain other enteroviruses, including *Coxsackie viruses A5, A9, A10, B1, B3* and *Herpes Simplex Viruses* as well. This infection occurs typically in nursery schools or kindergardens causing small epidemics, usually in summer and in the autumn months. Its usual incubation period lasts 1-3 days.
Symptoms: usually begin with low grade fever, malaise, abdominal pain, upper respiratory symptoms, followed by a painful oral maculo-papulous enanthema progressing to the vesicles surrounded by erythema and then ulcerating, and by blisters on the palms, soles and on the buttocks of small children and infants.
Secondary skin infections may occur. Severe complications can develop in case of CNS or cardiopulmonary involvements. These sequelae may include dysphagia, limb-weakness, cardiopulmonary failures and can even cause death.
Diagnosis: symptomatically, by identifying the virus by culturing and serology.
Treatment: symptomatically, as there is no specific treatment for hand-foot-and-mouth disease.

RFR method: can point to the immunosuppressed state and to virus elimination.
Detects and eliminates the pathogen microorganisms.
The most frequent resonances are: 307, 310-319, 321, 324-331, 342-350, 375-386 kHz
The resonant frequencies of HSV are: 291-293, 344-345 kHz
The resonant frequencies of Mycoplasma fermentans are: 440-451 kHz
The resonant frequencies of EBV are: 372-383, 518-519 kHz

17.1.4. Molluscum Contagiosum

Molloscum contagiosum is an infection of the skin caused by a *poxvirus* and which is caracterized by skin-colored, smooth waxy bumps, i.e. papules. The virus causing molluscum is contagious, spreads by water, direct skin contact and is often transmitted sexually. The incubation period varies between 2 weeks and 2 months. The bumps are elevated, waxy, pearly-white papules usually less than 6-12 mms in diameter, have a tiny dimple in their center, though the bumps may vary in number and size, ranging from 1 mm up to giant lesions of 1 to 2 cms in diameter. By squeezing one of these bumps, a curdlike, chessy material is expressed, which, upon electron microscopic examinations, proves to be loaded with virus particles. Smears of the curd prepared for an ordinary light microscopy show a specific diagnostic picture of clusters of cells containing eosinophilic, giant cytoplasmic inclusion-bodies. The papules may appear alone or in-groups. The lesions may persist and spread from months to years. The face, especially the eyelids, the trunk and the ano-genital areas are most often involved. Molluscum lesions are frequently traumatized and secondarily infected, these injuries can cause the resolving of these lesions. Spontaneous regression can eventually occur without scarring.
Diagnosis: symptomatically, by microscopy.
Differential diagnosis: by distinguishing it from keratoacanthoma, basal-cell epithelioma, and pyogenic granuloma.
Treatment: the bumps can be treated by freezing or by the removal of their core with a needle.
RFR method: detects and may eliminate the poxvirus.
The most frequent resonances are: 290-291, 307, 319-320, 325, 332-338, 348, 363-372, 376, 396, 401-410, 420, 425-426, 454, 482-486, 448-451, 518-522, 544-545, 551-554 kHz

17.2. Common Bacterial Skin Infections

17.2.1. Local Bacterial Skin Infections

17.2.1.1. Impetigo

This illness can be caused by *staphylococcus* and *streptococcus* species, sometimes associated with fungi, and is characterized by the formation of small, pus-filled pustules. Though these bacteria live on the human skin usually without causing any symptoms, they can enter the skin through a wound or other injuries and can cause infections, mostly among children under 6 years and among immune damaged people, too. Primary impetigo attacks the otherwise normal skin, while the secondary impetigo is an infection of an other skin disease present, f.i. a dermatitis, pruritus etc. The pustules vary in size. This disease primarily affects children usually between 2 and 6 years. Though impetigo can appear anywhere on the body, it is more frequently experienced on the face, arms and legs of children. The clinical appearance of impetigo is characterized by 3 forms: i.e. **impetigo contagiosa**, mostly starting on a child's face with an itchy, red sore and honey-coloured crusts, healing without scarring. The second form of the illness is the **bullous impetigo**

affecting infants younger than 2 years old. Its symptoms are painless, small or larger fluid-filled blisters with yellow crusts usually on the trunk and the extremities. **Ecthyma**, the third form, affects the deeper layers of the skin, too, i.e. the dermis, where painfull, fluid-filled ulcerations are to be seen with thick, grey-yellowish crusts on them, leaving scars when healing. The regional lymph nodes are usually swollen.
Impetigo caused by straphylococcus and streptococcus can rarely lead to kidney failure and heart failure.

17.2.1.2. Erythrasma

Erythrasma is an infection of the top layers of the skin caused by *Corynebacterium minutissimum*. Erythrasma affects mostly adults and those with diabetes. Likewise as a fungal infection, erythrasma often appears in areas where skin touches skin, such as under the breasts and in the armpits, and genital area, especially concerning men, where the thighs touch the scrotum. The infection causes irregularly shaped pink patches that may later turn into fine brown scales. The infection seldom spreads to other areas, too.
Treatment: by administering Doxycyclin or Erythromycin. A repeated treatment is often required.
RFR method: detects and may eliminate the Corynebacterium minutissimum.
The most frequent resonances of Corynebacterium are: 301, 308-320, 340-348, 397, 403, 442, 473 kHz
This list is not yet complete, as there exist other subspecies with different resonant frequencies.

17.2.1.3. Paronychia

Paronychia is the infection of skin around the fingernails. The ordinary paronychia, i.e. „run-around" is a superficial infection of the epithelium sides of the nail, being usually the result of tearing a hangnail. Paronychia can be caused by many different bacteria, including *Pseudomonas*, *Proteus*, *Staphylococcus*, and by fungi, such as *Candida*, either alone or in combination.
The infection often starts due to a break in the skin at the nail, or to vigorous manicuring and chronic irritation. This illness, if caused by bacteria can be rather painful. Hot applications will lead to subsidence of the paronychial cellulitis, though their does often appear a superficial pus-filled blister or the infection might lead to a painful subungual abscess. In the latter case incision and drainage with partial or complete removal of the nail becomes necessary. But the infection can, even in such a case spread further on. Chronic paronychia produced by fungi or bacteria often occurs concerning diabetic, psoriatic patients, and those suffering some subtype of pemphigus.
Treatment: by administering effective antibiotics and/or antifungal drugs or by surgery.
RFR method: detects and eliminates the pathogens. RFR method is only to be used after being diagnosed. RFR method can substitute antibiotic or antifungal therapy.
The most often found resonant frequencies are those of the
Pseudomonas group: 323-324, 330-336, 348, 351, 374, 380, 396, 401, 414, 438, 446-447, 492-496, 504, 507, 512-513, 579 kHz
Proteus species: 320-329, 333-339, 345-352, 408-416, 426, 516, 522-529, 535 kHz
Candida species: ; 293, 295, 297, 332, 345, 352-359, 372, 380-390, 396-397, 403, 410, 440-453, 520, 554-559, 572-586 kHz

17.2.1.4. Folliculitis

Folliculitis, the inflammation of the hair follicles is usually caused by bacteria, but sometimes by fungi, too. In case of *folliculitis Bockhardt*, named also Ostiofolliculitis, this inflammation occurs extremely superficially and is caused by *staphylococci* developing

usually secondarily to itching dermatoses. The normal folliculitis is characterized by a pruritic inflammation in the entire depth of the follicle. *Folliculitis barbae* occurs on the bearded area of men, is caused usually by *staphylococci* and characterized by erythematous follicular papules and pustules often progressing to raised crusting plaques.

17.2.1.5. Furunculus

Boils are large, tender, swollen, raised areas caused by *staphylococcal* infections affecting the hair follicles and their surroudings.

17.2.1.6. Carbunculus

Carbuncles are clusters of boils that damage the hairs, which can be pulled out easily. Carbuncle develops and heals more slowly than single boils and, being a more serious type of infection, may cause scar formation, fever and fatigue. Carbuncles occur most frequently among men, mostly on the back of their neck. Patients with immunodeficiency, diabetes and with chronic viral infections are more prone to get a carbunculus.

Treatment: The skin and its environment must be kept clean! It is advisable to use liquid antibacterial sulfur soap. Local desinficients, antiseptic cremes and solutions can be effective. In more serious cases administration of antibiotics should be taken orally. As a last resort surgery might be necessary.

RFR method: detects and may eliminate the bacteria.

The most frequent resonances are: 324-327, 331, 345, 372, 377, 380-387, 397, 402, 434, 442-451, 461, 482, 491, 555-558, 562-567 kHz

This list is not complete; there are other subspecies having different resonant frequencies.

17.2.1.7. Erysipelas

Erysipelas is an acute skin infection caused by *streptococci*. This infection appears mostly on the face, arms and legs, beginning sometimes where the skin is broken. If feet moisten in the gum-boots bacteria can easily infect the skin causing erysipelas.

A shiny, red, slightly swollen, tender rash develops, sometimes with small blisters. Erysipelas can be caused also due to the bite of an infected horse-fly. The lymph nodes of the infected area may become enlarged and painful; people with particularly severe infections develop fever, shaking, chills, fatigue and vomiting.

17.2.1.8. Cellulitis

Cellulitis is a spreading infection, sometimes developing beneath the deep layers of the skin. Cellulitis most often results from a *streptococcal* infection or, particularly after a wound occurs, from a *staphylococcal* infection. *Many other bacteria* can cause cellulitis, especially following bites of animals or injuries got in water. The infection occur most often on the legs, and begins with skin damage from a minor injury, a sore, or a fungal infection between the toes. Cellulitis produces swelling, tenderness, warmth and redness. Some areas may be bruised and small blisters can develop. Symptoms of the infection may include fever, chills and headache. More serious complications, such as confusion, low blood pressure and rapid heartbeat are symptoms caused by *streptococcus toxin*. Cellulitis can often become a chronic disease.

Some people are at a particular risk of contracting skin infections, including: diabetes patients, due to their poor blood flow to the skin, especially on hands and feet; AIDS patients, or others with damaged immune system. Genetic predisposition can also play a role in the development of cellulitis.

Diagnosis: by bacterial culturing.

Treatment: by administering effective antibiotics. People with mild cellulitis may take oral antibiotics; older people and people with rapidly spreading cellulitis, high fever, or

other evidence of a serious infection usually need an antibiotic injection before oral treatment.
RFR method: detects and may eliminate the bacteria.
The most frequent resonances are: 313-321, 360-375, 401-403, 420-444, 447-453, 508, 520-544, 548 kHz
It is important to measure carefully, as more bacterial and fungal species may be present at the same time in this illness.

17.2.1.9. Mycobacterial Skin Ulcer (Buruli)

Buruli ulcer is a chronic, infectious, necrotizing disease of the skin caused by *Mycobacterium ulcerans*. It is a tropical disease existing in aquatic areas. The mode of transmission is yet unknown, though fish, aquatic snails and plants are thought to play a role in it. Endemic areas are in Africa, f.i. in the Buruli County in Uganda, Zaire, Congo, Cameroon, Nigeria, Benin, Ghana and Liberia. The infection spreads in several other areas and countries, too, such as in Australia, Central and South America, Southeast Asia and New Guinea.
Symptoms: On the locus of a penetrating skin trauma the illness manifests itself initially as non tender, subcutaneous, firm nodules 1-2 cm in diameter. *M. ulcerans* secretes mycolactone, a strong immune suppressant, necrotising agent, an activator of cellular apoptosis and a lipid toxin, causing fat necrosis, so that within the next 1-2 months, the affected area becomes fluctuant and is followed by the formation of a painless, undermined ulceration. Healing may occur spontaneously, but more often, the disease is slowly progressing causing other ulcerations, granulation, scarring, and contractures. The infection may destroy the nerves, skin appendages and blood vessels, and occasionally invades the bone. A secondary infection caused f.i. by mycoplasma may occur by developing other nodules. The disease can cause hideous deformities.
Diagnosis: examining the smears from the necrotic base of the lesion stained with Ziehl-Neelsen and by PCR.
Treatment: by administering rifampicin and streptomycin for eight weeks in the hope of reducing the need of surgery.
RFR method: detects and may eliminate the M. ulcerans. Use RFR method combined with antibiotics for a long time!
The most frequent resonances are: 335-345, 430-437, 440-454, 522-534 kHz
The frequency of the antibiotic resistant bacterial population is high.

17.2.2. Staphylococcal Scalded Skin Syndrome

Staphylococcal Scalded Skin Syndrome (SSSS) is a widespread, toxin-mediated type of exfoliative dermatitis where the skin looks like as it had burned. Toxin-mediated staphylococcal syndromes comprise a group of blistering skin diseases, ranging in severity from localized bullous impetigo to a SSSS, in which a superficial blistering and exfoliation follows a widespread painful erythema. This syndrome can lead even to a toxic shock syndrome. SSSS is caused by exotoxins produced by staphylococci. SSSS also is known as Ritter von Ritterschein disease of newborns. Staphylococcal Lyell syndrome, Staphylococcal epidermal necrolysis and SSSS are synonym names of the said disease. The disorder is caused by toxigenic strains of *Staphylococcus aureus,* usually belonging to phage group 2 (types 3A, 3B, 3C, 55, or 71). Two exotoxins (ETs) of these bacteria, the epidermolytic toxin A (ET-A) and epidermolytic toxin B (ET-B) are responsible for the pathologic changes experienced in SSSS. These toxins are serine proteases specifically targeting desmoglein-1, a desmosomal cadherin protein that ensures the cell-to-cell adhesion of keratinocytes in the granular layer of the skin, and which is the target protein of an autoimmune blistering dermatosis, pemphigus foliaceus, too. The staphylococcal

epidermolytic toxins cause intraepidermal splittings through the granular layer at the cleavage of these desmoglein 1 proteins. ETs are also sources of superantigenic activity. They can cause an extreme degree of inflammation by activating macrophages in order to produce proinflammatory cytokines, such as TNFalpha and interleukin 6. The systemic symptoms of the illness, particularly the characteristic rash are very likely a direct result of these toxins. SSSS is common among neonates and children, though it can affect adult people as well. Diabetes patients having a poor blood flow to the skin, and people with depressed immune state, f.i. AIDS patients are at a particular risk to contract this syndrome. Localized *S. aureus* infections can occur on the skin, throat, nose, mouth, umbilicus and the GI tract. Infections like these are often not apparent before the SSSS rash appears.

Symptoms: The syndrome, beginning with an isolated, crusted infection may look like an impetigo. In case of neonates the lesions start usually around the stump of the umbilical cord during the first few days of their life, and is often accompanied by general malaise, fever, irritability and skin tenderness. By the loss of the protective skin barrier, other infective microorganisms can easily penetrate the body. Critical amounts of fluid can be lost due to oozing and evaporation. The staphylococcal toxins cause a small degree of general toxicosis as well. Cellulitis, pneumonia and sepsis are possible complications of the illness.

Diagnosis: The definitive diagnosis depends on the results of bacterial culturing and biopsy. Examination of frozen sections of the lesions can easily confirm the diagnosis. PCRs serum tests for the toxin are available.

Differential diagnosis: Staphylococcal Scalded Skin Syndrome must be distinguished from diseases that appear similarly, such as toxic epidermal necrolysis, which can be caused by many an other factor. Pemphigus foliaceus, a generally benign autoimmune skin disease with more subtypes is caused by genetic and environmental factors affecting mostly people between 50-60, though it can occur at any age.

Treatment: by administering penicillinase-resistant, antistaphylococcally effective antibiotics.

RFR method: should only be used together with antibiotics. Detects and eliminates Staphylococci. RFR method plays an important role concerning antibiotic polyresistant infections. Use the treatment consecutively, as long as needed.

The most frequent resonances of Staphylococcus are: 294, 308, 323-329, 345, 347, 367, 376-381, 388, 401-402, 421, 434, 448-453, 458, 463, 465, 482, 484, 486, 490-491, 504, 511, 517, 542, 552, 556-557, 563-568, 576 kHz

This list is not complete; there can be other subspecies having different resonant frequencies.

17.3. Common Fungal Skin and Nail Infections

Some fungal infections produce only a small amount of irritation, scaling and redness, or do not cause any symptoms at all, while other fungal infections cause itching, swelling, blisters, and severe scaling. Strangely, certain fungal infections on one part of the body can even cause rashes on not infected other parts of the body. These eruptions frequently represent allergic reactions to the fungus. From a clinician's point of view, most mycotic infections of the skin may be sorted into two major categories: i.e. dermatophytosis and candidiasis, each of which has an etiology, associated systemic disease and response to treatment all differing from each other.

Fungal diseases have assumed a new importance in medicine because of the increased number of patients treated with chemotherapy for leukemia and other neoplasms. Almost all of the saprophytic fungi are known to invade the tissues of patients treated with chemotherapeutic agents, who have had some organ transplants, are diabetics, or whose immune response is inadequate (see also Chapter 7.).

17.3.1. Dermatophytosis (Ringworm Infections)

The various types of dermatophytosis constitute ringworm infections. All of these infections respond to oral griseofulvin-type antifungal drugs and are confined to the epidermis, hair, toenails and fingernails. Dermatophytosis is most often caused by one of these three types of fungi:

1) *Microsporum audouini* is a human parasite, the principal pathogen among those which cause epidemic urban fungal infections of the scalp.

2) *Microsporum canis*, which affects the scalp and also the face, causing there boggy nodules, is a parasite of animals and originates mainly from young farm animals and pets.

3) *Trichophyton mentagrophytes*, *Trichophyton rubrum,* as well as some other species of this group and *Epidermophyton floccosum* are also human parasites and the most common causal agents of the dermatophytosis of the feet, most frequently infected. The fungi can also get into the newly-forming parts of the nails, producing thickened, lusterless and deformed nails. Infections of the toenails are much more common than those of the fingernails.

The types of fungi that can affect the upper extremities, face, and trunk include *Trichophyton, Microsporum*, and *Epidermophyton* species (see also Chapters 7.4.).

17.3.2. Cutaneous Candidiasis

Candidiasis represents the second major category of mycotic infections. These infections do not respond to oral griseofulvin at all and are often caused by Candida albicans, although occasionally by Candida tropicalis, Candida krusei, Candida glabrata and Candida albicans var. stellatoidea as well. Candida albicans can exist as a harmless saprophyte in the gastrointestinal tract and in the vagina. It is causing illness more often among females, among pregnant ones, those taking oral contraceptives or broad-spectrum antibiotics and among diabetic patients, or those with an otherwise compromised immune system. The association with diabetes mellitus is so common that all patients with candidiasis should be examined regarding DM. C. albicans can invade the epidermis if the skin is exposed to humidity or becomes macerated. Candida can affect the nails, the genital region causing vulvovaginitis with associated skin inflammations, can affect also areas where skin touches skin, such as under the breasts and in the armpits as well as the mucous membranes. Chronic paronychia of hands is often caused by C. albicans.

Symptoms vary depending on the location of the infection. *In skin folds* or in the navel candidal infections usually cause red rashes, often with patchy areas that ooze small amounts of whitish fluid. Small pustules may appear, especially at the edges of the rash itching or burning. Candidal rashes *around the anus* may be raw, white or red and itchy. Symptoms of *vaginal infections* include burning, itching, redness along the walls and the external area of the vagina and a white or yellow discharge. *Penile infections* most often affect men with diabetes or those whose female partners have candida vulvovaginitis. The latter infection produces a red, scaling and sometimes painful rash on the underside of the penis.

Candidal paronychia produces painful swelling and pus. Candida-infected *nails* may turn white or yellow and become separated from finger or toe (see also Chapter 7.3.1.).

17.3.3. Pityriasis Versicolor

This infection is caused by *Malassezia furfur* and is characterized by white to light brown coloured, slightly scaling patches on the skin of the chest and the back. This infection occurs rather frequently among young adults. It rarely causes itching and produces patches preventing these skin areas from tanning (see also Chapter 7.3.3.2.).

The differential diagnosis between dermatophytoses caused by any one of the three types of fungus already mentioned and candidiasis may without culturing the fungi be difficult, if not impossible. The direct examination of the scales got from a scaling eruption in the intertriginous area can be informative but not always diagnostic as mycelia can be found in dermatophytosis and candidiasis as well; spores, however, can only be seen in case of candidiasis.
Diagnosis: symptomatically, by microscopic examinations and fungal culturing in order to identify the causative species.
Treatment: by administering antifungal drugs locally or systemically, if needed.
RFR method: detects and may eliminate the fungi, but has to be used together with an antimycotic drug.
The most frequent resonances are: 315, 358, 453-455, 461, 501-504 kHz

17.4. Parasitic Skin Infections

Parasitic skin infection may be caused by a microparasite or macroparasite attack.

17.4.1. Cutaneous Leishmaniasis (Micro Parasitic Skin Infection)

Cutaneous leishmaniasis is a very frequent form of microparasitosis, and the most common form of leishmaniasis, affecting cc. 12 million people worldwide. This skin infection transmitted by the bites of sandflies is caused by a single celled parasite. There are about 20 different species of Leishmania that may cause cutaneous leishmaniasis, but not all are pathogen to people. The primary clinical forms of leishmaniasis are the cutaneous, the mucocutaneous and the visceral disease. Cutaneous leishmaniasis can be localized, diffuse cutaneous, recidivans or post-Kala azar dermal leishmaniasis.

17.4.1.1. Localized Cutaneous Leishmaniasis

After being infected by a sandfly bite inoculation on an exposed skin area, crusted papules and ulcers are developing from several weeks, rarely even for months. Lesions can be spread via the lymphatics so that a large number of ulcers can appear with scarring though healing usually spontaneously.

17.4.1.2. Diffuse Cutaneous Leishmaniasis

This form is analogous to lepromatous leprosy. Due to the impaired cell-mediated immune response of the patients this form is characterized by multiple, widespread cutaneous papules and nodules which cannot heal by themselves, relapses will even after treatments occur.

17.4.1.3. Cutaneous Leishmaniasis Recidivans

This rare clinical variant of leishmaniasis affects most often the face manifesting itself as an enlarging papule healing with central scarring, being long lasting and recurring, and can cause significant facial destructions as well. This infection may be transmitted via connatal route or blood transfusions, contaminated needle sticks and other carriers. HIV can easily be associated with the disease.

17.4.1.4. Post Kala-azar Dermal Leishmaniasis

This form is a sequel of Kala-azar that may appear on the skin of the affected individuals even up to 20 years after being partially treated, untreated or considered to be adequately treated. Symptoms are hypo-pigmented macules, papules, nodules and a facial erythema.

Though all microorganisms causing Kala-azar can lead to this form of illness, it is commonly associated with *Leishmania donovani* which gives different disease patterns in India from those experienced in Sudan. In case of the Indian variant nodules will get enlarged with time forming plaques rarely ulcerating, while, in contrast, the African variant often ulcerate when progressing. Nerve involvement is common in case of the African variant but is rare on the Indian subcontinent. Histology demonstrates a mixture of chronic inflammatory cells together with macrophages forming epitheloid granulomas.
Diagnosis: symptomatically and by PCR.
Differential diagnosis of the local cutan leishmaniasis includes impetigo, pyoderma gangrenosum, deep fungal infection, mycobacterial infection, sarcoidosis and Squamous Cell Carcinoma. Diffuse cutaneous leishmaniasis and Post Kala-azar dermal leishmaniasis resemble the lepromatous leprosy. Recidivans cutaneous leishmaniasis may mimic a cutaneous tuberculosis (lupus vulgaris, tuberculosis verrucosa cutis), psoriasis, a deep fungal infection, or a nummular dermatitis.
Treatment: by administering Amphotericin B, Miltefosine, Fluconazole and by surgery.
RFR method: detects and may eliminate the parasites.
The most frequent resonances of Leishmania brasiliensis are: 321-323, 399-406, 508-512 kHz
The most frequent resonances of Leishmania donovani are: 315-319, 397-403, 507-510, 537-541 kHz
The most frequent resonances of Leishmania mexicana are: 319-321, 401-405, 510-514 kHz
The most frequent resonances of Leishmania tropica are: 320-325, 400-408 kHz

17.4.2. Macroparasitic Skin Infections

Most macroparasitic skin infections are caused by tiny insects or worms that burrow themselves into the skin and live there. Some parasites live in the skin only for a part of their life cycles; others are permanent residents laying their eggs and reproducing there.

17.4.2.1. Scabies

Scabies is a mite infestation causing tiny reddish pimples and severe itching. The illness is caused by the bites of *Sarcoptes scabiei* mites. The infestation spreads easily from person to person being in physical and sexual contact, and is often spreading over an entire household. Mites can rarely be spread via clothing, bedding and other shared objects; their survival is brief, a normal laundering and ironing is enough to destroys them. Intensive itching, probably also due to an allergic-irritative reaction to the mites, reddish papules and water-filled blisters are the first experienced symptoms of the infection. The burrows of the mites may be scarcely seen, as the inflammation induced by scratching obscures them.
Diagnosis: symptomatically and by microscopic examinations in order to confirm the presence of the mites.
Treatment: by locally administered Permethrin, Lindane, Pyrethrin, etc.
RFR method: can be used together with antiparasitic drugs to detect and eliminate the scabiei.
The most frequent resonances are: 354, 365-370, 401, 417-418, 450, 471 kHz

17.4.2.2. Creeping Eruption

Creeping eruption is a hookworm infection transmitted to the exposed skin in warm, moist soil. The infection is caused by *Ancylostoma species* normally inhabiting dogs and cats. The eggs of these parasites are deposited in the ground from the feces of infected dogs and cats. If a person goes barefoot the hookworm can penetrate his/her skin.
Treatment: by applying a liquid preparation of Thiobendazole to the affected area.

RFR method: can detect and eliminate the parasite, used together with the topical treatment.

The most frequent resonances are: 383-404 kHz

17.5. Acne

Acne is a common skin disease of adolescent persons in case of which the pores of the skin become clogged, leading to cause pimples and to inflamed, sometimes infected abscesses. Acne vulgaris occurs predominantly on the face and, to a lesser degree, on the back, chest and shoulders. It tends to develop among teenagers caused by an interaction between hormones, skin lipid content and bacteria present on and in the skin and in the hair-follicules. During puberty, the sebaceous glands of the skin become more active producing an excessive amount of sebum. Acne is characterized by a variety of clinical lesions. These lesions may be noninflammatory as well as inflammatory papules and nodules. The noninflammatory papules are named comedones, which may be either open or closed. The closed comedones are precursors of the large inflammatory nodules, papules and pustules. In addition, cysts and scars of various sizes may occur. The typical acne scar is a sharply punched-out pit. The pustular and cystic lesions are usually sterile, despite the large amount of purulent exudates experienced following their incision, although they may contain *Corynebacterium acnes* and *Actinomyces israelii,* too.

Bacteria grow in the plugged pores and break down some of the fats in the sebum, irritating the skin. The irritated blackheads and whiteheads produce skin eruptions commonly known as acne pimples. *Corynebacterium acnes* is held to be responsible for a lipolysis with the release of fatty acids; causing an inflammatory process in the follicle wall. Theoretically acne develops as a result of the said primary inflammation of the follicle wall, where the follicle partly ruptures and due to its components being spilled-out, leads to the development of a perifollicular inflammatory process. The inflammatory infiltrate is initially lymphatic; but later on, as a result of the present keratinous material, gram-positive diphtheroids and sebum, there develops an inflammation with foreign-body giant-cell reactions. In case of deep acne, the infection produces large, red, raised inflamed areas, pus-filled cysts and abscesses.

Acne disease is a prevalent, sometimes severe cosmetic and medical problem affecting especially adolescent persons, its therapy is complex and can be prolonged.

Acne often becomes worse in winter, gets better in summer, probably due to the beneficial effect of the sun. In some cases certain foods can provoke outbreaks. Each menstrual period of young women may cause acne eruptions, which can clear up or get substantially worse during their pregnancy and because of certain effects of administered oral contraceptives. Secondary infections of acne disease caused by other bacteria or fungi may also be experienced. The inflammatory skin reaction to these microorganisms are usually characterized by chronic suppuration, extensive necrosis and intense fibrosis.

Diagnosis: symptomatically; by bacterial culturing

Treatment: depends on the severity of the symptoms. Topical Clindamycin, Erythromycin, Minocyclin, Doxycyclin, Benzoil peroxid, resorcinol, tretinoin etc. If they are ineffective, isotretinoin taken orally is the best treatment. Isotretinoin can harm a developing fetus, women taking it must take strict contraceptive measures in order to avoid becoming pregnant.

RFR method: is only effective against bacterial components and in case of secondary infections.

The most frequent resonances in acne are: 315, 324-328, 348, 372, 376-378, 383-389, 395-402, 409-410, 420, 438-444, 450-454, 456, 460, 505, 576 kHz

17.6. Rosacea

Rosacea can be a chronic skin disorder of adults affecting mostly the face, causing redness and tiny pimples. Its eruptions appear usually on the cheeks, chin and nose. Symptoms are characterized by swelling, pimples, hyperemic areas and, in their advanced stage, by thickened skin areas. Rosacea is associated with the disorders of the digestive tract, f.i. of its hypochlorhydric state, gastric and bowel-tract infections, food allergy and intolerance, etc. These infections may be simple bacterial infections, caused f.i. by *Helicobacter pylori*, pathogenic *Escherichia coli* species, *Salmonella*, *Pseudomonas*, etc., or may be the result of very complex conditions, f.i. parasitic infections; viral infections, such as caused by *Norwalk virus, Rotavirus*, *Enterovirus* and *Coxsackie viruses*; fungi such as *candida*; together with an allergic or food intolerance trigger. The bile duct and the intestinal tract are interrelated. Inflammatory bowel diseases and inflammations of the bile tract system may cause rosacea in case of predisposed people. Women are more often affected than men.

Diagnosis: symptomatically

Differential diagnosis: acne, chemical and physical irritants

Treatment: after the identification of pathogens antibiotic therapy, by administering friendly bacteria, nicotinacid and B-vitamins.

RFR method: detects and may eliminate the pathogen microorganisms; and helps to replenish the friendly bacteria flora.

The most frequent resonances are: 346, 355-362, 372, 377-379, 449-452, 554, 570-580 kHz

17.7. Urticaria

Urticaria (hives) is one of the most frequent dermatologic disorders. It is multicausal, its reaction pattern represents cutaneous mast cell degranulations, resulting in extravasation of plasma into the dermis, causing itchy, raised, well-circumscribed erythematous and edematous swellings involving the dermis and epidermis. Urticaria may be acute (lasting less than 6 weeks) or chronic (lasting more than 6 weeks).

The etiology of acute and chronic urticaria is numerous. The etiologic agent is more often identified in case of acute urticaria (50%) and less often in case of chronic urticaria (10-20%). The mast cell is the primary agent in the *pathogenesis* of urticaria. Mast cell stimulation results in the release of both preformed (histamine) and newly formed (prostaglandins) mediators from cytoplasmic granules, causing wheal formation, vasodilatation and erythema. Dermal mast cells secrete preformed mediators, including histamine (mainly the cause of pruritus), proteases, IL-1 and TNF-alpha. A number of mediators may be involved in the pathogenesis of urticaria, which may explain why antihistamines are not always effective.

Due to the diverse etiology of urticaria there are various urticaria forms including acute IgE–mediated urticaria, chemically-induced urticaria (non-IgE-mediated), IgG-mediated urticaria, urticarial vasculitis, autoimmune urticaria, cholinergic urticaria, cold urticaria, etc.

17.7.1. Acute IgE-mediated Urticaria

is the classic form of type I allergic reactions, is usually moderate, but may be accompanied by more serious clinical manifestations such as angioedema and anaphylactic shock. Food allergic reactions typically fit into this category. It is most often caused by the consumption of shellfish, nuts, eggs, fish, acid derivatives, dye, or some combination of these. The lesions of IgE-medicated urticaria usually show up in a few minutes after being exposed to the allergen and usually last less than 24 hours, are often migratory, leaving no residual skin signs.

17.7.2. Non IgE-mediated Acute Urticaria

can be caused by various factors, f.i. by being infected whith certain viruses or bacteria, such as *streptococci* or possibly *Helicobacter pylori*. In these cases, the hives may be exacerbated by other factors too, f.i. by those, listed below, under Physical Urticarias.

17.7.3. Chronic Urticaria

is often a frustrating condition for patients and caregivers and is characterized by recidive or persistent hives or wheals, which are edematous, mostly pruritic. Chronic urticaria may remain symptomatic and its symptoms remains medication dependent for years.

The primary subgroups of chronic urticaria include physical urticaria (symptomatic dermatographism, cholinergic urticaria, pressure urticaria), urticaria secondary to an underlying medical condition, and chronic idiopathic urticaria. Physical urticaria, reproducible with the appropriate stimuli, can be identified with a thorough patient's history and challenge testing.

Approximately one third of patients with chronic urticaria have either one or both antithyroglobulin antibodies and antimicrosomal antibodies, and up to one fifth have abnormal thyroid functions. A positive autolog serum test result supports an autoimmune process not indicating though which of the autoantibodies (anti-IgE, anti-FcεRI, or anti-FcεRII) is present. Affected patients may be categorized as having **autoimmune chronic urticaria**. Other non–IgE-mediated mast cell degranulators include radiocontrast media, morphine, codeine, and vancomycin. Patients with chronic urticaria may often develop an angioedema following the administration of aspirin or other nonsteroidal anti-inflammatory drugs. Food allergy does but rarely cause chronic urticaria.

The type II allergic response is mediated by cytotoxic T cells, causing deposits of immunoglobulins, complement factors and fibrin around the blood vessels leading to **urticarial vasculitis**. The lesions of urticarial vasculitis usually last longer than 24 hours, are painful and pruritic, and often leave purpuric and hyperpigmented lesions.

Type III immune-complex disease is associated with SLE and other autoimmune diseases causing urticaria. **Complement-mediated urticaria** can be caused by *viral* and *bacterial* infections, serum sickness reactions and transfusion reactions. Urticarial transfusion reactions occur if allergenic substances in the plasma of the donated blood product react upon the preexisting antibodies of the recipient. Certain drugs (opioids, vecuronium, succinylcholine, vancomycin, etc) and radiocontrast materials cause urticaria in a non-IgE mediated way. Physical urticaria forms are provoked by certain physical stimuli and are named immediate pressure urticaria, delayed pressure urticaria, cold urticaria and cholinergic urticaria. In case of the so-called idiopathic urticaria no concrete causative factor can be found.

Factors, known to cause often urticaria, are the following:

Medications: f.i. aspirin, other NSAIDs, opioids, ACE inhibitors and alcohol.

Contactants (causing **contact urticaria** locally and even generalized within 30 minutes after contact with the patients's skin): f.i. rubber latex, plants, animals (eg. caterpillars, dander), medications and food (fish, garlic, onions, tomato, etc).

Foods and food additives (by eating): shellfish, eggs, nuts, strawberries, certain baked goods, etc.

Arthropod assault (usually causing papular urticaria): scabies, bedbug bites, fleabites, etc.

Infections: *Hepatitis B virus*, *HTLV*, *Herpes Simplex Virus*. *Streptococci, Borrelia B.s.l. Mycoplasma species*, *Helicobacter pylori*, *Mycobacterium tuberculosis*, etc.

Autoimmune diseases: SLE, Cryoglobulinemia, Juvenile Rheumatoid Arthritis, Autoimmune Thyroid Disease, etc.

Autoinflammatory diseases: the genetically defined Muckle-Wells syndrome (characterized by amyloidosis, nerve deafness and urticaria) and Schnitzler syndrome (fever, joint/bone pain, monoclonal gammopathy and urticaria).
Pregnancy: pruritic urticarial papules and plaques of pregnancy(PUPP)
Physical factors: pressure (Immediate and Delayed pressure urticaria), cold, aquagenic (water exposure), heat, exercise and stress (cholinergic urticaria), sun exposure and vibration.
Emotional factors: psychological factors
Genetic factors: Hereditary angioneurotic oedema (HANO), characterized by recurrent attacks of angioedema (without urticaria) involving the skin, GI tract, respiratory tract, and mucous membranes. This disorder is an autosomal dominant hereditary disease caused by a functional deficiency of the C1 inhibitor protein.
Diagnosis: symptomatically, by laboratory tests, serology, allergy tests, autoimmune marker examinations, stool examination searching for ova and parasites. Serum cryoglobulins, C3, C4, C1-esterase inhibitor functional assays, etc.
Treatment: by administering antihistamines, corticosteroid and noradrenalin in case of anaphylaxis. In certain special cases by administering colchicine or dapsone, for patients with autoimmune urticaria methotrexate or cyclosporine can be effective.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances are: 291-293, 297-299, 324-327, 334-336, 344-346, 350, 370-377, 402-403, 408-410, 442-451, 456, 460-461, 475-479, 488, 534, 540, 560 kHz

17.8. Erythema Multiforme

Erythema multiforme is a characteristic allergic response of the skin and mucous membranes related to a number of different possible etiologies, including infectious agents such as *Herpes* or *other viruses*, *Mycoplasma pneumoniae*, and drugs such as penicillin, antipyretics, barbiturates, hydantoins and sulfonamides. The major pathology is an acute inflammatory infiltrate around the blood vessels; degenerative changes in the endothelial cells of the capillaries and a general allergic process. The lesions occur in a characteristic symmetrical distribution and favor the extensor areas of the distal parts of the limbs, the back of the hands, and the dorsum of the feet palms and soles are often involved, too. Oral lesions, i.e. blisters and erosions on the buccal mucous membrane, the gums and tongue, swelling and crusting of the lips are very often experienced.
Complications may occur f.i. a severe toxemia and prostration, fever, cough and inflammation of the lungs. The skin lesions are often characterized by a vivid redness gradually becoming duller, more indurate, together with pale and sometimes bullous centers. These iris like lesions are its characteristics.
Stevens-Johnson syndrome is a more severe form of the illness, which can progress even into a life threatening Lyell syndrome (Toxic epidermolytic necrosis) leading often to death.
Diagnosis: symptomatically.
Treatment: All drugs, food, chemical agents and infections, supposed to cause the symptoms should be eliminated. By administering antiallergic drugs, anti-inflammatory drugs and corticosteroids.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances are: 288-295, 318, 322-330, 342-349, 360-365, 372, 383, 398, 409, 413-420, 444-446, 477, 555 kHz
This list is not complete; the said numbers indicate only the most frequent and important resonances.
Checking for intestinal bacterial flora and worms, and the supplementation with friendly bacteria is advised.

17.9. Atopic Dermatitis

Atopic dermatitis (AD) is a pruritic disease of multifactorial origin that usually starts in early infancy and is typified by pruritus, eczematous lesions, xerosis (dry skin), and lichenification (thickening of the skin with increased skin markings). These patients have an inherited tendency to produce excessive antibodies of IgE type, in response to a number of different stimuli, f.i. food, chemicalies, viruses, bacteria, fungi, etc. AD is very often associated with other atopic diseases (i.e. asthma bronchiale, allergic rhinitis, urticaria) and with an acute IgE-type allergic reaction to certain foods, too. The worsening of the AD symptoms can be caused by many a factor, including emotional stress, changes in temperature and humidity and by contact with irritating substances. The prevalence of the illness is growing all over the world.

Pathogenesis: There are several cell types involved in the development of the illness, including (HTLV infected) T lymphocytes, eosinophils, Langerhans cells and keratinocytes as well as cytokines and IgE protein. An *imbalance of T_H2 cells* characterizes the acute process, and a *swing toward T_H1 cells* will occur in the chronic stages of the disease. Another factor may be the *defective barrier function* of the stratum corneum leading to the entry of antigens via the skin, provoking the production of various inflammatory cytokines.

Xerosis due to defective lipid (particularly ceramide) productions is known to be an associated problem in case of most AD patients. *Environmental antigens* f.i. got from food (involving the gut), dust mites (involving the lungs), and other factors that provoke the production of increased levels of IgE, and an increased histamine deliberation from mast cells. The genetic predisposition to react to various environmental allergens by a type I hypersensitivity reaction is usually superimposed with the mechanisms mentioned above.

The most frequent infectious agents associated with this disease are *HTLV, HBLV, Mycoplasma fermentans, Staphylococcus aureus* and *Streptococcus pyogenes*.

Symptoms: The primary signs of the disease can begin in infancy characterized by itchy dermatitis lesions locally or disseminated. Appearing in the first few months after their birth infants may develop red, oozing, crusted rashes on their face, scalp and diaper area, hands, arms, feet and legs. The affected skin area is xerotic, eczematous, lichenification may easily develop. The eczematous changes are seen at different locations, the morphology of the lesions are changing with age. In childhood AD the xerosis is often generalized, the skin being rough, lichenification caused by repeated rubbing of the skin mostly over the folds and bony protuberances is characteristic. The eczematous lesions are often exudative. Pallor of the face, erythema and scaling around the eyes are also common signs. Dennie-Morgan folds i.e. increased folds below the eyes are often to be seen. Flexural creases, (mostly the antecubital and popliteal fossae) and buttock-thigh creases are often affected. Excoriations and crusting are common getting worse by scratching.

In adulthood, lesions mostly affect the face causing a dry, scaling appearance. A diffuse brownish erythema of the whole skin is often to be seen, lichenification, xerosis are prominent. The pruritic, ekzematous lesions affect usually also the neck, the extensor and flexural regions involved already in childhood, can develop anywhere, though they spare the groin and axillary areas.

Complications: The *colonization* of the skin by *S aureus* is often experienced among children with AD. Scratching and rubbing can hurt the skin, opening the way for viruses, bacteria or fungi to cause *secondary infections*. Eczematous and bullous atopic lesions on the palms and soles are often infected with *beta-hemolytic group A Streptococcus*. A swollen, painful, red fingertip caused by *HSV* entering through a break of the skin is a herpetic whitlow, which occurs f.i. among health care workers after touching body fluids containing HSVs.

Kaposi varicelliform eruption *(eczema herpeticum)*, may be seen among AD patients usually occuring due to a primary, rarely due to a recurrent *HSV infection*. This severe, potencially even fatal disseminated vesicular disease can begin at any location, but particularly in areas of the atopic lesions. The virus spreads rapidly involving all eczematous areas and the healthy skin as well and the affected areas may become secondarily infected, too. *Vaccinia vaccine* given for the prevention of small pox might cause an *eczema vaccinatum* either due to the vaccination of the AD patients or their relatives. *Urticaria and acute anaphylactic reactions* to food (most often caused by eggs, milk, soya, peanuts, fish and seafood) can occur in increased frequency in patients with AD.

Differential diagnosis: by distinguishing it from seborrheic dermatitis, ichthyosis, contact dermatitis, psoriasis, scabies, etc.

Diagnosis: symptomatically and by personal and familial history of the patient. By testing to rule out immunodeficiency syndromes. In case of supposed allergy by specific antibody tests.

Treatment: symptomatically. By avoiding the contact with substances known to irritate the skin. By applicating emollients, corticosteroid creams, immunomodulator calcineurin inhibitor creams, by administering antihistamins etc. By administering probiotics in order to induce an immune response of T_H1-type instead of T_H2, thus to inhibit the development of allergic IgE antibody productions. In severe cases phototherapy (UV-A, narrow band UV-B, etc.), in most severe cases concerning adults the administration of methotrexate, azathioprine and cyclosporine may be of help. In case of eczema herpeticum the administering of acyclovir is effective, administering antimicrobal drugs if indicated.

RFR method: detects and may eliminate the pathogen microorganisms.

The most frequent resonances are: 290-293, 297-305, 311-315, 321-324, 330, 339-341, 348, 354-359, 360-365, 370-374, 380-382, 397, 403-410, 416-421, 424, 428-429, 442-453, 487-490, 494-497, 523, 530, 553-555, 557-558 kHz

17.10. Eczema and Dermatitis

Eczema (EZ) is the comprehensive term for several different types of dermatitis (skin inflammations). Eczema, a form of dermatitis, is an inflammation of the epidermis either with acute vesiculopapular rashes or with dry and recurring skin alterations and is characterized by one or more of the following symptoms: redness, skin edema (swelling), itching, dryness, crusting, flaking, blistering, cracking, oozing, or bleeding.

There are many a factors that can cause EZ including infections, irritating substances, allergens, genetic and psychogenic components. Eczematic lesions can also be caused by underlying immune diseases such as lymphoma, mycosis fungoides.

17.10.1. Contact Dermatitis

Contact dermatitis is caused by a cutaneous contact with a particular substance; this rash is confined to a specific area having clearly defined boundaries. Substances can cause skin inflammations by way of one of two mechanisms, i.e. irritation or allergic reaction. So that the contact dermatitis has two types:

17.10.1.1. Allergic Contact Dermatitis

Allergic contact dermatitis is characterized by a delayed type allergic reaction to certain allergens where the inflammation of the skin is manifested by erythema, edema and vesiculation of varying degrees. The most common causes of allergic contact dermatitis are f.i. cosmetics (e.g. fragrances found in perfumes, cologne wateres, aftershave lotions, deodorants, soaps, etc.), metal compounds in jewelry (e.g. nickel), poison and irritant

plants (e.g. poison ivy and poison oak, ragweed and primrose), medical drugs in skin creams (e.g. antibiotics, antiseptics, etc.), and chemicals used in clothing manufacturing.

17.10.1.2. Irritant Contact Dermatitis

In this case the inflammation of the skin is induced by chemicals, f.i. detegents, that directly damage the skin. This irritant type is the most common cause of the occupational skin diseases.

17.10.1.3. Phototoxic Contact Dermatitis

This type of dermatitis develops if a patient's skin contacts certain substances and this skin area is then sufficiently exposed to sunlight. In case of an associated allergy, the disease is named photoallergic contact dermatitis. Substances causing this disorder include sunscreens, aftershave lotions, certain perfumes, antibiotics, coal, tar, oils, etc. The permanent elimination of the causative substance and even its traces from the environment can cease the symptoms.

Diagnosis of contact dermatitis forms: symptomatically, by patch testing, by bacterial and fungal culturing, by virus examination with PCR.

Treatment: the cause of the contact dermatitis must be identified. Symptomatically, f.i. by topical corticosteroids, immunomodulators, Psoralen plus UV-A, immunosuppressive agents, such as azathioprine or cyclosporine, used in recalcitrant cases of severe chronic widespread allergic contact dermatitis.

RFR method: detects and may eliminate all the pathogen viruses, bacteria and fungi.

The most frequent resonances are: 296-297, 299-303, 324-327, 332-340, 368, 370-374, 384-386, 391, 397-399, 402-403, 411-415, 442-453, 464, 472-476 kHz

17.10.2. Xerotic Eczema

is the name of a disorder in case of which the patient's skin becomes seriously dry, cracked, itchy and eczematously inflamed. This problem affects mostly the limbs and trunk, is worsening in winter and occurs mostly among elderly people. Ichthyosis is a related disorder.

Discoid eczema is characterized by round spots of oozing or dry rashes with clear boundaries affecting mostly the lower parts of the legs and is usually worse in winter.

17.10.3. Seborrhoeic Dermatitis

The disease affects all sebum-rich areas i.e. the scalp, face and trunk. This dermatitis is linked to *Malassezia species,* to immunologic abnormalities (*HTLV* infected T cells), to the activation of the complement system as well as to environmental factors. A certain genetic predisposition has also a role in the etiology. The disease is aggravated by changes of the humidity, changes of the seasons, trauma f.i. scratching and emotional stress. Certain drugs, f.i. buspirone, chlorpromazine, cimetidine, ethionamide, gold, griseofulvin, haloperidol, lithium, methoxsalen, methyldopa, phenothiazines, psoralens, stanozolol, etc. may induce or worsen the symptoms.

Symptoms: The severity of seborrhoeic dermatitis varies from mild dandruff to exfoliative erythroderma. Itchy, dry or greasy scaling of the scalp and eyebrows, and a centrofacial dermatitis together with scaly pimples and red patches on the face are most often to be experienced. In more severe cases, yellowish scaly pimples appear also behind the ears, in the ear canal, on the bridge of the nose, around the nose and on the chest. Among newborns the most often sign is a thick, yellow crusty scalp rash and sometimes yellow scaling behind the ears and red pimples on the face.

Diagnosis: is based on the symptoms waxing and waning and by the distribution of its involvement, by fungal culturing in order to rule out tinea capitis. Some dandruff on a child's head represents a fungal infection more likely.
Treatment: by administering topical corticosteroids for short-term use. Skin involvement responds to antifungal creams and gels. Topical calcineurin inhibitors (pimecrolimus, tacrolimus), sulfur or sulfonamide combinations and propylene glycol can all be effective.
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequent resonances are: 299-304, 313-317, 324-327, 358, 370-376, 384, 391, 413-415, 422, 442-451, 453-464, 501-505 kHz

17.10.4. Dyshidrosis (Pompholix)

This type of ekzema occuring only on palms, the sides of fingers, soles and toes is an acute, recurrent or chronic relapsing form of the vesicular palmoplantar dermatitis. The illness is caused by various endogenous and exogenous factors. The etiology of dyshidrotic eczema is multifactorial. It is considered as *a reaction pattern* caused by many a factor concerning patients with a genetic predisposition to this form of skin reaction. Dyshidrotic eczema often is associated with atopy. The band 18q22.1-18q22.3 is the locus of the pompholyx gene in case of the autosomal dominant form of familial pompholyx. Serum total IgE levels are usually elevated. Occasionally, dyshidrotic eczema is the first manifestation of an atopic diathesis.
The provoking or exacerbating causes of its symptoms can be local and systemic infections (mostly *bacterial* and *fungal* infections, caused f.i. by *dermatophyton species*), exogenous allergens (eg. contact dermatitis to nickel, cobalt; sensitivity to ingested metals; balsami etc.) emotional stress and environmental factors (f.i. hot or cold temperatures, humidity).
Symptoms: an itchy vesiculous dermatitis accompanied often by the thickening and cracking on the palms, fingers, soles and toes usually worsening at night and in warm weather. The blisters get scaly, red and oozing. Chronic distant fungal infections (f.i. onychomycosis caused by *Trichophyton species*) may cause palmar pompholyx as an id reaction. The often found associated co-infections are caused by *HTLV, Staphylococci, Borrelia B.l.s. and Mycoplasma species*. A dyshidrotic eczema may develop as an immune reconstitution inflammatory syndrome among *HIV*-positive patients shortly after being treated with highly active antiretroviral drugs.
Diagnosis: symptomatically, by bacterial and fungal culturing in oder to exclude secondary infections, by blood tests for IgE and by patch testing. By biopsy histological analysis, (which shows spongiosis, an epidermal lymphotic infiltrate and intraepidermal vesicles not associated with sweet glands.)
Treatment: the elimination of all provoking factors can heal the skin disease. Symptomatically, using mostly local corticosteroid preparations. In case of a severe refractory pompholyx systemic corticosteroid, azathioprine, methotrexate mycophenolate mofetil, cyclosporine and etanercept may be of help.
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequent resonances are: 294-305, 317-319, 321, 361-365, 370-374, 380-386, 391, 397-398, 407, 413-418, 422, 442-453, 464, 470-476 kHz

17.10.5. Varicous Vene Eczema

Varicous vene ekzema, particularly common at the ankle area of people over 50, can occur among people with an impaired venous circulation of the lower extremities having varicous veins and leg edema.
Its **Symptoms** are an itchy, red, scaly, often vesiculopapulous dermatitis together with the darkening of the affected skin area. The disorder often affects the area of leg ulcers superinfected with fungi and other skin pathogens.

17.10.6. Stasis Dermatitis

can be a chronic red, scaling dermatitis accompanied by a swelling inflammation of the lower legs, which often ends in a dark brown skin. Stasis dermatitis results from the pooling of blood and fluid under the skin caused by chronic heart damages and by over weight; and, it tends to occur thus also among people who have varicose veins and local lymph damages. Infections do frequently cause complications, sometimes even severe skin damages can occur.

17.10.7. Neurodermatitis (Lichen Simplex Chronicus)

This skin disease, also named localized scratch dermatitis, causes usually only one itchy area of a thickened, pigmented circumscribed dermatitis resulting from habitual rubbing and scratching. The cause of neurodermatitis is known to be multifactorial: f.i. genetic predisposition, lymphocyte damage caused by *HTLV infection* and *other viral, bacterial and fungal infections,* psychosomatic and mental effects, allergies, etc. In certain cases, neurodermatitis can develop associated with other skin conditions such as dry skin, eczema or psoriasis and *fungal* skin infections. Stress and anxiety can trigger and make the itching worse, too. Women of 50-60 are most often affected.

Symptoms: The most often affected loci are thw neck, wrist, forearm, thigh and ankle. The disorder can affect sometimes the genital areas, i.e. the vulva or scrotum as well. Neurodermatitis can begin if something simply rubs or irritates the skin, f.i. a tight clothing or a bug bite. By rubbing or scratching the area, it gets more itchy, the more one scratches, the more it itches. The patches will become raw, red or darker. There also exist a suspicion of obsessive-compulsive behaviors leading to this form of dermatitis.

Diagnosis: is based on symptoms and on the patient's history concerning his scratching. By biopsy, blood tests and lab studies, etc.

Treatment: by breaking up the itching-scratching cycle. The effectiveness depends on the identifying and eliminating all factors aggravating the problem, on stopping the scratch by modifying his/her behavior. Hereafter local anti-inflammatory medications, f.i. over-the-counter and prescription creams can help, though after the scratching being stopped, it can take months until the skin area gets normal.

RFR method: detects and may eliminate all pathogen viruses, bacteria and fungi.

The most frequently found resonances are: 315-317, 325-328, 331, 337-340, 370-374, 378-379, 396-397, 402-403, 457-459, 470, 540-541, 560-564 kHz

17.10.8. Autoeczematization (Autosensitization, Id Reaction)

This process is a generalized eczematous reaction of the skin to an infection caused by viruses, bacteria, fungi and parasites. The appearance varies depending on the cause and is always occuring some distance from the original infection. It is completely curable by eliminating the causative pathogens. The microbial antigen can attach itself to certain skin structures, changing thus its antigen structure. T cells responding to self antigens do also exist in normal tissues. Autoreactive T cells (i.e. *HTLV* infected ones) can be specifically deleted when they develop in the thymus; though those that survive and reach the peripheral tissues are kept mitotically quiescent and are otherwise unresponsive by stimuli from other immune cells. An autoimmune disease arises if clones of these autoreactive cells overcome the usual safeguards, expand, and become activated. Certain findings suggest that some allergic diseases arise due to intrinsic defects in $CD4^+$ T cells, leading to Th2-skewing and hyperreactivity to antigens. This defect leading to hyperractivity may be caused by a *HTLV* infection. If the autoimmune manifestations are prominent, children will

suffer fromsevere allergic eczema, will have elevated IgE levels, eosinophilia and food allergy.
This type of eczema occurs by autosensitization due to the stimulus given by the person's own cells of altered epitopes caused by the substance taking part in the allergic reaction. Patients can suffer from coin-shaped eczema, contact dermatitis, chronic ulcers, the incidence of hematoma, from an improper or excessive physical or chemical stimulation, bacterial infection, which factors may all cause self-sensitiveness. Autoeczematisation can occur at any age but most often among middle-aged and elderly people.
Autoimmune eczema is a T-lymphocyte dependent process, in case of which the T-lymphocyte is altered by pathogens. In case of autoimmune eczema *Mycoplasma species, Borrelia B.s.l. species* and other viruses, bacteria and fungi may all prove to be pathogenetic factors. One can mostly find resonances of these microorganisms among patients suffering from autoimmune eczema.
Treatment: symptomatically, using skin creams containing f.i. salicylic acid, sulfur and corticosteroids, by administering antihistamines, corticosteroids and by phototherapy.
RFR method: detects and may eliminate the viruses, bacteria and fungi.
The most frequent resonances of the altered pathogens are: 297-299, 311-315, 321, 330, 339-341, 354, 359, 365, 370-374, 382, 397, 416, 426-428, 432-433, 439, 442-451, 453-455, 459-464, 476-479, 482, 487-490, 493-497, 525-530, 574 kHz
After eliminating the pathogen microorganisms, the symptoms will decrease and the dermatitis can completely disappear.

17.10.9. Nummular Dermatitis

Nummular dermatitis is also a form of eczema. It is characterized by round-to-oval erythematous plaques mostly to be found on arms and legs though may be widespread, too. Lesions start by being papules or vesiculopapules, containing serous exudate, which later on grow into plaques, may ooze and form crusts. The alterations are usually very pruritic and are frequently accompanied by xerosis.
Its etiology is multifactorial, but there is not much known about the pathophysiology of the disease. Autoeczematization may account for the presence of the multiple plaques. There exist theories and some proved cases about the role of a delayed type sensitivity to certain materials f.i. sensitivity to nickel, cobalt, or chromates, colophony, nitrofurazone, neomycin sulfate, potassium thioglycolate, etc. Nummular eczema was found in association with *giardiasis* and other gastric and bowel infections, caused f.i. by *Helicobacter pylori, Hepatitis C* and *Staphylococcus aureus.*
Diagnosis: symptomatically, by examinations with specific blood tests (RAST) or (PRIST), patch tests, skin biopsy, etc.
Treatment: symptomatically, by rehydration of the skin, by treating every infection, and by reducing the inflammation with local and systemic antiinflammatory drugs, by administering sedative drugs, antihistamines and corticosteroids in order to control and suppress the symptoms. Topical immunomodulators can also help. In most severe cases the administering of immunosuppressant drugs (cyclosporine, azathioprine and methotrexate) can be effective.
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequent resonances are: 324, 327-329, 331, 345, 350, 370-374, 377, 380-381, 397, 402, 434, 442-451, 456, 462, 475-482, 491, 537-540, 557-559, 560-567 kHz

17.10.10. Autoimmune Progesterone Dermatitis

Many women complain of the worsening of their acne and water retention during their menstrual cycle, though some actually have autoimmune progesterone dermatitis, a condition in which the menstrual cycle is associated with a number of skin disorders, such

as urticaria, eczema, etc. Among affected women, the skin symptoms occur typically three to ten days prior to the menstrual flow and generally cease two days after the beginning of menstruation. The mechanism of this autoimmune skin disorder triggered by progesterone is similar to that mentioned in [redacted] According to my opinion, a certain, perhaps symptomless infection does also play an important role in the pathogenesis of this disease. The fact, that the disorder is not continuous, may support my opinion.
Diagnosis: symptomatically, by auto-antibody examinations, by checking non manifest infections, by PCR, by examining blood eosinophilia and cytokine levels.
Treatment: symptomatically. Progesterone may sometimes be part of the therapy planned.
RFR method: detects and may eliminate the pathogen microorganisms.

17.11. Skin Photosensitivity, Sun Allergy and Photoallergy

Sunburn and sun damages take usually a certain amount of time to manifest. Some people have unusual skin reactions even if they were for but a few minutes exposed to the sun. These reactions can be redness, peeling, hives, blisters and thickened, scaling patches. Many a factor may contribute to sun sensitivity. Widespread use of certain drugs causing photosensivity renewed the interest as to the reactions of the human skin to light. Drugs, f.i. phenothiazine and tetracycline derivates; some other antibiotics and diuretics, can all alter the cutaneous reaction to sunlight. Photosensitivity reactions can be caused by soaps, perfumes, f.i. scented toilet water, coal tars used for the treating of dandruff and eczema, and certain substances found in plants, f.i. meadow grass and parsley. Some *microorganisms* can sensitize to light. Exposure to sunlight is an significant cause of basal cell carcinoma and Squamous Cell Carcinoma developing on parts of the body exposed to sun. Sunlight causes discomfort and photosensitivity reactions concerning patients with certain types of porphyria, especially those with erythropoetic protoporphyria. Degenerative and neoplastic conditions associated with solar radiation include basal cell carcinoma, Squamous Cell Carcinoma, malignant melanoma, solar keratoses and chronic sun-induced skin degenerations. Photosensitivity related to drugs, and photosensitivity related to increased plasma levels of photosensitizing porphyrins can occur among patients suffering from all types of porphyria excepting acute intermittent porphyria.
Electromagnetic emanations of the sun comprise a wide range of radiation including the electric waves, radio waves, infrared rays, visible light, ultraviolet light, roentgen rays, gamma rays and the secondary cosmic rays. The amount and type of solar radiation reaching a given part of the earth at any specific time is determined by a great variety of factors, such as the latitude, time of day, season, altitude, local atmospheric conditions, variations in the thickness of the ozone layer, and height of the sun above the horizon.
Photoallergy to drugs or microorganisms can be considered to represent an acquired, altered capacity of the skin to respond to light energy in the presence of a photosensitizer. Presumably, this condition is dependent on an antigen-antibody reaction or a delayed hypersensitivity response mediated by mononuclear cells. The absorbed energy of light seems to promote a photochemical reaction between the drugs or microorganism products and the proteins of the skin; these molecules act as a haptenic group: they are either combined directly with the protein to form a photoantigen, or are altered by the absorbed energy. This altered haptenic group becomes an antigen.
Diagnosis: by photopatch tests.
Treatment: symptomatically, by administering anesthetics, corticosteroids and histamins locally and systemic as well.
The most frequent resonances are: 322-328, 336, 349, 357, 361-365, 378-388, 399 kHz
This list is not complete; it only contains the sensitivity factors of the microorganisms.

17.12. Discoid Lupus Erythematodes

Discoid lupus erythematodes (DLE) is a chronic recurring disorder characterized by well defined, round, erythematous (red) alterations of the skin, usually confined to the face, neck, arms and the scalp. Scaling, keratotic plugging, telangiectasia and skin atrophy are characteristic. If the untreated lesions subside, deep scars can remain.

The characteristic erythematous skin lesions may persist or come and go for years. The appearance of the affected skin will change over time, being at first red, round and approximately 2-4 cm in diameter, scarring comes but later on. The scarring lesions of the scalp cause irreversible hair loss. This discoid type of lupus erythematosus is but seldom associated with systemic symptoms in contrast to the subacute cutaneous lupus erythematodes and SLE, where autoantibodies and autoimmun processes are being present. The skin is photosensitive in case of DLE, so that sun exposure worsens the symptoms.

Treatment: by using local antiinflammatoric and corticosteroid preparations, sunprotector preparations of an effect of 40 SPF.

RFR method: detects and may eliminate the pathogens.

The most frequent resonances are: 318, 324, 339-341, 359-368, 389, 394-398, 407-411, 416, 433, 442-452, 462, 471-474, 482, 492-500, 508-513, 525, 541-544, 553, 557-560 kHz

17.13. Generalized Pruritus and Chronic Anal Itching

Generalized and local pruritus (i.e. itching) is a frequent and very important differential diagnostic problem for the general physician. An intense generalized pruritus can be the only symptom of many a patient. The degree and quality of pruritus is but seldom characteristic concerning the causative disease, f.i. the itching, felt in case of an obstructive biliary disease is similar to that one felt in case of a lymphoma. Therefore, the clinician must rely on the anamnestic data, the physical examination, and the laboratory studies in order to establish the origin of the itching. The psychogenic pruritus, which is a reaction to stress and strain is one of the most important causes of pruritus. This type of pruritus often affects the skin of the scalp, and may be associated with other sensory complaints such as a bitter taste in the mouth or burning of the tongue.

Older persons with a dry skin may have generalized pruritus unrelated to any multisystem disease.

Pruritus in case of hepatic diseases has no specific qualities. A generalized pruritus may frequently be the first sign of biliary cirrhosis, and may occur even months before the onset of jaundice. Itching may also be the first sign of lymphoma, and, rarely, of carcinoma as well. Pruritus may have a sudden onset, and may be very intense just from the beginning.

Dermatological disorders in which itching is a characteristic sign include scabies, Dermatitis Herpetiformis Duhring, lichen ruber planus, urticaria, mycosis fungoides, insect bites, eczematous dermatitis and atopic dermatitis.

A subtle and important cause of general pruritus may be, even without visible rashes, a reaction to drugs, such as aspirin, quinidine derivates, and most especially opiates and their derivates.

The receptors for the itch stimuli reside in the papillary layer of the dermis, but there are no specific end organs for itching. Itching is a sensation carried principally by unmyelinated, slowly conducting fibers of the C groups to central neural pools in the spinal cord. Many itching skin disorders require certain dermatologic knowledge, and particularly biopsy of the skin, in order to establish an exact diagnosis.

Chronic anal itching is a symptom of many different illnesses. Localized scratch dermatitis (i.e. lichen simplex chronicus, neurodermatitis) is a chronic, itchy inflammation on the top layer of the skin (see Chapter 17.10.7). Itching, perianal inflammatory lesions may be

associated with inflammatory bowel diseases or with diverticulosis. Anal fissures are superficial erosions of the anal canal, usually healing rapidly thanks to a conservative therapy. Anal ulcers are usually chronic and deep, and occur together with painful spasms of the external anal sphincter during and after defecation. Bleeding may occur caused by fissures or ulcers, the healed ulcer is often associated with a hypertrophic anal papilla and an anal contracture. The fistula, a chronically inflammed canal made up of fibrous tissues surrounding the granulation tissue, the lumen of which may be difficult to find. Perirectal abscesses often contain purulent materials escaping from the rectosigmoidal bowel part into the anal area. Diverticulitis, Crohn's disease [See Chapter 13.5.1] colitis, a previous anal or rectal surgical therapy may also be their cause. Fistulas between the rectum and vagina and between the rectum and bladder represent serious complications of granulomatous, septic, or malignant disorders, requiring the patient to be hospitalized for definitive diagnostic and therapeutic procedures.

The most frequent important causes of anal itching (pruritus ani) are as follows:

1. Allergic reactions, such as contact dermatitis caused f.i. by anesthetic preparations applied to the skin, various ointments, or chemicals used in soaps.
2. Certain food such as spices, citrus fruits, coffee, beer and cola.
3. Certain persisting bacterial or fungal infections
4. Parasitic infections caused f.i. by pinworms (Enterobius vermicularis) or hookworms (ancylostomiasis, onchocerciasis), and, less commonly, from scabies or lice infestation (pediculosis) [See Chapter 9]
5. Antibiotics may cause anal itching if the patient has a fungal infection or is allergic to antibiotics.
6. Certain diseases such as diabetes mellitus, cirrhosis, jaundice, uremia, hyperthyroidism, anal disorders (e.g. skin tags, cryptitis, or draining fistulas), and cancers (e.g. Bowen's disease) [See the specific Chapters]
7. Generalized and severe pruritus occur f.i. in case of Hodgkin's disease. Pruritus may be the only systemic symptom of Hodgkin's disease, especially concerning young women [See Chapter 26.3.5.1.]
8. Chronic pruritus, such as that associated with chronic biliary tract disease and lymphoma, may lead to generalized brown hypermelanosis.
9. In case of polycythemia vera, pruritus occurs particularly after bathing.
10. Poor hygiene that leaves irritating feces, excessive rubbing, and use of soap are frequent causes of anal itching.
11. Warmth and excessive sweating caused by wearing pantyhose, tight underwear, as well as the person's obesity, hot weather may also cause anal itching.
12. Periods of emotional stress can cause general pruritus and anal itching.

Diagnosis: by physical examinations, by discussion aimed at differential diagnosis (cutan allergic reaction examinations, bacterial and fungal microbiological examinations, lab tests, serology etc.).

Treatment: causal treatment (treatment of the basic process) and symptomatic treatment, such as corticosteroid creams, emollients, or other topical treatments. Hypnotic and sedative drugs and antihistamine products can also be used.

RFR method: can be a causal treatment. It is advised to search for pathogen fungal, bacterial and viral resonance frequencies [See the specific Chapters] The RFR method is not suitable for clinical diagnosis. After a clinical diagnosis has been established, the RFR method may be used if the clinical treatment proves to be ineffective.

If the clinical examination is negative, measure these frequency resonances: 291-293, 372, 384-387, 389-390, 416-422, 448-451, 476, 488-492 kHz

Check the in case of allergy most frequently found resonances: 324-327, 336, 372, 378, 396, 402-403, 448-451 kHz

In case of jaundice [redacted] check the following resonances: 332, 383-395, 408-411, 450, 454, 477, 508, 544-546, 592 kHz
If measurements correspond with any of these frequencies, apply RFR method to eradicate the pathogen microorganisms.

17.14. Lichen Ruber Planus

Lichen (ruber) planus (LP) is an immune-mediated, chronic mucocutaneous disease characterized by pruritic, eruptive papules affecting the skin and the oral mucosa. This illness presents itself as brownish-red papules, lesions or rashes of polygonal shape sometimes with a fine scaling. LP affects mostly the flexor surfaces of the upper extremities, the genitalia and the mucous membranes.
The pathology of this illness is based on a genetical predisposition (HLA-B7, HLA-DR1 and HLA-DR10) and on a special infection caused f.i. by *Mycoplasma fermentans, HTLV, HPV* and *Hepatitis C virus*. The causative agent can, in some rare cases, be even certain drugs activating lichen planus. Secondary to these, an infection got from *Candida albicans* or from other *fungi* is frequently observed. Langerhans cells process Mycoplasmal antigens, which are then presented to T lymphocytes. The stimulated, HTLV-infected lymphocyte group is epidermotropic attacking keratinocytes. During this process, the keratinocytes release cytokines that attract lymphocytes. This process has been referred to as lichenoid tissue reaction.
The microscopic appearance of lichen planus is pathognomonic concerning the illness: hyperparakeratosis with the thickening of the granular cell layer, the development of a "saw-tooth" appearance of the rete pegs, the degeneration of the basal cell layer and the infiltration of inflammatory cells into the subepithelial layer of the connective tissue.
Symptoms: Lichen planus is an itchy, sometimes recurring papulous disease, forming groups of discrete red bumps at the beginning that later on can form rough plaques. The most frequently affected skin areas are the mouth, the flexor sides of the wrists, legs and ankles, as well as the lumbal and genital region, though it can be also disseminated everywhere. Similar rashes can occur among people if they are exposed to drugs containing gold, bismuth, quinine derivates, quinidine derivates, or quinacridin derivates, as well as if they are exposed to certain chemicals that are used to develop color photographs. Lichen planus may be the body's response to some external chemical substances or to certain pathogens, such as *Hepatitis C virus, etc.* The first episode may begin gradually or suddenly and can persist for weeks or months. Although lichen planus usually heals by itself, the patches nevertheless might return and the said episodes may recur year for year.
The mouth sores of lichen planus are particularly vexing; usually bluish-white and may be linear. Mouth sores appear often before the appearance of skin rashes, are usually painless, though the deeper sores may be painful. Occasionally, lichen oris plaques may become cancerous.
Its genital involvement is common among men. Typically, an annular configuration of papules can be seen on the glans. Less often, linear white striae, similar to the lesions on the vulva and the vagina, can be seen on the male genitalia. Vulvar involvement can range from reticulate papules to severe erosions. A burning sensation and pruritus are common. Vulvar and urethral stenosis can develop. Its vulvar lesions may also be associated with squamous cell carcinoma.
Diagnosis: symptomatically, by skin biopsy.
Treatment: symptomatically. Drugs and chemicals that may cause lichen planus should be avoided. By administering antihistamines, corticosteroids applied to the skin, or given orally, etc.
RFR method: detects and can eliminate the pathogen microorganisms.

The most frequent resonances present are: 324, 336, 340-345, 456, 475-479, 541, 561 kHz, and there may be found different HTLV and HPV frequencies as well (See their special Chapters).

17.15. Pemphigus Vulgaris and Bullous Pemphigoid

Bullous pemphigoid and pemphigus vulgaris are chronic autoimmune diseases, affecting primarily adults. Pemphigus has serious consequences for the patient. These two disorders can be distinguished by biopsy and by using immunofluorescence techniques. Although the cause of these diseases are unknown as yet, drugs can be provoking factors. There are data of the significant association between these autoimmune diseases and the development of certain tumors.

Pemphigus is a rare, sometimes fatal autoimmune disease, in case of which flaccid intraepidermal blisters of varying size appear on the skin, on the lining of the mouth, the vagina, on the thin covering of the penis, and on other mucous membranes. Pemphigus has four different forms, and the most important one among them for the general physician to be recognized is pemphigus vulgaris. This disease affects primarily middle-aged persons, particularly those between forty and seventy.

Symptoms may begin in the nasal or oral mucous membranes. The clinical lesions appear even from the beginning on as flaccid bullae. The denuded areas that form at the loci of the ruptured bullae increase in size as the epidermis detaches. Secondary infections can cause serious problems. As regards nearly every patient oral and nasal mucosal lesions also occur, and more than half of them have lesions on the mucous membranes of the mouth as the first manifestation of the disease. Unless immunosuppressive agents or steroids are given, this disease can lead to death.

Bullous pemphigoid is a subepidermal, blistering, autoimmune skin disease rarely involving the mucous membranes. It can be clinically a relatively benign disorder of limited duration, but if untreated, it may last from months to years with periods of remissions and exacerbations proving to be a severe disorder. Drugs known to be associated with BP are ibuprofen, other NSAIDs, captopril, furosemide, penicillamine, antibiotics, etc.

Diagnosis: symptomatically, by biopsy and by light and immunofluorescence microscopy by testing of specific antibodies in the serum; by specific intercellular antigen examinations of the skin.

Differential diagnosis: By distinguishing them from each other. Grouped vesicles occur in Herpes Zoster Virus and Herpes Simplex Virus-1 and 2, scattered discrete vesicles occur in case of varicella.

Treatment: by administering corticosteroids and immunosuppressive drugs.

RFR method: detects and eliminates the pathogen viruses.

The most frequent resonances are: 287-295, 340-361, 415-420, 455-461 kHz

17.16. Vitiligo

Vitiligo is an aquired skin disorder in which the loss or the dysfunction of melanocytes causes smooth, whitish depigmented patches on the skin. Vitiligo may be either localized or generalized. If localized, the hypomelanosis of the skin and hair may be restricted to but one region, f.i. the anogenital area, scalp, or hands. If generalized, the pattern of hypomelanosis is typical, with lesions occurring particularly on the face, axillae, neck, hands and extremities, and with pigment loss in the hair. Idiopathic vitiligo is fairly common, it affects 1-2 percent of the population. The lesions are completely lacking in pigment; its snow-whiteness is distinctive and often serves to differentiate vitiligo from

other hypomelanoses. Vitiligo is believed to be inherited as an autosomal dominant trait with irregular penetrance. In the majority of the cases, vitiligo is idiopathic, but typical vitiligo, as said before, is known to occur with a variety of other diseases, such as Addison's disease, hyperthyroidism, hypoparathyroidism, pernicious anemia, physical trauma, diabetes mellitus and alopecia areata. Leukodermia can be caused by certain microorganisms; which are mostly melanicidal fungal infections. All of these disorders are believed by some researchers to be caused by autoimmunity. The changes are most striking in darkly pigmented people. Just like in case of albinism, the unpigmented skin is extremely prone to sunburn. The areas of the skin affected by vitiligo may also produce white hair owing to the melanocytes, which may disappear from the hair follicles.
Diagnosis: macroscopic examinations and electron microscopic studies of the idiopatic vitiligo of the skin reveal a marked reduction or, more commonly, a total absence of detectable melanocytes.
Differential diagnosis: by distinguishing it from other hypomelanotic states.
Treatment: symptomatically. Sunscreen and the covering of the affected areas can protect against sun exposure to prevent sunburn. 5% beta-naphtolum and potassium soap cream are usually used.
RFR method: detects and eliminates the microorganisms.
The most frequent resonances are: 307-309, 332-334, 371-373, 401-403, 450-454, 537-542 kHz
After treatment avoid the tanning of white skinned people.

17.17. Hyperpigmentation

Melanin is the principal pigment colouring the human skin, hair and eyes. Its primal function is to shield the dermis from the deleterious effects of solar radiation. The melanin content of the skin and hair can alter in case of a number of diseases, which irregularities of the pigmentation may provide important diagnostic clues.
Tyrosine is the precursor of melanin, secreted into the epidermal space by melanocytes, which dendritic cells behave as unicellular exocrine glands. Melanocytes are situated in the dermoepidermal interface, in hair bulbs, in the uveal tract, the retinal pigment epithelium, the inner ear, and the leptomeninges. The melanocyte system constitutes a cytological, functional and biochemical unit. The melanocytes in all these locations derive from the natural crest, and can hydroxylate tyrosine into dopa and, ultimately, into the pigment tyrosine-melanin.
If the skin is exposed to sunlight, the melanin production will increase and cause tanning. Increased amounts of melanin, i.e. hyperpigmentation can be a response to hormonal changes, occuring f.i. in case of Addison's disease, pregnancy as well as in case of using oral contraceptives, The skin can darken also in case of certain other diseases such as hemochromatosis and hemosiderosis or due to certain medications if applied on the skin, swallowed, or injected.
Moles are small, usually hyperpigmented skin growths that develop from pigment producing cells in the skin. Some moles closely resemble malignant melanoma, which a cancer of the pigment-producing cells in the skin, and can only difficultly be distinguished. Noncancerous moles can develop into malignant melanoma.
Atypical moles are flat or raised dark skin growths, they can be bigger than ordinary moles and are not necessarily round. They vary in color from tan to dark brown, usually on a pink background.
General hyperpigmentation i.e. hypermelanosis can be caused by genetic, metabolic, nutritional, endocrine, chemical and physical factors, as well as by inflammation and infections, miscellaneous and neoplasmal factors.

Acute severe inflammations of the skin caused f.i. by sunburn and by various infections of the skin f.i. fungal, bacterial, viral infections can cause transient hyperpigmentation. Chronic inflammations caused by skin diseases such as lichen planus, lupus erythematosus, atopic dermatitis, psoriasis, pinta, acrodermatitis chronica atrophicans, localized scleroderma can heal with hyperpigmentation. Certain disorders such as hepatic disorders, Whipple's disease, erythema dyschromicum perstans, acanthosis nigricans are also associated with hyperpigmentation. (Regarding lentigo and melanoma, see the special Chapters.)

Diagnosis: symptomatically and by skin biopsy.

Treatment: by eliminating the causative factors or by healing the original disease.

RFR method: detects and may eliminate the different pathogen microorganisms.

As to the frequencies, see the special Chapters.

17.18. Porphyria Cutanea Tarda

Porphyria cutanea tarda (PCT) is the most commonly occurring form of porphyria, characterized by liver damages, hypertrichosis and photosensitive dermatitis, showing blistering inflammations and the hyperpigmentation of the skin. The skin is usually sensitive to light and mechanical trauma. The blisters appear on the light-exposed skin areas, ulcerate frequently and lead to scar formations. The skin lesions are indistinguishable from those observed in porphyria variegata, but abdominal pain and neurologic complications are absent. The photosensivity is similar to that of the erythropoietic uroporphyria. Excessive hepatic synthesis and uroporphyrinuria are characteristic for PCT. In case of this hepatic porphyria, the uroporphyrinogen decarboxilase, an enzyme in the liver, necessary for heme synthesis, becomes inactive. Contributing factors include an infection with Hepatitis C virus, iron deficit, chronic alcohol toxicosis, hexachlorobenzene toxicosis and polychlorinated phenol-derivate liver toxicosis, high estrogen level, and genetic factor leading to Bantu cirrhosis in South Africa or certain other hepatic parenchymal iron overload. Chronic Hepatitis B virus infection causes liver damage and enzyme abnormalities. Porphyrins are responsible for the photosensitivity of the skin in case of PCT.

Diagnosis: serum, urine and stool examinations for porphyrins. In case of PCT the level of porphyrins in urine and stool is increased.

Treatment: by phlebotomy, and iron removal

RFR method: detects and may eliminate the Hepatitis C virus.

The most frequent resonances of Hepatitis C virus are: 324-339, 350-352, 370-374, 396, 400-402, 450-456, 475-482, 540-541, 559-563 kHz

17.19. Psoriasis

Psoriasis is an autoimmune skin disease of recurring tendency, liable even to become chronic, recognizable by silvery scaling papules, various-size plaques and raised patches.

It is a noncontagious, inflammatory disorder, based on a genetic predisposition, manifesting itself in case of combined viral, mycoplasmal and bacterial infections. This skin disease is often associated with psoriatic arthritis, less often with different allergic or autoimmune processes occuring in certain organs, f.i. the eyes.

The inflammatory mechanism present in the psoriatic skin lesions is immune based, initiated and maintained primarily by T cells in the dermis, increasing the turnover rate of the epidermal cells from the normally 23 days to 3-5 days. This abnormally high rate of growth and turnover of the keratinocytes leads to the scaling of the affected skin lesions.

Genetic predisposition and autoimmune-allergic mechanisms are thought to play a role in its *pathogenesis.*

The genetic predisposition of the affected families often leads to the manifestation of the illness. HLA-B13, -B17, and -Cw6 haplotypes are often associated with plaque type psoriasis. Many families appear to exhibit an autosomal dominant pattern of inheritance with decreased penetrance. There were several putative genetic susceptibility loci identified, including psoriasis susceptibility 1 (PSOR1) locus on chromosome 6, which is associated with up to 50% of the cases. Six other psoriasis susceptibility loci (PSOR2, PSOR3, PSOR4, PSOR5, PSOR6, PSOR7) were also found, as well as the transcription factor *RUNX1*. Though these findings point certainly to genetic factors, the absence of a 100% concordance among monozygotes suggests that environmental factors must also play a role in the pathophysiology of this disease, moreover, multifactorial inheritance mechanisms and etiologies without any genetic component can not be excluded as yet.

The immune-autoimmune inflammatory processes of this illness are initiated and maintained primarily by *HTLV* infected T cells in the dermis. T cells (which normally help to protect the body against an infection) if getting infected by *HTLV* they will become hyperactive and release cytokines (in particular tumor necrosis factor-alpha) causing inflammation and leading to a rapid production of skin cells. HTLV infections decreasing the number of CD4+ T cells and leading thus to the overactivity of the CD8+ T cells, will worsen the psoriatic illness. *HTLV* can directly enhance the proliferation of the keratinocytes as well.

Langerhans cells, which are antigen-presenting cells in the skin, migrate from there to the regional lymph nodes, where they will interact with these *HTLV*-infected T cells. The presentation of *mycoplasmal* and/or other antigens to T cells, as well as a number of co-stimulatory signals will trigger an immune response with T-cell activation and the release of certain cytokines, f.i. tumor necrosis factor alpha leading to inflammation and to cell-mediated pathological immune-autoimmune responses (see the Chapters of mycosplasma and HTLV), and induce the epidermal hyperproliferation observed in psoriatic persons as well as a deregulated inflammatory process with an enhanced production of various cytokines. The excessive reproduction of skin cells is thus a secondary phenomenon of the immune response.

The most frequent infective agents activating psoriasis are: *HTLVs, HIV, mycoplasmas* such as *Mycoplasma fermentans, Mycoplasma penetrans, group A Beta-hemolytic streptococcus, Leishmania species* such as *L. brasiliensis, L. tropica,* and fungi, f.i. *Trichophyton species*, etc.

Psoriatic skin diseases are activated mostly by pharyngeal *streptococcal* infections. *HIV* and other *Human T-cell Lymphotropic viral* infections are always present at the manifestation of this illness, while *mycoplasmas* are usually pathogen coinfective agents in psoriasis.

The mycoplasmal superantigen can cross-react with the dermal collagen and modify the function of lymphocytes as well.

Local factors: all types of trauma f.i. physical, chemical, electrical, surgical, infective, and inflammatory injury types can be associated with the development of a plaque type psoriasis. Excessive scratching can aggravate and provoke localized psoriatic skin lesions. The Koebner phenomenon, also named isomorphic response, refers to the appearance of lesions along a site of injury, f.i. the eyebrows, armpits, the navel and the groin are often affected with psoriatic skin lesions in case of a local mycotic process.

Psoriasis can thus be manifested due to very different combinations of factors such as genetic predisposition together with HTLV or other viral and/or bacterial (f.i. HSV, Borrelia B. sensu lato, Streptococcus, Leishmania, etc.) infections, or f.i. with allergic factors of ascaris and taenia antigens. Taenia may cause a specific skin allergy, but certain other antigens of the worm may also start allergic processes. Cutaneous leishmaniasis may be present as a single or multiple chronic ulcer, as destructive lesions, or if being a

disseminated infection, it manifests itself together with other pathogens as a typical psoriasis.
Different chemicals and medicines (f.i. beta blockers) also can stimulate the formation of psoriatic plaques.
Symptoms: Psoriasis typically involves the scalp, the elbows, the knees, the lumbosacral area and the intergluteal clefts.
Plaque psoriasis (psoriasis vulgaris) is the most common form of psoriasis. It typically appears as raised areas of inflamed skin, covered with silvery white scaly skin. Plaque psoriasis is most typically characterized by circular-to-oval red plaques distributed over the extensor body surfaces and the scalp. The scaling of the plaques results from epidermal hyperproliferation and dermal inflammation. The extent and duration of this form of disease is highly variable. It can develop into a more severe form, such as into pustular and erythrodermic psoriasis.
Psoriatic arthritis involves the joints and the connective tissue and causes their inflammation. It can affect any of the joints, but mostly the joints of the fingers and toes resulting in a sausage-shaped swelling of the fingers and toes known as dactylitis. Psoriatic arthritis often affects also the hips, knees and the spine (spondylitis). About 15% of psoriatic people also have psoriatic arthritis. Psoriatic arthritis is held to be a seronegative spondyloarthropathy occuring therefore mostly among patients with tissue type HLA-B27. The treatment of psoriatic arthritis is similar to that of rheumatoid arthritis. More than 80% of patients with psoriatic arthritis have psoriatic nail lesions characterized by the pitting of the nails, or even by the loss of the nail (onycholysis).
Erythrodermic psoriasis means a widespread inflammation and exfoliation of the skin all over the body and is often the result of the exacerbation of an unstable plaque psoriasis, particularly following an abrupt withdrawal of systemic treatment. It can be accompanied by severe itching, swelling and pain.If not treated effectively, this form of psoriasis can be fatal, as it hinders the skin, just as if it were burned, from serving as a protective barrier against injury and infection.
Flexural psoriasis appears as smooth inflamed patches of the skin of the skin folds, particularly around the genitals (between thigh and groin), the armpits, under an overweight stomach (pannus), and under the breasts (inframammary fold). It can be aggravated by friction and sweat, and is sensitive to fungal infections.
Guttate psoriasis is characterized by numerous small round spots. These numerous spots of psoriasis appear usually on the trunk, limbs and the scalp. Guttate psoriasis is associated most often with a *streptococcal* throat infection.
Pustular psoriasis shows many a raised bump filled with non infective pus. The skin under and surrounding these pustules is red and tender. Pustular psoriasis can be localized, mostly to the hands and feet (named palmoplantar pustulosis), or be generalized with widespread patches occurring randomly on any part of the body.
Other pustular psoriasis forms are f.i. pustular psoriasis of Barber type, annular pustular psoriasis, acrodermatitis continua and impetigo herpetiformis.
Nail psoriasis produces a variety of changes in the appearance of the finger and toe nails. These changes include the discolouring under the nail plate, the pitting of the nails, lines going across the nails, the thickening of the skin under the nails, and the loosening (onycholysis) and crumbling of the nails. Their fungal infection does often occur.
Other forms and causes of psoriasis are f.i. seborrheic-like psoriasis, napkin psoriasis, and drug-induced psoriasis.
Psoriasis can severely affect the health-related quality of life to an extent similar to other chronic diseases.
Prognosis: This disease can be a lifelong lasting chronic illness, though various treatments can help to control the symptoms. Many of the most effective agents used to treat severe psoriasis carry an increased risk of serious morbidity including skin cancers and

lymphoma. Affected persons experience often flares and remissions throughout their lives. The controlling of the symptoms requires usually a lifelong therapy.
Diagnosis: psoriasis in its early stage may be misdiagnosed. As psoriasis develops, the characteristic scaling pattern is usually easy recognizable, so that diagnostic tests are usually not needed. Confirming the diagnosis by a skin biopsy. Identifying the eggs of the worm, in a stool sample. Leishmania can be confirmed by skin biopsy and stool examination, etc.
Treatment: symptomatically, by locally administered corticosteroids, vitamin D, UV-light, sunbathing, Psoralen, PUVA treatment, methotrexate, cyclosporine, etretinate, isotretinoin, etc.
RFR method: detects and can eliminate all the pathogen microorganisms. The first step to take is the elimination of mycoplasma, than that of HTLV and that of the other bacteria and fungi.
The generally found resonances are: 370-376, 440, 442-452, 493-495 kHz
The most frequently found further resonances are: 291-293, 297-301, 311-312, 317-320, 332, 339-342, 344, 348, 360-366, 384-389, 392-397, 401-411, 480, 511-514, 520, 544-545, 555-558, 563 kHz
As mentioned above, secondary infections and thus their resonant frequencies often accompany to psoriasis.

17.20. Werner Syndrome

Werner syndrome (WS) is a disease of the skin and the subcutaneous tissue of the extremities, which shows epidermal thinning, loss of rete ridges, and dermal fibrosis with or without collagen hyalinization. The pilosebaceous structures are deformed. Newly synthesized, hyalinized collagens tend to replace the subcutaneous fat. There is usually no inflammatory infiltrate to be experienced.
WS is also known as progeria adultorum, "Adult progeria". This disease is the most common type of the premature aging disorders. Progeria can also refer to Hutchinson-Gilford Progeria Syndrome, which is described as a lamin A gene defect beginning early in life. Werner Syndrome is an autosomal recessive disorder affecting the connective tissues at all parts of the body. A *rubeola* infection may play a role in the development of this disease. The *Rubeola virus* is able to attach to DNA of the host's cells. This entity is caused by a mutation at the WS gene (WRN) locus, which belongs to the family of RecQ helicases. Werner Syndrome is caused by a helicase defect. The disease is connected with the excessive synthesis of collagen types I and III, which is dependent on elevated messenger RNA (mRNA) levels. The collagenase level is also increased by several times.
Human progeroid syndromes are linked up with mutations in single genes accelerating certain but not all, features of normal aging. Most syndromes are associated with defects in genome maintenance.
Symptoms include, a scleroderma-like appearance with nose and lip atrophy: the nose is pinched, and the cheeks are sunken because of fat loss, causing a birdlike facial appearance. Loss of subcutaneous fat, complicated with ulceration, can be observed on the shins and feet regarding most patients, calluses, hyperkeratosis and ulcerations on the soles are present mainly above the bony prominences. Graying, loss of hair and nail dystrophy can usually also be observed.
Diagnosis: symptomatically. Any laboratory abnormalities are related to concomitant diseases often seen in WS, f.i. diabetes mellitus, arteriosclerosis and hypogonadism.
Treatment: symptomatically and by the treatment of related disorders.
RFR method: detects and may eliminate the virus.
The most frequent resonances are: 282, 364-373, 381-390, 452-455, 478, 532-536, 564 kHz

17.21. Hair Disorders Associated with Infections

The scalp has approximately 100-200 thousand hairs; the hair growth cycle usually lasts for approximately two to six years, after which there comes a resting period of three months. About 70 hairs are normally falling out each day, the growth cycle of the beard hairs is similar. Eyebrows and eyelashes grow for three months and then rest for nine.
Hair infections can be caused by the attack of certain bacteria and fungi. Hair loss may be caused by many a factor, such as infections, inheritance, x-ray, cytostatic treatments, toxins and drugs, androgen inbalance, or other hormonal inbalances caused by anabolic steroids or corticosteroids, by general avitaminosis, zinc deficiency, iron deficiency and may be caused by systemic diseases as well, f.i. by hypothyroidism and hyperthyroidism, autoimmune dieseases, syphilis, AIDS etc. Certain metabolic disorders, f.i. homocystinuria or orotic aciduria, the administration of anticoagulants and psychogenic factors may all lead to hair loss. The origin of alopecia sometimes remains idiopathic. Hairloss can lead to various forms of the lack of hair, even to total baldness. The most common forms of hairloss are Androgenic alopecia, mostly experienced by men, a progressive hair thinning and hair loss on certain parts of the scalp and acute or chronic Telogenic Effluvium, mostly experienced among women, a diffuse hairloss of the scalp. The amount and patterns of hairloss can vary, ranging from male and female pattern alopecia (synonyms: androgenic alopecia, androgenetic alopecia and alopecia androgenetica) to alopecia areata, which involves a certain loss of hair on a round patch of the head, and alopecia totalis, meaning the total loss of hair of the scalp, as well as its most extreme form, alopecia universalis, which means the loss of hair from the whole head and body.

17.21.1. Alopecia Areata

Alopecia areata (AA) is a nonscarring, sometimes recurring type of hair loss that can affect any hair-bearing area. Clinically, AA can present many different patterns. Although medically benign, AA can cause tremendous emotional and psychosocial stresses of affected patients and their families.
There exists many a hypothesis supposing AA to be an autoimmune condition. The pathogenesis appears to be T-cell mediated, though antibodies directed to hair follicle structures are with increased frequency also present among AA patients compared to control subjects. By using immunofluorescence method, antibodies to anagen phase hair follicles were found in as many as 90% of patients with AA compared to fewer than 30% of the control subjects. The autoantibody response is heterogeneous and targets multiple structures of the anagen phase hair follicles. The outer root sheath is the structure targeted most frequently and is followed by the inner root sheath, the matrix and the hair shaft. Whether these antibodies play a direct role in the pathogenesis or whether they are an epiphenomenon is not yet known.
Histologically, lesional biopsies of AA show a perifollicular lymphocytic infiltrate around the anagen phase hair follicles. The infiltrate consists mostly of T-helper cells and, in a lesser extent, of T-suppressor cells.
Many a factor favors a genetic predisposition for AA and there exist some data telling about the importance of the HLA types (f.i. DQ3, DQ7 and DR4). Another gene of interest is the interleukin-1 receptor antagonist gene, which may correlate with the severity of the disease. The high association of Down syndrome with AA may suggest the involvement of a gene located on chromosome 21. AA seems to be influenced by polygenic factors. The role of environmental factors in initiating or triggering the condition must likewise be determined.
According to the hypothesis of infectious origin, certain pathogens, such as *mycoplasma, HTLV, cytomegalovirus and various fungi* may also be pathogenetic factors. The immune-

autoimmune AA can develop due to the infectious effect of the *HTLV* or/and the *mycoplasma*, and may be triggered by *fungal* infections as well.

17.21.2. Alopecia Caused by Fungi

Some fungal infections can cause massive hair loss. Ringworm, the common name of dermatophytosis, which is a fungal infection mostly of the skin, can cause patches of hair loss beginning as a small pimple progressively expanding in size, leaving scaly patches causing temporary baldness. In case of *Tinea capitis*, a superficial infection of the scalp caused by ringworm the fungi get into the hair fibers at the affected area, the hairs become brittle and break off easily, leaving a bald patch often itching, red and inflamed, with oozing blisters. The patches are usually redder around the outside with a normal skin tone in the center The fungus *Microsporum audouini* often causes scaly patches, the affected hairs are covered with the fungi and break off in a hight of some millimeters above the skin. *Trichophyton tonsurans* can also cause tinea capitis, especially in Latin American and Australian countries. Other fungi that may cause tinea capitis include *Trichophyton schoenleinii, Trichophyton megninii* in Southern Europe and Africa, and *Trichophyton violaceum* in the Middle East. The fungus *Microsporum gypseum* can sometimes cause tinea capitis as well. This fungus is present in the soil and may be transferred to human beings by contact with infected animals.
One can get ringworm from pets that carry the fungus, cats in particular are common carriers. It can be passed from one person to the next by direct skin-to-skin contact. One can catch ringworm also through contact with contaminated materials such as combs, unwashed clothing, and rarely even by contact with soil.

17.21.3. Scarring Alopecia

Alopecia of the scalp, eyebrows and eyelashes can occur without any visible associated change in the skin, or secondarily due to a severe, local, deep inflammation or scarring, like f.i. in case of a chronic discoid lupus erythematodes. Scarring alopecia can be caused by burns or x-ray therapy. Less obvious causes of scarring include lupus erythematosus; lichen planus; persistent pyoderma, deep viral, or fungal infections on the scalp, sarcoidosis and tuberculosis. Scarring alopecia and other visible changes of the skin of the scalp usually require a skin biopsy in order to be diagnosed.

17.21.4. Traction Alopecia

Traction alopecia is most commonly found among people with ponytails or cornrows, pulling their hair with excessive force.
Trichotillomania is the loss of hair caused by compulsive pulling and bending, which mostly done by children and psychotic adults. In this condition affected hairs are not absent from the scalp but broken.

17.21.5. Telogen Effluvium

Traumas such as chemotherapy, childbirth, major surgery, poisoning, severe stress, many a chronic infections and chronic illnesses may cause hair loss of telogen type.
The alterations in sexual hormon levels after delivery to its normal extent often cause severe hair loss. A similar situation occurs concerning women who take clomiphene, a fertility-stimulating drug.
Iron deficiency is a common cause of hair loss of less degree.
Radiation to the scalp, as happens when radiotherapy is applied to the head for the treatment of certain cancers there, can cause baldness of the irradiated areas.

Diagnosis of hair disorders: by bacterial and fungal cultures, by biopsy and histological examinations, by hair bulb examinations, by genetic predisposition examinations, by antibody examinations, etc.
Treatment: is depending on the diagnosis: in case of an infectious process by elimination of the microorganisms, in case of an autoimmune condition, by administering immunomodulators, such as steroids, in case of androgenic alopecia by administering topical Minoxidil, coffein, by using low-level laser therapy, etc.
RFR method: detects and may eliminate the locally or systemically present pathogen microorganisms.
The most frequent resonances of the pathogens found are:
In case of Mycoplasma fermentans: 442-451 kHz
In case of HTLV: 321, 330, 370-376, 432-433, 494-498 kHz
In case of Cytomegalovirus: 408-410, 530-536 kHz
In case of Trichophyton tonsurans: 390-392, 460-468 kHz
In case of Trichophyton rubrum: 385, 472-475 kHz
In case of other Trichophyton species: 294-297, 300-310, 331-334, 374-377, 387-389, 395-398, 413-415, 430-439, 534-542 kHz
In case of Microsporum audouini: 291-300, 312-318, 424-426, 432, 512 kHz
In case of other Microsporium species: 332-345, 410-413, 420-423, 496-498 kHz
In case of Tinea capitis: 352-361 kHz

17.21.6. Baldness and Graying

Hair disorders include also excessive hairiness, baldness and ingrown beard hairs. Baldness is much more common in men than in women. It can be due to genetic factors, aging, local skin disorders, certain infective and systemic diseases. Some medications, such as those treating cancer, also cause hair loss as a side-effect.
Male pattern baldness (Androgenic Alopecia) is the most common type of the loss of hair affecting men. It depends on the sensitivity of the hair bulbs to the androgen hormones present. Baldness, being a hereditary condition, runs in families. The alopecia may be circumscribed or diffuse. Loss of hair usually begins on the sides, near the front, or on the top of the head toward the back. Some people lose only small amounts of hair and develop a bald spot at the back or a receding hairline. Some patients, whose loss of hair begins at a young age may get completely bald.
Female pattern baldness is less common than male pattern baldness. This disorder causes the hair to thin at the front, sides, or on the crown.
The use of certain toxic drugs, such as those used in chemotherapy and hormon therapy, thallium and A vitamin derivates can end in toxic baldness.
Substance deficiençies, f.i. iron, selen, zinc etc. can cause baldness and localized patches of white hair, i.e. poliosis.
The total loss of hair pigmentations, which means that the hair turns completely white, is a common condition of old age, though microorganisms may also play a very important role in this occurrence. Graying hair is an age-dependent process, though the graying of poliosis may be the result of fungal infections, too.
Diagnosis: by hair biopsy and microscopic examinations.
Treatment: the first thing to do is to determine the type of baldness. Systemically given finasteride may promote hair growth of men. Corticosteroid may help in alopecia areata.
RFR method: is to be used if the disease is caused by microorganisms, may help the detection and eradication of the pathogens.
Its most frequent resonances are: 296, 330, 386-390, 397, 409, 448-451, 460-462 kHz
The most frequent resonances in case of graying of hair are: 535-544 kHz

17.22. Callus and Corn (Hyperkeratosis, Clavus)

A **callus** is an area on the uppermost layer of the skin, the stratum corneum (i.e. the keratin layer), that becomes abnormally thick, forms a protective pad in response to repeated rubbing. Calluses can form anywhere on the body, but usually develop over a bony spot on the hands, feet and elbows, or on other areas subjected to repeated wear or use, f.i. a violinist's jaw. Calluses may be avoided by removing the source of irritation, or, if not possible, by wearing gloves, using pads, or other protective devices. An abnormally thick keratin layer may also be caused by fungi.

A **corn** is a pea-sized, thickened area of keratin on the feet. Hard corns often appear over the joints of the toes, corns between the toes are usually soft. Corns can sometimes be confused with many a different wart virus, which also contain a thickened keratin layer. Warts are very sensitive if squeezed from the sides, while corns are more sensitive to direct pressure downward or inward against the bone. The corn develops a central zone, i.e. the eye of the corn.

Corns and calluses, especially those on the feet, may heal slowly in case of persons with diabetes and poor circulation.

Diagnosis: symptomatically.

Differential diagnosis: verruca vulgaris, verruca filiformis, etc.

Treatment: Corns and calluses are easier to prevent than to treat. By using protective pads and rings of suitable shape, for this purpose. By using keratin solving medications, f.i. salicylic acid.

RFR method: detects and may eliminate the viruses or fungi.

The frequencies in case of corn warts are: 290-298, 419-423 kHz

The frequencies in case of verruca warts are: 329, 352, 392, 403, 407, 448-452, 487, 506-508 kHz

The most frequent frequencies of plantar warts are: 343-346, 402-407, 418-423, 443-447, 459-464, 468-470 kHz

Repeat the eliminating procedure and remeasure to ensure its effectiveness.

17.23. Bedsore (Decubitus)

Bedsore is a form of skin damage resulting from lack of blood flow and from irritation to the skin over a bony projection, where the skin has been under pressure f.i. from a bed, wheelchair, cast, splint, or other hard objects for a prolonged period. The skin has a rich blood supply delivering oxygen to all its layers. Pressure is a common cause of reduced blood flow to the skin. In case of most people, bedsores cause pain or itching; while patients whose senses are severely dulled, may have painless deep sores. Once the skin is broken, infection may occur. Certain antibiotic-resistant microorganisms frequently inhabiting hospital environments can induce or complicate this disease, f.i. *Staphylococcus.* Prevention is the highest priority, even deep bedsores can almost always be prevented with intensive nursing care. Any sign of redness is a signal that immediate action is needed to prevent bedsore.

Treatment: by mobilization, physical trainings, by local wound therapy, by surgery.

The most frequent resonances are: 296-304, 372-373, 448-452, 476-481 kHz

18. DISEASES OF THE MUSCULOSKELETAL SYSTEM AND THE CONNECTIVE TISSUES ASSOCIATED WITH INFECTIONS

The skeleton, muscles, tendons, ligaments and other components of the joints form the musculoskeletal system. Disorders of the musculoskeletal system are often characterized by chronic inflammations and physical disabilities. Inflammation is a natural response to tissue irritations and damages, its signs are swelling, redness, heat and loss of functioning. The inflammation may affect only a small part of the body, f.i. a single joint or an injured tendon, but it also may be widespread, as occurs in case of certain inflammatory diseases such as rheumatoid arthritis. An inflammation can become chronic and persistent due to various causes, including: continuous movements, mechanical stresses, immune reactions, infections with bacteria, viruses, or fungi or due to deposits of abnormal materials. Infections of the musculoskeletal system can cause crippling. Immediate treatment can prevent the permanent joint damage.

Many connective tissue diseases feature abnormal immune system activities causing inflammation in the connective tissues as a result of an immune system directed against the patient's own body tissues (autoimmune diseases). Diseases in which an inflammation or weakness of collagen tends to occur are also referred to as collagen diseases. Collagen vascular diseases are associated with collagen and blood vessel abnormalities and autoimmune-like processes. Connective tissue diseases can show a strong genetical predisposition or weak inheritance risks, but can be caused also by environmental infectious factors such as *Mycoplasma*.

Heritable connective tissue disorders are Marfan syndrome, Ehlers-Danlos syndrome, Osteogenesis imperfecta, Stickler syndrome, etc.

Autoimmune connective tissue disorders are Systemic Lupus Erythematosus (SLE), Systemic sclerosis(SSc), Rheumatoid Arthritis, Sjögren's Syndrome, Mixed Connective Tissue Disease, Dermatomyositis (DeMy) and Polymyositis (PM).

The cause of triggering many connective tissue diseases is an infection with *Mycoplasma fermentans* or/and *HTLVs*. Mycoplasmal and HTLV antigens adhere to the connective tissue cells, provoking thus autoimmune-like processes. (See the Chapters of Mycoplasma and HTLVs). Connective tissues form a framework, or matrix for the body and are composed of two major structural protein molecules: collagen and elastin. Mycoplasmal antigens are able to be adsorbed to collagen structures. Collagen plays an important role in the development of the disease. Connective tissue diseases can have a strong or weak association with inheritance, as autoimmune processes may have genetic and environmental (infectious) causes as well.

18.1. Acute and Chronic Infectious Arthritis

Septic acute arthritis is a medical emergency and has to be promptly recognized and treated appropriately in order to avoid permanent joint damages. Microorganisms usually reach the joint by spreading hematogenously from the locus of the primary infection being elsewhere, though their source sometimes cannot be found. Acute and chronic septic arthritis may occur also by direct extension of the infection from the adjacent bone, or from the adjacent soft tissue.

Many a different type of bacteria can cause acute bacterial arthritis, including *Neisseria gonorrhoeae* (see Chapters 6.3.1. and 16.2.), *Staphylococcus aureus* (see Chapter

Streptococcus pneumoniae *Streptococcus pyogenes* *Haemophilus influenzae* *Escherichia coli* *Salmonella* and *Pseudomonas*

Patients with *Salmonella* arthritis often show evidence of an underlying osteomyelitis. An increased susceptibility to joint infection occurs among patients with diabetes, as well as those with lymphomas, and those receiving corticosteroids or immunosuppressive drugs. In addition, joints previously damaged f.i. by rheumatoid arthritis, gout, systemic connective diseases and trauma, are more susceptible to septic infections.

Symptoms: a bacterial acute arthritis usually develop suddenly and is accompanied by fever and chills. One or several joints may be involved. The affected joint is hot, erythematous, swollen and painful. Muscle spasms are common. The larger joints, such as the hips, knees and shoulders are more commonly affected, while the wrists, ankles, elbows and the sternoclavicular and sacroiliac joints are less likely involved.

The causes of chronic arthritis include *Syphilis* *Mycobacterium tuberculosis* *M. triviale*, *M. kansasii*, *M. scrofulaceum* and *Mycobacterium leprae* *Borrelia B. sensu lato* and *Brucellosis* Systemic mycoses causing chronic arthritis include *Candidiasis* *Coccidioidomycosis* *Histoplasmosis* *Blastomycosis* *Cryptococcosis* *Actinomycosis* and *Sporotrichosis*

Symptoms: a chronic infectious arthritis develops usually affecting only one single joint, its development lasts over weeks and less intensive symptoms can be experienced. The affected joint is often indolent, with only a minimal or no swelling, warmth and redness of the affected area.

Diagnosis: symptomatically, by examinations including aspiration, fluid, biopsy, cultures of the microorganisms, x-ray, blood cultures, and blood examinations.

Treatment: septic arthritis requires prompt treatment with appropriate systemic and/or local antibiotics, antifungal substances, anti-inflammatory drugs and analgesic drugs.

RFR method: is to be used after a laboratory examination and diagnosis, together with the antimicrobial treatment. By looking for the pathological microorganism based on laboratory diagnosis and culture.

18.2. Rheumatic Fever

Rheumatic fever (RF) is a systemic inflammatory disease occuring following *group A beta-haemolytic streptococcus (GABHS)* infections This illness causes exudative and proliferative inflammatory processes resulting in chronic, progressive damages of the heart, joints, central nervous system (CNS), skin and the subcutaneous tissues. Acute rheumatic fever (ARF) comes after a previous group A streptococcal infection of the upper respiratory tract. Certain *Beta-streptococcal serotypes (M, 3, 5, 18, 19 and 24)* are directly linked to ARF.

Antibiotics are widely used to treat streptococcal infections at their early stage. All organs of the body are affected by the process of rheumatic fever, in case of which the immune response is often insufficient and inadequate.

Symptoms: of this postinfectious illness are at first joint pain and fever. One or more joints feel tender when touched and become suddenly painful, red, hot and swollen, and contain fluids. Ankles, knees, elbows and wrists are often affected; and shoulders, hips, and the small joints of the hands and feet may be involved as well.

RFR method: is most important in case of poly-resistant streptococcal infections. In resistant cases the most effective antibiotics should to be administered, then RFR method

has to be used in order to measure and detect the pathogen Streptococci. As regards the frequencies of the most frequent pathogen Streptococcus groups see below.
The most frequent resonances are: 288, 307, 310-321, 324, 337, 345, 351-353, 358-361, 363-376, 381-385, 389, 391, 397-413, 418, 426, 432, 434, 440-452, 466-468, 478, 498, 508, 516, 520-523, 538-542, 548-551, 576 kHz
This list may not be complete, as there are other Streptococcus subspecies with different resonant frequencies.

18.3. Reiter's Syndrome

Reiter's syndrome is caused by an autoimmune inflammation and is characterized by arthritis, urethritis, conjunctivitis and mucocutaneous lesions. The genetic predisposition is an important factor of the development of this syndrome, which is caused by the combined attack of various bacteria and viruses, including *Yersinia, Salmonella, Chlamydia, Mycoplasma*, etc. The majority of the patients are HL-A-B27 antigen positive. Reiter's syndrome may result from *bacillary dysentery* as well.
This syndrome exists in two forms:

1. one type occurs via sexually-transmitted infections, such as *chlamydial, herpes viral* and *adenoviral* infections, occuring mostly among young men and women,
2. the other type usually follows an intestinal infection such as *salmonellosis, shigellosis*, and/or a *proteus* bacterial infection.

People, who develop Reiter's syndrome after exposure to these infections appear to have a genetic predisposition to this type of reaction, related to the same gene found in patients with ankylosing spondylitis.
Symptoms: The penis, prostate gland, vagina and urinary tract can become inflamed and painful. The conjunctiva becomes red and inflamed, is itching, or burning and edematous. Usually there are several joints affected, including the knees, toes and loci, where tendons are attached to bones. The skin lesions are named keratodermia blenorrhagica and are microscopically indistinguishable from those of pustular psoriasis. The inflammatory changes of the mucosa are similar, but the keratinized cells are not accumulated. This syndrome often begins with urethritis after a sexual intercourse, followed in a few days or weeks by conjunctivitis, mucocutaneous lesions and arthritis. This acute inflammation affects usually two or more joints. The joints are mostly warm, erythematous and painful. The joints of the lower extremites are predilected: the ankles, knees and the metatarsophalangeal and proximal interphalangeal joints of the toes are often affected. Its urethritis is characterized by a mucopurulent discharge and dysuria, it may be asymptomatic and overlooked unless the urethra is milked. The urethral meatus can be edematous and reddened, urethral strictures may develop. Prostatitis and seminal vasculitis can occasionally occur. Hemorrhagic cystitis leading to frequent urination, suprapubic pain, hematuria, and rarely to the obstruction of the ureters can also come to pass. Mucocutaneus lesions can also be experienced most frequently on the glans of the penis, on the palms, soles and in the mouth.
Diagnosis: symptomatically and by baterial culturing, HLA examinations, etc.
Differential diagnosis: by distinguishing it from gonococcal arthritis, rheumatoid arthritis, psoriatic arthritis, Sjögren's Syndrome and PAN.
Treatment: the first step to be taken is to administer antibiotics in order to reduce infections. Arthritis is usually treated with NSAIDs, sulfasalazine, methotrexate, corticosteroids can occasionally also be administered.
RFR method: detects and eliminates the bacteria and viruses.
The frequencies of Chlamydia are: 316-319, 374-386, 429, 440-444, 479-482, 566 kHz
The frequencies of Salmonella enteridis are: 325-339, 361-386, 389, 496-498 kHz
The frequencies of Shigella dysenteriae are: 310, 315-321, 388-398, 410, 425, 496 kHz
The frequencies of Proteus mirabilis are: 320-326, 345-352, 411, 516 kHz

The frequencies of Proteus vulgaris are: 327-329, 333-339, 408-416, 426, 522-529, 535 kHz
The frequencies of Adenovirus are: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz
The frequencies of Cytomegalovirus are: 305, 327, 349, 408-411, 512, 534, 548 kHz
The frequencies of Epstein-Barr Virus are: 337, 339, 347, 372-383, 518-519 kHz
The frequencies of Herpes Simplex Virus-1 are: 290-294, 332-337, 344-346 kHz
The frequencies of Herpes Simplex Virus-2 are: 352-365, 413, 425, 434 kHz
The frequencies of Herpes Zoster Virus are: 416-421 kHz
The frequencies of Mumps virus are: 328, 344, 363, 372-373, 388-392, 472, 595 kHz

18.4. Chlamydial Arthritis Associated with Mycoplasmal Infection

Chlamydial arthritis might be the last thing a doctor would suspect if a young person has chronic, unexplained pain in a joint. If certain sexually transmitted diseases are left untreated, they can cause arthritis. *Mycoplasma and/or chlamydia* infections are more frequent than before and often cause arthritis. In case of chlamydial and mycoplasmal infection-induced arthritis, the survival of these microbas within the monocytes, which are the primary synovial host cells, leads to a persistent interaction between host and microbas, determining the pathogenesis of the arthritis. Reactive arthritis may develop usually 2-4 weeks following a genitourinary or gastrointestinal infection. About 10% of patients have no preceding symptoms of these infections.

Inflammations of the joints, the axial skeleton, skin, the mucous membranes, the gastrointestinal tract and eyes may occur. Most of the affected patients prove to be HLA-B27 positive. HLA-B27 positive people are cc. In a 50-fold increased danger to develop reactive arthritis, though this illness can occur also among HLA-B27 negative persons.

HLA-B27 positive persons, as well as those with a strong family clustering of the disease, tend to develop a more severe and long-term disease. About 1-4% of patients (often HLA-B27 positive ones) gets ill with reactive arthritis after certain enteric infections, the mechanism of which is not yet known. In these cases synovial fluid cultures prove to be negative to enteric pathogens or Chlamydia species. However, a systemic and an intrasynovial immune response (intra-articular antibodies and reactive T cells) to these pathogens can be experienced, so that in case of arthritis of this type a coinfection with *Chlamydia and mycoplasma* and an immune-mediated synovitis comes to pass.

Symptoms of Chlamydial arthritis usually include dactylitis and an asymmetric involvement of the weight-bearing joints, such as knees, ankles and hips, as well as the inflammation of the axial joints, which, if severe, can be similar to that of ankylosing spondylitis, which all can cause pain, tenderness and swelling. The Achilles insertion may also be affected, as well as the plantar fascial insertion on the calcaneus, ischial tuberosities, iliac crests, tibial tuberosities and the ribs.

Keratoderma blenorrhagica observed on the palms and soles is indistinguishable from pustular psoriasis. Erythema nodosum can also occur. Nails can become thickened and crumbling, resembling a mycotic infection or psoriatic onychodystrophy, but nail pitting does not exist.

Conjunctivitis, acute uveitis, episcleritis, keratitis and corneal ulcerations may also be caused. Chlamydial arthritis is frequently associated with secondary infections.

Enteric infections, such as those of *Salmonella, Shigella, Yersinia* and *Campylobacter* species. also can trigger reactive arthritis. Some patients have permanently intermittent bouts of diarrhea and abdominal pain, and get lesions resembling ulcerative colitis or Crohn's disease found by ileocolonoscopy in patients with established reactive arthritis.

The most frequently occuring co-infection is caused by *Mycoplasma fermentans*. This pathogen increases the autoimmune character of the disease.
Secondary infection may be *HIV*, causing immune deficiency.
Diagnosis: symptomatically and by clinical laboratory examinations, HLA-B27 testing, synovial fluid analysis, bacterial culturing of the throat, the stool, or the urogenital tract in order to isolate the causative pathogen. By serology of Chlamydia species, PCR.
Treatment: by administering NSAIDs, antirheumatic drugs, corticosteroids together with antibiotics, f.i. Doxycycline. In case of chlamydia-induced reactive arthritis, an appropriate treatment of the acute urogenital infection can prevent reactive arthritis and the treatment of reactive arthritis in its active phase for 3-month by administering Doxycycline shortens the time of this illness.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances of Chlamydia are: 301-303, 317-319, 378-383, 440-443, 479-483, 491, 560-569 kHz
The most frequent resonances of Mycoplasma fermentans are: 442-451, 491-495 kHz
Mycoplasma, or HIV *should be eliminated first. This type of arthritis can be cured by administering effective antibiotics for a long time together with RFR method. Reinfection do often occur.*

18.5. Ankylosing Spondylitis (Bechterew's Disease)

Ankylosing spondylitis (named also rheumatoid spondylitis, Marie-Strümpell disease and Bechterew disease) is chronic, usually progressive and inflammatory involving the articulations of the spine and its adjacent soft tissues. The sacroiliac joints are always affected. The involvement of the joints of the hip and shoulder often occurs; while the peripherial joints are less frequently affected. This disease affects predominantly young men mostly in their third decade. A high association is to be found between this disorder and the HLA-B27, HLA-W27 haplotypes. The clinical features of this disease are distinctly different from those of rheumatoid arthritis. The etiology of this disease is a combined viral infection and the development of an allergic-autoimmune response, non effective on the pathogens, but damaging the host's structures.
Symptoms: The most common symptom is back pain, varying in intensity. Bending forward often relieves the pain and the associated muscle spasms of the lower back. Occasionally pain of the large joints, such as the hips, knees and shoulders, acute, or chronic iritis, inflammation of the heart valves, nerve damages, impotence, anemia, urinary incontinence, loss of the reflexes, ossification, bone erosions, medial necrosis at the root of the aorta causing dilatation and severe deformities are present aswell. Fibrous tissue may enter the membranous septum and invade the atrioventricular bundle, causing conduction defects. Bilateral pulmonary fibrosis, involving the upper lobes may also come to pass.
Diagnosis: symptomatically, by x-rays, erythrocyte sedimentation and other laboratory tests examinations, HLA-B27 identification, MRI, etc.
Treatment: symptomatically, by administering anti-inflammatory drugs, corticosteroid derivates, muscle relaxants and by surgery.
RFR method: detects and may eliminate the pathogen microorganisms, being usually a lot of them.
The most frequent resonances are: 307-309, 313, 320-321, 332-334, 339-441, 348, 370-382, 397, 402-403, 419-411, 434, 442-444, 447-451, 493-495, 518-519 kHz

18.6. Psoriatic Arthritis

The prevalence of arthritis among patients with psoriasis is, even if the degenerative joint diseases and rheumatoid arthritis cases are excluded, higher than that found in the general population. A significant association between the HL-A-B27 antigen and psoriatic arthritis

has been found concerning patients with sacroiliitis and spondylitis. Psoriasis usually precedes the onset of arthritis by months or years; the onset of both may however coincide, or, very rarely, arthritis may precede psoriasis as well.
Psoriatic arthritis is a form of arthritis of patients suffering from psoriasis of the skin or nails. The disease resembles rheumatoid arthritis, but autoantibodies characteristic of RA are absent. The chronic affected joints (mostly the fingers and toes) may become swollen and deformed. The joint of the hips and spine are often affected as well. The prognosis for psoriatic arthritis is usually better than that for rheumatic arthritis as less joints are affected.
Diagnosis: symptomatically and by the patients history of psoriasis.
Differential diagnosis: The asymmetry of the joint involvement, negative test for rheumatoid factor, absence of rheumatoid nodules help to distinguish psoriatic arthritis from rheumatoid arthritis.
Treatment: symptomatically. The administering of corticosteroids will worsen the psoriatic skin symptoms. The treatment of coinfections (by worms, bacteria, etc.) is necessary.
RFR method: detects and eliminates all the pathogen agents.
The most frequent resonances are: 307, 310, 318-319, 332-339, 348, 370-373, 397-399, 401-409, 426, 442-451, 513, 544, 555-558, 563 kHz

18.7. Gout

Gout is characterized by sudden, recurring, very painful attacks of arthritis caused by deposits of monosodium urate crystals, which accumulate in the joints due to an abnormally high uric acid level in the blood. The pathological lesion of gout is the tophus, a urate deposit surrounded by an inflammation and a foreign-body reaction.
Primary gout represents a group of inborn metabolic disorders leading to hyperuricemia, recurrent attacks of a characteristic acute arthritis, and tophaceous deposits of sodium urate. Nephrolithiasis and a parenchymatous renal disease can often develop in the course of the illness.
Secondary gout is an acquired form of the disease, which develops in the course of certain illnesses in which hyperuricemia occurs.
The genetic determinants of hyperuricemia are part of a multifactorial process. Owing to the metabolic and genetic heterogeneity of gout there are specific subtypes, so that the patterns of development of each may be determined. Environmental factors, f.i. diet, alcohol and drugs together with genetic factors determine the degree of hyperuricemia. Experiences suggests that certain microorganisms also have a role in the pathogenesis of hyperuricemia. According to these suppositions this disease can be caused by *Nanobacteria*, or *Chlamydia,* in combination with *Herpes viruses*.
In case of this illness urates tend to deposit in the cartilages, epiphyseal bones, periartricular structures and the kidneys. Less commonly, they occur in the skin of the fingertips, palms and soles, the tarsal plates of eyelids, the nasal cartilages, in the cornea of the eye, or along the nerves, causing carpal tunnel, or tarsal tunnel syndromes. They rarely are present in the myocardium, the aortic and mitral valves, vocal cords and the arytenoid cartilages. Cartilaginous degeneration, synovial proliferation and pannus, destruction of the subchondrial bone, proliferation of marginal bones, and sometimes fibrous or bony ankylosis develop in the affected joint.
The only distinctive histological feature of the gouty kidney is the presence of sodium urate crystals in the medulla, or pyramids and the giant cell reaction surrounding it. The earliest structural abnormalities in the kidneys are tubular damages associated with interstitial inflammatory reactions. There is a distinctive glomerulosclerosis to be experienced, with the uniform fibrillar thickening of the glomerular capillary basement

membranes, differing from that of nephrosclerosis and diabetic glomerulosclerosis. The inflammatory changes appear to be of infectious origin.
In case of chronic gouty arthritis, the development of tophi correlates with the level of the serum urate concentration, the severity of renal involvement and the duration of the disease.
Every acquired hyperuricemic state may be complicated by secondary gout, including chronic myelogenous leukemia, multiple myeloma and chronic hemolytic anemia.
Symptoms: the disorder affects most often the joint at the base of the large toe, causing podagra, and often affects also the insteps, ankles, knees, wrists and elbows. Crystals can form in these peripherally located joints being cooler, than those in the central part of the body, hence the urate tends to crystalize at cooler temperatures. Other symptoms of acute gouty arthritis can include fever, chills, a general sick feeling and rapid heartbeat. Gout affects usually middle aged men and women following their menopause. The first attack affects often only one joint, lasts for a few days to disappear then gradually, leaving no symptoms until the next attack. Untreated attacks can last longer, occur more frequently, and affect several joints, which may be permanently damaged. A severe chronic gout may develop, which can potentially cause a deformity.
Diagnosis: symptomatically, by high uric acid levels in the blood, identification of urate crystals by polarization microscopic examinations used in biopsy, and by x-ray.
Differential diagnosis: by distinguishing it from other types of arthritis and hyperuricemia.
Treatment: by administering NSAIDs, other analgetica, colchicine, or allopurinol.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances in case of gout are: 290-296, 305-310, 320, 324, 332-336, 345-346, 372-387, 398-403, 409, 414-420, 424-425, 440-452, 456-460, 467, 556 kHz
Gout patients have many different pathological resonances. Intense attacks of acute gout may occur for a short time after RFR method, and the inflammation may also increase. The patient has to drink a lot of fluids. It remains unclear, whether the pathogens of the found frequencies cause the illness, or is only the result of secondary, independent processes.

18.8. Tendinitis and Tendovaginitis

Tendinitis is the inflammation of a tendon; tenosynovitis or tendovaginitis is a tendonitis accompanied by inflammation of the protective sheath around the tendon; both produce pain in the affected regions. Tendons are fibrous cords of the tissue connecting the muscles to the bones. Most cases of tendonitis occur among persons of middle and old age, as the tendons become more susceptible to injury. Certain tendons, especially those of the hand, are particularly susceptible to inflammation. Inflammation of the tendon that extends the thumb away from the hand is named *de Quervain's disease*. Inflammation affecting the tendons working when catching, can cause a popping feeling. Tendinitis above the biceps muscle in the upper arm causes pain when the elbow is bent, or the forearm rotates. The Achilles tendon in the heel and the tendon that runs above the top of the foot can also become often inflamed.
An important form of tenosynovitis is the *carpal tunnel syndrome*, the symptoms of which may be confused with those of the thoracic outlet syndrome, cervical disk disease, and a local vascular insufficiency. The thickening or swelling of the tendons as they pass through the flexor compartment at the wrist, as well as amyloid deposits in the said locus in case of multiple myeloma and a bone enlargement can exert pressure on the median nerve, causing nocturnal paresthesias and pain in the fingers, wrist and forearm. By the worsening of this condition atrophy of the thenar eminence does develop, as well as weakness and sensory loss in the territory of the median nerve. There may be tenderness felt on pressure above the carpal ligament and electrical tingling experienced in the fingers when the wrist is tapped. The nerve conduction may be delayed at the wrist, which confirms the diagnosis.

Calcific and noncalcific tendonitis is common in the bicipital and supraspinatus tendons and in the common tendon at the origin of the forearm extensors and in the tendons of flexors at the lateral and medial epicondyle of the humerus, respectively.
Symptoms: acute tendonitis is characterized by severe local pain and tenderness with marked limitation of mobility and activity. A chronic process may cause calcified deposits as well.
The tendonsheaths can also be affected in case of joint diseases, such as rheumatoid arthritis [redacted] fibromyalgia syndrome [redacted] systemic scleroderma [redacted] osteoarthritis [redacted] Reiter's syndrome [redacted] and gout [redacted] *Gonococcal* and *Borrelia B. sensu lato* bacteria can cause tenosynovitis, usually affecting the tendons of the shoulders, wrists, fingers, hips, ankles and feet.
Diagnosis: by physical examinations and x-ray.
Treatment: by administering NSAIDs, corticosteroids, analgesics, physiotherapy and surgery.
RFR method: detects the pathogen microorganisms and eliminates them.
The most frequent resonances are: 307, 327, 337, 364, 370-374, 378-382, 512 kHz, as to the other frequencies, see their special Chapters. *Mycoplasma fermentans* may be found in case of chronic processes, its frequencies being: **442-444, 447-451, 493-495 kHz**

18.9. Morphea

Morphea, named also localized scleroderma, is an inflammatory, autoimmune disease of infectious origin and is characterized by an excessive collagen deposition leading to the thickening of the dermis, subcutaneous tissues, or both. Morphea is classified into subtypes such as plaque, generalized, linear and deep ones, according to the clinical presentation and the depth of their tissue involvement. Overproduction of collagen by fibroblasts in affected tissues characterizes all of the forms of morphea, though the mechanism by which these fibroblasts are activated, may differ. The pathogenesis of morphea includes endothelial cell injuries, activation of T lymphocytes and other inflammatory processes by *Borrelia B. sensu lato, Mycoplasma fermentans, Human T-cell Lymphotropic Viruses and/or Human B-cell Lymphotropic Viruses* and the dysregulation of the collagen production, as well as vascular and peripherial nerve damages. Women are affected approximately 3 times as often as men concerning all forms of morphea except the linear one, which has only a slight female predominance.
Plaque-type morphea lesions are characterized by circumscribed indurated plaques ranging from 1 cm to more than 20 cm in diameter. They often begin as erythematous and violaceous, slightly edematous patches, or plaques. With the progression of the disease, sclerosis develops centrally, while the lesions undergo a peripheral expansion. The centre of the lesions gradually develops a waxy, ivory color. In the active phases of the disease, a violaceous border (lilac ring) surrounds the indurated region. Hyperpigmentation often ensues as lesions evolve and eventually involute. The sclerotic lesions of **guttate morphea** are typically whitish in color, and its clinical appearance may overlap that of extragenital lichen sclerosus.
With the loss of hair follicles and sweat glands the surface becomes over time smooth and shiny, and after months or years the dermis becomes atrophic.
The multiple, coalescent lesions of **generalized morphea** are often hyperpigmented to silvery.
Idiopathic **Atrophodermia Pasini and Pierini** is thought to be an abortive form of morphea. It is characterized by hyperpigmented, slightly atrophic, circumscribed areas of the skin with no induration. Its histologic features are atrophy of the epidermis, melanin pigment deposits among the basal cells and infiltrations with lymphocytes and-monocytes. Similar hyperpigmented patches with minimal induration are seen among persons with

superficial morphea, which, unlike atrophodermia Pasini and Pierini, is histologically characterized by sclerosis of the upper dermis.

Frontoparietal linear morphea, named also en coup de sabre, is characterized by a linear, atrophic depression suggestive of a stroke from a sword.

Parry-Romberg syndrome, a progressive hemifacial atrophy represents the severe, segmental form of craniofacial linear morphea.

Deep (or subcutaneous) morphea is characterized by bound-down, sclerotic plaques of a „cobblestone," or „pseudo-cellulite" appearance, and can involve the deep dermis, the subcutaneous tissues, fascia and even the superficial muscles. The **disabling pansclerotic morphea** of children, a subtype of deep morphea, involves all the tissues from dermis to bone. This rare, aggressive and mutilating variant of deep morphea begins among children before they are 14, has a course of relentless progression causing severe disabilities. Every form of morphea beginning during childhood is named juvenile morphea. This disabling illness begins on the extensor side of the extremities and progresses to the trunk, the flexor side of the extremities, the face and scalp as well, sparing only the fingertips and toes. An other subtype of deep morphea, **Eosinophilic fasciitis (Shulman's syndrome)** involves primarily the fascia and is characterized by an acute onset of pain and edema symmetrically affecting the extremities, followed by progressive indurations with an appearance similar to deep morphea. In case of eosinophilic fasciitis the fingers and toes are usually spared; while the trunk is occasionally involved.

Bullous morphea is a rare variant in which tense subepidermal bullae develop overlying plaque-type, linear, or deep morphea lesions. This phenomenon may result from the stasis of lymphatic fluids caused by this sclerodermatous process or by a coexisting lichen sclerosus.

Muscle weakness may occur in case of patients with CNS abnormalities related to craniofacial linear morphea and those with peripheral nerve involvements caused by vascular demages. Signs of carpal tunnel syndrome may be experienced by patients with deep morphea, or eosinophilic fasciitis affecting the wrist.

Coinfections, caused by *EBV, VZV, measles, HTLV, HBLV, Mycoplasma fermentans* and mostly by *Borrelia B. sensu lato* are supposed to be possible triggers of the process of this disease.

Borrelia afzelii can be an etiologic cofactor of morphea. Borrelia was detected within morphea lesions from many a European and Japanese patients, representing *Borrelia afzelii* and *Borrelia garinii* rather than B burgdorferi sensu stricto, the predominant subtype in the United States.

Diagnosis: symptomatically, by clinical laboratory examinations and histology.

Treatment: by administering antibiotics, corticosteroids, antiinflammatory drugs and vitamin D analogs.,

RFR method: detects and may eliminate the coexisting pathogens.

The most frequent resonances are: 311-312, 315, 321, 324, 330, 340-342, 365, 370-390, 402-403, 416-421, 442-444, 447-451, 493-495 kHz

The frequencies may differ as regards the various types of morphea, but the most frequently found resonances are present in case of every morphea patient. Morphea should be treated in combination of the found characteristic frequencies for a long time.

18.9.1. Scleroderma-like Condition

A scleroderma-like condition may manifest itself either strictly limited to the skin, as in case of morphea, of be a multiorgan disorder similar to systemic sclerosis. Accordingly, its cutaneous manifestations vary clinically. In case of nodular or keloidal form, patients develop lesions clinically indistinguishable from keloids; its histopathological findings, however, are more variable, showing besides the morphea characteristics those of a hypertrophic scar.

The symptoms of a scleroderma-like condition can be those of fasciitis and myositis. Eosinophilia, and hypergammaglobulinemia are often present. Tenderness and swelling of the extremities can develop with the onset of symptoms related often to unusual exertion. There can be a marked pigmentation of the affected skin area present, the lesion can arise from the healthy skin and a local sclerosis can be manifested. The keloidal and sclerotical skin is hard. Its biopsy examination shows perivascular infiltrations of lymphocytes, histiocytes, plasma cells and sometimes extensive calcification in the dermis, the subcutaneous fat, the inflammated fascia and in the underlying muscles. In case of scleroderma-like conditions there can develop sclerosis and fibrosis in the connective tissues involving a variety of internal organs. Later on fibrinous or/and calcificated deposits can appear on the surfaces of the tendon sheaths and in the overlying fascia.
The etiology of the scleroderma-like conditions is based on an inherited genetic predisposition combined with infection.
The most frequent causative infectious agents are *mycoplasma*, *HTLV*, *HPV*, *nanobacteria*, *Coxsackie viruses*, *EBV*, *Adenoviruses* and *Borrelia B. sensu lato*.
Diagnosis: by complex laboratory examinations and by biopsy with histological analysis.
Treatment: symptomatically, by administering NSAIDs and by immunosuppressive therapy.
RFR method: detects and can eliminate all pathogenic infective agents.
The most frequently found resonances are: 287-304, 311, 313, 317-319, 324-325, 336, 343-345, 353, 357-358, 364, 370-381, 387-388, 398, 438, 444-451, 481-482, 510, 517-518, 543-546, 554-555, 560-568 kHz

18.10. Progressive Systemic Sclerosis (Scleroderma)

Progressive systemic sclerosis (PSS) is a systemic disorder of the connective tissues that leads to fibrosis of the skin and to a variety of internal organs, most notably to the gastrointestinal tract, lungs, heart and kidneys. A thin epidermis overlies compact bundles of collagen, lying parallel to the epidermis. Finger-like projections of collagen extend from the dermis into the subcutaneous tissue and bind the skin to it. The dermal appendages become atrophied, the rete pegs will be lost. The degree of fibrosis in the esophagus is less high than that in the skin, ulcerations of the esophagus and of the gastrointestinal mucosa are often experienced, and may be developed either due to PSS or owing to a superimposed peptic esophagitis. Similar changes may be found everywhere in the gastrointestinal tract, especially in the distal part of the duodenum, the jejunum, and the large intestines.
Diffuse interstitial fibrosis often goes with the thickening of the alveolar membrane and with peribronchial fibrosis accompanied by bronchiolar epithelial proliferations. Ruptures of the septa in the lungs produce small cysts and areas of bullous emphysema. Small pulmonary arteries and arterioles show intimal thickening, fragmentation of the elastica, and muscular hypertrophy.
Synovitis and arthritis are similar to those seen in early rheumatoid arthritis, histologically edema is observed with the infiltration of hyperactive lymphocytes and plasma cells. Later on, there will fibrinous deposits appear on the surfaces of tendon sheaths and on the overlying fascia, leading to audible creaking above the moving tendons.
In the heart, myocardial interstitial fibrosis replaces the myocardial fibers. This fibrosis leads to defects of conduction and to arrhythmias. Fibrinous pericarditis and pericardial effusion are often found as well.
The renal lesions of PSS are characterized by intimal hyperplasia, fibroid necrosis, thickening of the glomerular basement membranes, cortical infarctions, glomerulosclerosis and can cause malignant hypertension as well.
Symptoms: the first clinical manifestations include usually initial symptoms of the thickening and swelling of the ends of the fingers. Raynaud's phenomenon (in which the fingers are suddenly paling and tingling or become numb in response to cold or to

emotional upset) is also frequently experienced. Scleroderma can damage large areas of the skin, which become taut, shiny and darker than usual.
Telangiectasia appear on the fingers, chest, face, lips, and the tongue. Bumps composed of calcium can develop on the fingers, on other bony loci and at the joints. *Mycoplasma species* may play a role in the development of this progressive systemic disease [redacted]

The **CREST syndrome** (the abbreviation of Calcinosis, Raynaud's phenomenon Esophageal motility disturbance, sclerodactylia and telangiectasia) named also limited cutaneous sclerosis, is a less severe form of the disease.
Laboratory tests cannot identify the disease, though a test for the antibody to centromere, which is a component of chromosome, may help to distinguish the limited cutaneous scleroderma form from the systemic one.
Diagnosis: symptomatically, by complex examinations and by biopsy.
Treatment: by administering NSAIDs, penicillamine, calcium-channel blockers, ACE-inhibitors. The administration of corticosteroids is only advised in case of exacerbation f.i. of pneumonitis and myositis.
RFR method: detects and eliminates the pathogen microorganisms.
The most frequent resonances are: 305, 313-319, 336, 343-344, 348-350, 353-358, 370-380, 387-389, 438-440, 442-452, 482-486, 510-512, 516-519, 534-539, 538-539, 545-548, 555, 564 kHz

18.11. Raynaud's Syndrome

Raynaud's syndrome (RS) affecting the connective tissues is characterized by brief contractions of the small arteries named vasospasms, which vessels supply blood to the arms and legs, hands and feet. Raynaud hypothesized that this syndrome, primarily occuring among females, is caused by the hyperactivity of the sympathetic nervous system. Diseases causing Raynaud phenomenon include rheumatological diseases, connective tissues diseases such as scleroderma, lupus erythematosus, dermatomyositis, rheumatoid arthritis, hypersensitivity vasculitis, Wegener's granulomatosis, Takayasu arteritis, giant cell arteritis and other arterial diseases, carpal tunnel syndrome, and hypothyroidism. Drugs, f.i. sympathomimetics, amphetamine, etc. may also cause this syndrome.
If somebody has an attack of RS, the small arteries of the arms or legs have a spasm, limiting the blood flow. The tissues become deprived of the blood's oxygen, causing color change of the skin. At first, the skin blanches, turns very white, getting then blue as the tissues get colder. The person will complain of numbness of the fingers and occasionally of pain. The affected skin feels very cold. The areas suffering from the lack of oxygen are very well demarcated, usually occurring at the joint lines.
The exact pathophysiological mechanisms involved in RS remain dubious. Recently discovered endothelium-derived vasoconstrictor and vasodilator substances are involved. Vasospastic RS people have normal digital arterial pressures at room temperature, but exhibit abnormal vasoconstrictor responses to cold, causing vessel closure. There exists a familial predisposition to RS and a high incidence concerning females.
The cause of the RS includes an infection with *nanobacteria,* which increase the local amount of endothelin-1 (ET-1), a potent endothelium-derived vasoconstrictor. Several studies show that the baseline levels and the post-cold stimulus levels of ET-1 are significantly heightened in people with RS. Calcitonin gene-related peptide (CGRP) is an endogenous vasodilator, located in the nerve terminals of unmyelinated sensory afferent fibres in the skin, and exists in higher concentrations in the extremities. In case of a RS-affected skin of the fingers, the CGRP level is in local nerve terminals decreased.
In case of people with RS an infection with *Mycoplasma fermentans* can be often experienced.
Diagnosis: symptomatically.

Treatment: Avoidance is the best preventive measure to be taken against RS attacks. Affected individuals should keep their extremities warm, avoid all known stimuli. Smoking should be avoided. Drugs associated with RS symptoms should be likewise avoided.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent pathogens found, are:
Nanobacterium: 324-325, 375-381, 560-568 kHz
Mycoplasma fermentans: 442-444, 447-451, 493-495 kHz
HTLV virus [redacted]

18.12. The Development of Autoimmune Diseases

Autoimmunity, i.e. the immune reaction against the person's own antigens is a part of the normal immune response and is inhibited by autotolerance. In case of an autoimmune disorder the immune system produces antibodies in great amounts, directed against the patient's own healthy tissues. These autoantibodies may arise following the abrogation of the autotolerance of the normal immune system by exogenous antigens cross-reacting with the body's own tissue-antigens, or, if a damaged immune system loses its capacity to distinguish between foreign and innate elements. Autoantibodies do not necessarily indicate an autoimmune disease. The latter term must be restricted to situations in which the autoimmune response, humoral or cellular, is responsible for a tissue injury. The consequences of the immune response are named immunity, in case it is beneficial for the host, and hypersensivity or allergy if detrimental for the patient.
Infection with *Borrelia B. sensu lato* is one potential cause of the development of an autoimmune disease. The first step in this unusual immunological process occurs when these bacteria invade the tissue. Borrelia has an outer surface and certain specific antigens upon it. The host's immune system responds to the infetion by producing specific antibodies. The Borrelia antigens attached to this outer surface ring absorbs the antibodies of the host. The Borrelia hereafter emerges from its outer surface ring and from the host's tissue as well, so that only the antigen-antibody complex remains there. The immune system recognizes this immune complex and attacks it, but as the live Borrelia species is not there within the ring, the attack turns against the host's own tissue, causing inflammation. Thus, the Borrelia survives the attack, and the false immune response damages the host's tissues. This process can result in the development of an autoimmune disease. In certain phases of borreliosis there are no detectable free antibodies in the serum, as they are in the immune complexes on the tissues. In this way the patient's serologic test will be falsely negative.
In case of a *chlamydial infection* the process appears to be similar. Co-infections with other pathogen microorganisms may also influence the immune reactions. Moreover, certain worm antigens may cause further allergic processes to develop in addition to the existing autoimmune disease.
Autoimmune and degenerative diseases are a highly complex group of diseases, affecting multiple organs. *Mycoplasmal* and/or *Human T-cell Lymphotropic viral infections* are causative factors regarding these chronic syndromes [redacted] Chapter 23.9.).

18.13. Rheumatoid Arthritis

Rheumatoid arthritis is an autoimmune disease in which the joints, mostly those of the hands and feet, are symmetrically inflamed, swollen, painfull, and later on the joint's interior part can be destructed. Many different pathogenic factors, including genetic predisposition, can cause or influence the autoimmune reactions of the host. *Mycoplasma* may have a causative role in the development of rheumatoid arthritis [redacted]

Other important and frequent bacterial provoking factors include *Chlamydias, Borrelia Burgdorferi sensu lato* and *Streptococci. Parasites* may also iniciate this disease.
Symptoms: The onset of rheumatoid arthritis is frequently subtle, affecting gradually various joints. The inflammation of the joints is mostly symmetric. Typically, the small joints of the fingers, toes, hands, feet, wrists, elbows and ankles become inflamed first. These inflamed joints are usually painful and stiff, especially after awakening or after a prolonged inactivity. This morning stiffness lasts characteristically for more than an hour, and over 6 or more weeeks. The swelling of the joints affects three or more joints and lasts more than 6 weeks. Rheumatoid factors (RF), which are autoantibodies against the Fc portion of IgG type antibodies of the patient, can characteristically be found in the blood, so also are the antibodies directed against the citrullinated proteins (f.i. the anti-cyclic citrullinated peptide (aCCP) which have an excellent diagnostic and good prognostic potential for rheumatoid arthritis. The reactivity of these latter antibodies is citrulline-dependent. Typical erosions and decalcifications of the joints of the hands can be experienced by x-ray examinations. These affected joints can get quickly deformed.
Rheumatoid arthritis can cause low grade fever and occasionally vasculitis, too, which can result in nerve damages and leg ulcers. Pleuritis, pericarditis and/or inflammations and scarring of the lungs can also occur, leading to chest pain, difficulty when breathing, as well as causing an abnormal heart function. Some patients develop swollen lymph nodes, Sjögren's Syndrome, and eye inflammations. Rheumatoid arthritis is a disease associated with autoimmune processes. Patients with **Felty's syndrome** have a low white blood cell count, an enlarged spleen and rheumatoid arthritis.
Diagnosis: is based on the characteristic criteria of the disease, established by x-ray examinations, laboratory tests and symptoms. The prognosis, process and therapy of the disease can be monitored by RF or by aCCPs in the serum.
Differential diagnosis: by distinguishing it from other arthritis forms such as from psoriatic arthritis, Reiter's syndrome, lyme arthritis and other reactive arthritis forms.
Treatment: symptomatically, f.i. by administering nonsteroidal anti-inflammatory drugs (ibuprofen, aspyrin), or by using using basis therapy with slow-acting drugs (sulfasalazine, penicillamine, hydroxychloroquine), corticosteroids, and other immunosuppressive drugs (methotrexate) and in most severe cases biologic response modifiers (etanercept, infliximab and anakinra).
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances of Borrelia Burgdorferi s. l. are: 377-388 kHz
The most frequent resonant frequencies of its vegetative forms are: 301-305, 341, 420-422, 555-556 kHz
Other resonant frequencies of the CWD forms of Borrelia are: 300-302, 327-329, 341-342, 412-420, 421-424, 429, 510-511, 547-548, 556, 562-565 kHz
Supposed other borrelial resonances are: 309, 312-319, 336-338, 347, 372, 401, 453, 481-482, 494-496, 513-515, 520, 524, 541 kHz
The resonant frequencies of Chlamydia are: 316-319, 374-386, 429, 440-444, 480-482, 566 kHz
The resonant frequencies of Streptococcus are: 313-321, 358-375, 368-385 kHz
The resonant frequencies of Cytomegalovirus are: 305, 349, 406-412, 512, 534-536, 546 kHz
The resonant frequencies of Mycoplasma fermentans are: 440-452, 493-495 kHz
Other non identified frequencies are: 383, 409, 460, 487, 524 kHz
The most frequent resonances of osteoarthritis are: 313-316, 378-383, 394, 440-452 kHz

18.14. Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a chronic autoimmune connective tissue disease which can affect any part of the body. As it occurs in case of other autoimmune diseases as well, the immune system attacks the body's own cells and tissues, resulting in inflammation and tissue damages. The course of this disease is unpredictable, having periods of illness alternating with remissions depending on the patient's immune state. This chronic inflammatory disease is believed to be a type III hypersensitivity response with a potential type II involvement. In case of SLE the body's immune system produces antibodies against itself, particularly against the proteins present in the *mycoplasma* infected cell nucleus.

SLE can develop in case of genetical predisposition and can be triggered by infectious agents such as *viruses*, *bacteria* and *mycoplasmas*. Mycoplasmal and viral antigens adsorbed to the intracellular or/and extracellular membranes provoke an autoimmune response.

The genetic predisposition is very complex: the most important genes are located in the HLA region on chromosome 6, where mutations may occur randomly or be inherited. HLA class I, II, and III are associated with SLE, but only class I and class II are contributing independently to an increased risk of SLE. Other genes containing risk variants concerning SLE are IRF5, PTPN22, STAT4, CDKN1A, ITGAM, BLK, TNFSF4 and BANK1.

Fcg RIIa binds itself to IgG_2 and is encoded by 2 codominant alleles-H131 (or high affinity) and R131 (or low affinity). The low-affinity phenotype (homozygous for R131 allele; 131R/R) is associated with lupus nephritis in black persons.

Fcg RIIIa binds itself to IgG_1 and is encoded by 2 codominant alleles-V158 (or high affinity) and F158 (or low affinity). The low-affinity phenotype (homozygous for F158 allele; 158F/F) is associated with SLE.

Cytokine genes: Certain polymorphisms of the *IL10* gene (high producers) and possibly the *IL1RN* and *TNFA* genes (low producers) are associated with SLE.

Mannose-binding lectin genes: These gene polymorphisms are associated with an increased risk of SLE.

Apoptosis genes: Defects of several apoptosis genes including *CD95* (Fas) and *CD178* (FasL) are associated with lupus-like syndromes in mice and, rarely, SLE in human beings.

Infectious agents causing SLE: the most frequent infectious agents are *HTLV*, *HBLV* and *Mycoplasma fermentans*. Other different viruses or/and bacteria can play a role in the development of SLE as well.

The development of this multicausal disease is triggered by *viral* infections among genetically predisposed people, mostly women, as it occurs nine times more often in women than in men. *mycoplasma infection* is a causal factor of this syndrome. Abundant evidences show that abnormal immune processes (defects in the function of the complement system, the complement receptors, apoptosis and in the DNS repair mechanism) are also important factors as to its pathogenesis. A hallmark of this disease is the presence of a number of antibodies to nuclear components and other immunological abnormalities as well.

Symptoms:

Its initial and chronic signs are fever, malaise, joint pain, myalgia, fatigue, erythema, depression and systemic vasculitis.

Its dermatological manifestations can be alopecia, nasal and vaginal ulcers. The characteristics of the illness are facial erythematous rashes (vespertilio), sensitivity to sunlight, mouth and nasopharyngeal sores. Raynaud's phenomenon can also occur.

Its skin lesions are characterized by basal cell degeneration, edema of the upper dermis, infiltrated by activated lymphocytes, plasma cells and histiocytes and by an increased vascularity. On the immunofluorescent staining IgG and C3 deposits of the epidermal-dermal junction can be experienced.

Fibrinoid deposits may be found in the blood vessels, among the collagen fibers and on the serosal surfaces. The presence of Hematoxylin bodies found by microscopic examinations shows degenerated nuclei to be interacted with antinuclear antibodies.
Arthritis and arthralgia are frequent symptoms of the illness. Fleeting arthralgia involving usually the joints of hands and feet and also the large joints are often encountered. Redness, warmth, tenderness and synovial effusions are frequently present. Deformities do but very seldom develop, erosions characteristic of rheumatoid arthritis are unusual. Aseptic necrosis may occur, partly due to the therapy using corticosteroids. Profound muscle weakness and tenderness can occur reflecting myositis.
Hematological manifestations such as anemia, haematolytic anemia, low blood cell count, lymphopenia and thrombocytopenia. autoimmune markers in the serum (antinuclear antibodies, aDNA antibodies etc.) are often experienced.
Pulmonary manifestations such as pleura inflammation, pleural effusion, lupus pneumonitis, chronic diffuse interstitial lung disease, pulmonary hypertension, pulmonary embolism, pulmonary hemorrhage and shrinking lung syndrome can also come about.
Cardiac manifestations such as pericarditis, myocarditis, endocarditis, Libman-Sacks endocarditis.
Renal involvement such as painless hematuria or proteinuria, lupus nephritis, glomerulonephritis and glomerular basement membrane damages.
The renal lesions of patients with SLE are classified as being focal glomerulonephritis, diffuse glomerulonephritis and typical membranous lupus nephritis [redacted] Lupus nephritis is one of the most serious illness of SLE. Lupus nephritis is histologically present in most patients with SLE, even in case of those without any clinical sign of a renal disease. In case of a membranous lupus nephritis the basement membranes of glomeruli are diffusely thickened, tubular atrophy and interstitial mononuclear cells can be seen when histologically examined.
The symptoms of lupus nephritis are usually hypertension, proteinuria and renal failure.
Neuropsychiatric manifestations such as neuropsychiatric syndromes, cognitive dysfunction, mood disorder, cerebrovascular disease, seizures, polyneuropathy, anxiety disorder, psychosis, acute confusional state, aseptic meningitis, autonomic disorder, demyelinating syndrome, mononeuritis multiplex, movement diorder, chorea, myasthenia gravis like state, and plexopathy can be present. SLE may resemble epilepsy and certain psychological disorders, too.
SLE most often harms the heart, joints, skin, lungs, blood vessels, *liver*, kidneys, and the nervous system. Systemic lupus erythematosus is a classic autoimmune disease which causes only episodically inflammations in the joints, tendons, other connective tissues and organs.
Uremia, heart failure, hemorrhages, certain severe CNS symptoms and intercurrent bacterial infections can all lead to the patient's death.
Differential diagnosis: by distinguishing it from other connective tissue diseases.
Diagnosis: by the diagnostic criteria of the American College of Rheumatology classification, by examinations of autoimmune markers, etc.
Treatment: being a chronic disease with no known definite healing cure as yet, the treatment of SLE is symptomatic. In case of remission, the illness does only require little or no treatment at all. Disease-modifying antirheumatic drugs can be used preventively in order to reduce the incidence of flares and the need for using steroids. By avoiding sunlight and using creams with 40 SPF.
In case of a more severe state the treatment has to begin immediately by giving corticosteroid (prednisolone, or methylprednisolone). Cyclophosphamide can effectively suppress the autoimmune attack of the body. Combinations of these drugs are mostly used in case of severe kidney and CNS diseases and in case of systemic vasculitis. In certain cases the administering of azathioprine and other immunosuppressive drugs,

hydroxychloroquine (chloroquine and quinacrine), cyclosporin A, plasmapheresis, IVIG, biological response modifier drugs may also be indicated.
RFR method: detects and can eliminate the viruses and bacteria. The first step to be taken is the elimination of mycoplasma and than that of HTLV.
Certain other viruses often found in case of lupus patients are VZV, mumps virus, EBV, CMV and bacteria such as Chlamydia, and Borrelia B. sensu lato.
A small amount of corticosteroids may be needed after eradicating the viruses.
The most frequent resonances of SLE are: 370-376, 442-451, 493-495 kHz
Other often found resonances are: 310-315, 318-319, 324, 338-341, 347, 356, 359-360, 394-397, 407-410, 416, 420, 432-433, 450-452, 462, 471, 482, 492-497, 499, 503, 508-511, 514, 518, 525, 543-544, 553, 558 kHz
Different viruses cause different forms of SLE.

18.15. Sjögren's Syndrome

Sjögren's Syndrome is the most common chronic inflammatory autoimmune disorder caused by a combined attack of *various viruses* and is characterized by excessive dryness of the eyes (keratoconjunctivitis sicca, xerophtalmia), the mouth (xerostomia), and other mucous membranes as well. In some people, only mouth or eyes become dry. The dryness of the eyes may severely damage the cornea, the lack of tears can cause permanent eye damages. Insufficient saliva in the mouth can dull the sense of taste and smell, make eating and swallowing painful, and cause cavities. The lack of secretions may involve the entire respiratory tract, the vagina and skin as well. Sjögren's Syndrome can be diagnosed if any two of the three clinical features: dry eyes, dry mouth, and/or arthritis are observed. Arthritis is often the first symptom of Sjögren's Syndrome. This syndrome is often associated with rheumatoid arthritis, in which case usually the arthritis appears first. Sjögren's Syndrome sometimes is associated with systemic lupus erythematosus, polymyositis, scleroderma and periarteritis nodosa. Also patients with autoimmune liver diseases such as chronic active hepatitis, primery biliary cirrhosis and cryptogenic cirrhosis can suffer Sjögren's Syndrome.
The earliest histological findings of this disorder are periductal hyperactive lymphatic cell infiltrations leading to atrophy of the acini. Hyperplasia and hyperactivity of the ductal lining cells causes narrowing and obstruction of the duct and leads to its dilatation. The development of lymphadenopathy and extrasalivary lymphoid abnormalities of patients with this syndrome may suggest the development of a malignant lymphoma, though this disorder can be classified to be between neoplasia and hyperplasia, named pseudolymphoma. Later on, in the course of the disease, the atrophied parenchymal tissue is replaced by an adipose one.
Occasionally, patients experience anterior chest pain caused by a thoracic skeletal involvement mimicking angina pectoris. The manubriosternal and sternoclavicular joints may also cause chest pain. Dysphagia, atrophic gastritis, pancreatitis, chronic hepatitis, hypergammaglobulinaemic vasculitis and interstitial nephritis can also come about.
Blood tests can detect SS-B autoantibodies, which is highly specific for this syndrome. Patients can produce rheumatoid factors and antinuclear antibodies, too. The histological and immunological findings suggest that abnormalities of both humoral- and cell-mediated immunity are involved in the pathogenesis of this syndrome.
Its symptoms may be persistent or intermittent for months or years.
Diagnosis: symptomatically and by Schirmer test.
Differential diagnosis: by distinguishing it from other connective tissue diseases and from Reiter's syndrome.
Treatment: symptomatically, by administering NSAIDs and oculoguttae in order to hinder ocular symptoms.

RFR method: detects and eradicates the viral and/or bacterial components, the combination of which may be the cause of this disease.
Its most frequent resonances are those of the
Adenovirus: 370-387, 390-392, 393, 394-400 kHz
Cytomegalovirus: 305, 327, 349, 408-411, 530-536 kHz
Epstein-Barr Virus: 337, 339, 347, 372-383, 518-519 kHz
Mycoplasma fermentans: 442-451 kHz

18.16. Antiphospholipid Syndrome (APS)

There are theories concerning the role of infectious agents in the provoking of the production of antiphospholipid antibodies (aPL) and in the development of the antiphospholipid syndrome (APS). The antiphospholipid syndrome, named also antiphospholipid antibody syndrome is a disorder of coagulation, causing blood clots (thrombosis) in arteries and veins, as well as pregnancy-related complications such as miscarriage, preterm delivery and severe preeclampsia. This syndrome is characterised by producing autoantibodies to phospholipid-binding plasma proteins, rather than to phospholipid itself, a cell membrane component. More over, the disease is characterized by producing antibodies against cardiolipin (anti-cardiolipin antibodies) and β_2 glycoprotein-I, which latter is the most important among the phospholipid-binding proteins, including prothrombin, proteins C and S and Annexin V. The term *„primary antiphospholipid syndrome"* is used in case of APS occuring in the absence of any other related disease. APS is commonly seen associated with other autoimmune diseases; so that the term *„secondary antiphospholipid syndrome"* is used, if APS coexists with these diseases, f.i. with SLE and other autoimmune disorders, the origin of which is an infection caused by certain pathogenic microorganisms.
Sometimes, APS leads to rapidly developing organ failures due to generalised thrombosis with a high risk of death; termed Catastrophic antiphospholipid syndrome.
There exists many a term for APS. Some of its synonyms can be confusing, f.i. the term Lupus anticoagulant (LA) syndrome is misleading, as patients with APS may not necessarily have SLE and this LA is associated with thrombosis rather than with hemorrhagic complications. To attempt further confusions, APS is nowadays the preferred term for this clinical syndrome.
APS is an autoimmune syndrome caused by coexisting infections with Mycoplasmas, HTLV, CMV and/or EBV. The infectious species of the Mycoplasmas are mostly *M. penetrans* and rarely *M. fermentans* and *M. pneumoniae*, etc. *HTLV-1* and *HTLV-2* are frequently present, HTLV-3, HTLV-4 and HTLV-5 are but rarely present in APS. The trigger of this syndrom can be the coexisting autoimmune and rheumatic diseases, infections and drugs, causing LA antibodies, and/or anticardiolipin antibodies. The presence of these autoantibodies does not always associate with the symptoms of APS.
Supposed characteristic mechanisms of this syndrom: Being bound to phospholipid-binding proteins, aPL antibody may interfere with the maintenance of coagulation homeostasis. Antiphospholipid can also activate endothelial cells, induce the activation of monocytes, and interact with placental Annexin V in case of pregnant women. Complement activation may be an additional mechanism causing the loss of fetus. According to a hypothesis infectious agents can induce, via a molecular mimicry mechanism, the production of aPL antibodies in the host.
The familial occurrence of aPL antibodies suggests a genetic association with HLA-DR4, DR7 and DRw53 haplotypes.
As to the clinical symptoms of this syndrome, see Chapter 14.62
Mycoplasmal and HTLV infections can develop an autoimmune response, see the special Chapter. These infections and the familiar genetic predisposition may lead to APS. I have found these infections mentioned above among APS patients by RFR technique.

Diagnosis: symptomatically, by finding aPL antibodies and abnormalities in phospholipid-dependent tests of coagulation.
Treatment: symptomatically, f.i. by administering intravenous heparin followed by warfarin in case of thrombosis, subcutaneous heparin and aspirin in case of pregnant women with a history of recurrent fetal loss, etc. The same prophylactic therapy can also be advisable f.i. concerning patients with SLE, etc. The coexisting mycoplasmal infection should be treated by administering effective antibiotics.
RFR method: detects and may eliminate the pathogen microorganisms!
The most frequent resonances of Mycoplasma are: 307-308, 321-324, 342-350, 442-451, 491-495 kHz
The most frequent resonances of HTLVs are: 297-299, 307, 311-315, 320-340, 354, 359, 365-367, 370-376, 382-383, 397-400, 406, 416, 428-439, 453-455, 474-476, 480-482, 484, 487-490, 493-504, 523-530, 540-545, 570-578 kHz
The most frequent resonances of EBV are: 372-383, 518-519 kHz
The most frequent resonances of CMV are: 408-410, 530-536 kHz

18.17. Chronic Myopathy, Myositis and Myalgia

A diffuse pain can be felt and be caused by inflammation or other problems associated with the muscles, fascia, fibrous tissues, aponeuroses and the muscle-effecting nerves. Only clinical facts are available: a muscle, or a group of muscles becomes tender and painful after exposure to cold, dampness or minor trauma, or even for no discernible reason at all. Neck and shoulders are the most commonly affected parts of the body. *Coxsackie virus A9* and/or *A3-4* are known to cause this type of myositis, though the same symptoms can be caused by *ECHO viruses types 2, 3, 6-9, 11-14, 18, 19*, and *22-24*. If this muscle pain is remarkably intense, and is localized to especially one group of muscles, the most likely diagnostic possibility is epidemic myalgia, also designated as epidemic pleurodynia, devil's grip, painful neck, or Bornholm disease caused by Coxsackie virus B3 and B5. Poliomyelitis, influenza, brucellosis, Colorado tick fever, denge fever, rheumatic fever, relapsing fever, malaria, measles, borreliosis, etc. are all diseases associated with transient or chronic muscle disorders.

18.18. Acute Viral Myositis

Myositis is an inflammation of the muscles. There are two types of myositis: i.e. polymyositis and dermatomyositis. Polymyositis can be an acute viral and a chronic, autoimmune polymyositis. The causative pathogens of acute viral polymyositis has not yet been identified, but their resonance frequencies can be measured. Some individuals suffer muscle pain. Fatigue, fever and poor appetite are all common signs of this illness. The causative virus infiltrates the muscles, joints and the surrounding soft tissues. Arthralgia may also be present.
In case of these diseases, the inflammatory cells (lymphocytes and later on plasmocytes and granulocytes) surround, invade and destroy the normal muscle fibers as if they were defective, or foreign to the body. In some rare cases, inflammation of the heart, lungs, intestines and skin may also occur. These diseases occur but rarely, but if, they cause fatigue and muscle weakness among children and adults. Occasionally, individuals with acute viral myositis have associated lung symptoms, such as cough and difficulty when breathing. If the infection affects the musculature of the esophagus difficulty in swallowing and a feeling of heartburn can be experienced.
The treatment of myositis often improves the symptoms, but if getting chronic, they need continued therapy for several years. An acute viral myositis can grow into an autoimmune chronic inflammatory myopathy. As regards polymyositis and dermatomyositis [illegible]

Diagnosis: symptomatically, by physical examinations, blood tests to detect muscle inflammations. By further studies of the nerves and muscles, muscle biopsy specimens, electromyography examinations and MRI.
Treatment: symptomatically. By administering corticosteroids, methotrexate, or azathioprine. Treatment may to be continued for several years.
RFR method: detects and may eliminate the viruses.
The most frequent resonances of acute viral myositis are: 286-290, 294-305, 308-323, 390, 407-409, 442-451, 528-533, 574-576 kHz
RFR method is the main form of treatment for this viral infection. If the causative virus is eliminated, an autoimmune process will not develop, neither will chronic inflammatory myopathies, such as polymyositis and dermatomyositis develop.

18.19. Polymyositis and Dermatomyositis

Polymyositis (PM) is a chronic autoimmune disease of the connective tissue, characterized by the inflammation and degeneration of the muscles. Dermatomyositis (DeMy) is an autoimmune polymyositis accompanied by skin inflammation. *Viruses, mycoplasmas* and autoimmune reactions play a role in the development of these illnesses. PM, DeMy, and Inclusion body myositis are important members of idiopathic inflammatory myopathies. Clinical features, characteristic muscle biopsy findings, immune markers, and histopathologic findings differentiate these illnesses from one another. PM and DeMy are inflammatory myopathies causing symmetric muscle weakness developing over weeks to months. Inclusion body myositis is the most common inflammatory myopathy showing but minimal inflammations, affecting patients older than 50 years. In case of PM and DeMy *mycoplasmal and HTLV* infections trigger the T-lymphocytes and cause immune-mediated muscle inflammations and vascular damages. In case of PM, the immune system attacks certain previously unrecognized muscle antigens. In case of DeMy a complement-mediated damage of the endomysial vessels and of the microvasculature of the dermis is the cause of the clinical symptoms.
Symptoms: can begin with high fever and with painless, tender, symmetric proximal muscle weekness and loss of weight. Later on, muscle atrophy, dysphagia, arthralgias, difficulty when kneeling, climbing or descending stairs, raising arms, and arising from a seated position, when holding the head up can develop.
Characteristic rashes of the face, trunk and hands is only to be seen in case of DeMy. Erythematous nail beds and scaly, purple erythematous papular eruptions (Gottron papules) above the dorsal metacarpophalangeal and interphalangeal joints, periorbital purple-red macular eruptions (a heliotrop rash) are characteristic. Extramuscular manifestations, such as congestive heart failure, arrhythmia, lung disease, dysphagia, arthralgias, vasculitis may also come to pass.
Family history and medication history can exclude other causes of myopathy. Inherited mycoplasmas and HTLV infections may exist. *Borreliosis, toxoplasmosis, mycoplasmal, chlamydial infections, HTLV, CMV and other viral infections* attacking together can cause PM and DeMy to develop. Both diseases can be associated with malignancies.
Diagnosis: by muscle biopsy observed under microscopy, electromyography, MRI, CT, chest radiography, ultrasonography and by biochemical examinations such as creatin kinase, aldolase in the serum, etc.
Treatment: Symptomatically, by administering corticosteroids and cytostatics such as methotrexate and cyclophosphamide. Certain patients can not respond to these therapies.
RFR method: detects and may eliminate all pathogen microorganisms. Polymyositis apparently involve different disease mechanisms from those of dermatomyositis.
The most frequent resonances are: 288-317, 319-325, 334, 336-346, 372-387, 416-420, 401-409, 429, 442-451, 480-482, 508-511, 518, 538-539, 548, 556, 565 kHz

18.20. Fibromyalgia Syndrome

Fibromyalgia syndrome (named also myofascial pain syndrome, fibromyositis), is a syndrome characterized by pain and stiffness of the soft tissues, including the muscles, tendons and ligaments. This fibromyalgia may touch the whole body or may be restricted to certain locations, as is f.i. in case of myofascial pain syndrome. Fibromyalgia affecting the whole body attacks mostly women, 20-50 of age. Men are more likely to develop myofascial pain or fibromyalgia in a particular area, affecting f.i. one shoulder, which pain may be caused f.i. by an occupational or recreational muscle strain. As regards the **primary fibromyalgia syndrome**, its causatives are unknown. This syndrome usually occurs among young women and juveniles who are depressed, anxious or stressed, and often suffer from interrupted, not restoring sleep, though prevously they had been healthy. *Mycoplasmal* infection may be a causative factor of this syndrome See Chapter 13.9.

As regards the **secondary fibromyalgia**, the cause of which can be identified, the intensive pain usually tends to be confined to a specific area, beginning suddenly. In both types, the pain usually worsens with straining and overuse. The affected areas hurt when touched, muscle tightness and spasms often come to pass. Although any of the fibrous tissues or muscles can be affected, those of the neck, shoulders, chest and rib cage, lower back and thighs are especially prone to pain. Reducing stress can sometimes alleviate the pain.

Secondary fibromyalgia may be triggered by physical or mental stress and certain infections, and can be associated with rheumatoid arthritis or some other related disorders.

Diagnosis: Determining whether pressure produces pain at one tender point or whether the pain seems to travel to other areas or trigger points.

Treatment: Occasionally, local anesthetics with corticosteroids injected directly into the tender area can be effective, thereas NSAIDs are usually of no use.

RFR method: detects and eradicates the pathogens. Fibromyalgia has a typical resonance frequency, microorganisms causing this syndrome are, as follows:

The most frequent pathological resonant frequencies in fibromyalgia syndrome are: 310, 334, 339, 348, 370-372, 378-383, 409, 442-451, 474, 488, 535-545, 557 kHz

18.21. Osteomyelitis

Osteomyelitis is a bone infection usually caused by bacteria and sometimes by fungi. The bones, which are usually well protected from infection, can become infected in three ways: via the bloodstream, via direct invasion, and by spreading from the adjacent soft tissue. The bloodstream may carry the infection from other parts of the body to the bones. The bacteria of *tuberculosis* can infect f.i. the vertebrae. Pathogens may invade the bones directly through open fractures, during bone surgery, or by contaminated objects piercing the bones.

Staphylococci are responsible for the majority of cases of acute osteomyelitis. This infection attacks mostly children under twelve, adults are especially susceptible to acute osteomyelitis of the spine. The frequent locus of the illness is the diaphyseal end of the long bones, may be owing to the endarterial circulation of the diaphysis. Rarely, the joint capsule is penetrated, causing pyogenic arthritis. In case of bone necrosis, there will a sequestrum be developed, followed by the formation of a new bone, named involucrum. Occasionally, an indolent subacute staphylococcal infection of the bone can remain localized within a central necrotic cavity surrounded with granulation tissues. A local infection like this can persist for years and is named Brodie's abscess. Osteomyelitis caused by fungi develop only occasionally, mostly among immunocompromised patients and are usually caused by *coccidioides* and *blastomyces* species.

Symptoms: The area above the bone may be sore and swollen and painful when moving. Fever, continous pain and leukocytosis can also occur. This pain worsens when moving and can not get relieved by resting or by applying heat. Osteomyelitis caused by the

infection of adjacent soft tissues or by direct invasion causes pain and swelling in the area above the bone, abscesses may also be formed in the surrounding tissue. If treated unsuccessfully, there can develop a chronic osteomyelitis.
Diagnosis: by x-ray, CT, MRI, by identification of the causative bacteria or fungi.
Differential diagnosis: Ewing's sarcoma and other bone tumors, osteoarthritis, acute rheumatic fever and pyogenic arthritis.
Treatment: by administering effective antibiotics and antifungal drugs and by surgery.
RFR method: detects and eliminates the pathogens.
The most frequent resonances are: 287-290, 294-300, 313-319, 340, 348, 353, 372, 383-384, 396-397, 403, 409, 425, 442-451, 463-464, 513-514, 544, 555 kHz

18.22. Osteoarthritis

Osteoarthritis (named also degenerative arthritis), is a chronic joint disorder characterized by the degeneration of the joint cartilages and by the decrease of the synovial fluid lubricating this joint, and can cause pain and stiffness of the affected joint. Osteoarthritis is the most common joint disorder, affecting to some degree people seventy years old. Men and women are equally affected, but in case of men the disorder tends to develop at an earlier age. Osteoarthritis can be either

– a primary or a so-called idiopathic process, which occurs in case of a combination of different bacterial and/or viral infections, such as *Borrelia B. sensu lato*, *Chlamydia*, *Mycoplasma* and *Nanobacteria*, or
– a secondary one, in which case, its cause is another disease, such as Paget's disease.

The joint cartilage has unique properties of compressibility and elasticity due to the combined presence of collagen fibers and proteoglycans. Collagen cartilages occupy the place of the hyaline cartilages. The articular cartilage contains a unique type of collagen, named type-II collagen.
In normally developed cartilages, chondrocytes do not synthesize DNA and do not divide anymore. The pathogenic events of osteoarthritis reactivate DNA synthesis and cell division. Chondrocytes getting metabolically active continuously rebuild the matrix of cartilage. The early pathologic changes of osteoarthritis are recognizable in the joint cartilages. New bone formation develops in the subchondral bone and at the margins of the articular cartilage. Low-grade inflammatory changes are present in the synovium and the joint capsule, the latter thickening with fibrosis.
The **symptoms** usually develop gradually and affect at first only one or a few joints. Joints of the fingers, base of the thumbs, neck, lower back, large toes, hips and knees are commonly affected. The first symptom is the worsening of the pain by exercising. After sleeping, or some other inactivity, the joint may be stiff, but the stiffness usually subsides within 30-40 minutes after moving.
Bony growths named Heberden's nodes commonly develop in the joints at the ends of the fingers. Similar deformities are the so-called Bouchard's nodes at the proximal interphalangeal joints.
Osteoarthritis frequently affects the spine, mild back pain and stiffness being its most common symptom. However, osteoarthritis in the neck or lower back can cause numbness, odd sensations, pain and weakness in an arm or leg, if the overgrowth of the bone presses the nerves. Rarely, the blood vessels supplying the back of the brain are compressed, causing vision problems, a whirling sensation, nausea and vomiting.
Diagnosis: by x-ray
Differential diagnosis: by distinguishing it from rheumatoid arthritis, gout, pseudogout, Paget's disease and other infectious arthritic diseases.
Treatment: by administering acetaminophen and other analgetic drugs such as NSAIDs,

COX-2 inhibitors, corticosteroid injections into the affected joints, glucosamine and chondroitin sulfate, by getting prostheses.

RFR method: detects the resonances and eliminates the microorganisms. Osteoarthritis has typical resonance frequencies caused by microorganisms.

The most frequent resonances in osteoarthritis are: 313-316, 378-381, 384-386, 394, 442-451, 488-502 kHz

The pathogen microorganism is maybe a non-determined type of Chlamydia, Mycoplasma, or some other specific virus.

18.23. Osteoporosis

Osteoporosis is a disorder of diverse etiology and is characterized by the reduction in the mass of the bone structure, which mass is required for an adequate mechanical support function. Histologically, osteoporosis is characterized by the decrease in number and size of the trabeculae of the affected bone, with normal width of the osteoid seams. Osteoporosis is the most common metabolic bone disorder involving the entire skeleton presumably owing to effects of systemic factors acting on the skeleton. Osteoporosis frequently causes morbidity in elderly patients. The bones contain minerals such as calcium and phosphorus, making them hard and dense. To maintain bone density, the body requires an adequate supply of calcium and other minerals and must produce a proper amount of several hormones, such as parathyroid hormone, growth hormone, calcitonin, estrogen in women, and testosterone in men. An adequate supply of vitamin D is needed to absorb calcium from food in order to incorporate it into the bones.

The bones of people being over forty years old slowly decrease in density. If the body is not able to regulate the mineral content of the bones, they become less dense and more fragile, getting osteoporotic.

The cause of the age-associated decrease in the mass of the bones, the increase in the bone resorption, and the osteoporosis occurring among women following their menopause is not known for sure. Solely the cause of the osteoporosis associated with endogeneous, or exogeneous Cushing's syndrome is clear. Although the occurrence of osteoporosis together with other disorders is a frequent association, the related mechanism leading to this kind of osteoporosis is not yet known.

There are several different types of osteoporosis:

Postmenopausal osteoporosis is caused by the lack of estrogen, the main female hormone, which helps to regulate the incorporation of calcium into the bones in women. Usually, symptoms develop in women approximately at the age of fifty, but can begin earlier or later as well. Not all women are at an equal risk to develop postmenopausal osteoporosis.

Senile osteoporosis is probably caused by an age-related calcium deficiency and an imbalance between the rate of the bone breakdown and the formation of new bones.

Secondary osteoporosis can be caused by other clinical illnesses, f.i. chronic renal failures, hormonal disorders (Cushing's syndrome, thyreotoxicosis) and/or by drugs, f.i. corticosteroids, barbiturates and anticonvulsants. Excessive alcohol consumption and cigarette smoking may worsen the condition.

Disorders and conditions where osteoporosis is a common feature and where its pathogenesis is partially understood include. hypogonadism, hyperadrenocorticism, thyreotoxicosis, malabsorbtion, malnutrion, scurvy, calcium deficiency, immobilization and systemic mastocytosis.

Disorders in case of which osteoporosis is associated, but the pathogenesis is not understood include f.i. rheumatoid arthritis, epilepsy, diabetes and Menkes' syndrome.

Idiopathic juvenile osteoporosis is a rare type of osteoporosis the cause of which has not yet been identified. It occurs among children and young adults having normal hormone levels and functions, normal vitamin levels, and no obvious reason to have weak bones.

Heritable disorders of the connective tissues associated with osteoporosis include osteogenesis imperfecta, homocystinuria, Ehlers-Danlos syndrome and Marfan syndrome.
Symptoms: initially osteoporosis has no symptoms. If the density of the bone decreases so that the bones collapse or break, pain and deformities develop. The collapse of vertebrae causes chronic back pain. If several vertebrae break, an abnormal curvature of the spine may develop, causing muscle strain and soreness. One of the most serious fractures is the fracture of the hip, causing often disability of the affected elder people.
Diagnosis: symptomatically, by x-ray, x-ray absorptiometry, and hormone level measurements.
Prevention: is far more successful than treatment, meaning the maintaining and increasing bone density by consuming an adequate amount of calcium and engaging in weight-bearing exercises. Replenishment of the so-called friendly intestinal flora is essential. This flora is needed for the recycling of the natural estrogen of women. Soya estrogen can also help.
Treatment: is aimed at increasing the density of bones. Raloxifen, alendronate, calcitonin, fluoride derivates, vitamin D, and hormone treatment can be administered.
RFR method: detects and eliminates the intestinal pathogens.
The most frequent resonances in osteoporosis are: 307, 318-326-339, 332-334, 372, 388-392, 397-402, 408-416, 448-451, 460 kHz
Following the RFR method friendly bacteria complex should be given: f.i. Lactobacillus bifidus, L. acidophilus, L. plantarum, L. casei, L. bulgaricus, Bifidobacterium longum, B. lactis, B. breve, Streptococcus termophilus in combination.

18.24. Paget's Disease

Paget's disease, also named Osteitis deformans, is a chronic skeletal disorder typically resulting in enlarged and deformed bones. Its characteristic feature is an increased resorption of bone accompanied by increasing bone formation, which in case of healthy people are always adequately compensated. The early stage of Paget's disease is characterized by bone resorption caused by a predominant osteoclastic activity. This disease is but rarely diagnosed in its initial lytic phase. Unless it occurs in the tibia, Paget's disease usually begins on the end part of a bone. A characteristic sharply demarcated zone of osteolysis may begin in the subcortical bone and advance along the diaphysis. Osteoblastic activity lags behind; so that a radiolucent fibrous tissue replaces the normal bone. The intermediate or mixed phase reveals the evidence of an osteolytic and disorganized osteoblastic activity. The new bone develops abnormally, demonstrates characteristically a coarsened trabecula and a cortical thickening in the cancellous and compact bone. The final or cold phase of the disease is characterized by dense lamellar bone formation, by a negative external calcium balance, the bones are exceedingly vascular.
The excessive breakdown and the formation of bone tissue occuring in case of Paget's disease cause weak bones, bone pain, arthritis, deformities and fractures.
Etiology: An environmental trigger of Paget's disease has been since a long time considered, but never proven. *Measles-viral* messenger RNA sequences were found in osteoclasts and other mononuclear cells of pagetic bones. *Canine distemper virus* nucleocapsid antigens are also found in osteoclasts from patients with Paget's disease. However, the presence of these paramyxoviruslike nuclear inclusions do not prove that these are responsible for the development of pagetic lesions; but may rather be markers of the disease itself.
Bone biopsies of patients with Paget's disease demonstrate the presence of several different Paramyxoviridae viral antigens, (measles virus and RSV) and/or of other viruses, such as HTLV and HPV located within the osteoclasts.

An infection with Mycoplasma species plays a very important. role in the development of Paget's disease. Thus, Paget's disease may perhaps be caused by a combined *paramyxoviral* infection (such as *measles*, *Canine Distemper Viral*, *Respiratory Syncytial viral)* and/or *Coxsackie* viral infections together with a former *HTLV, HPV* and *Mycoplasmal* infection, already present many years before the symptoms of Paget's disease appear.

Genetic predisposition can also be supposed to be present, as several genetic theories suggest the role of the gene of human leukocyte antigen (HLA) on chromosome 6 and the gene on chromosome arm 18q, though there does exist a genetic heterogeneity.

Symptoms: The clinical signs of patients with Paget's disease are widely various, depend on the extent of the disease, the particular bones involved and the presence of associated complications. Many patients are asymptomatic. In case of these persons the disorder is discovered by radiological findings during the course of examination of the pelvis or spine for an unrelated disease or complaint, or owing to the elevated level of plasma alkaline phosphatase found. Others may gradually become aware of the swelling or deformity of one of their long bones or may develop a disturbance in their gait due to the unequal length of their bones, and experience the change in the distribution of forces in their lower extremities. The most frequent complaint is pain, most commonly in the back and hip, followed by pain in the long bones and pelvis. Weight bearing may exacerbate pain in the spine, pelvis, or in the lower extremity. Disease in the skull can be accompanied by headache. Deafness too can occur, caused by cranial nerve compression or by middle-ear ossicle involvement. Pain may be present due to secondary arthritis or nerve compression. Secondary arthritis most often affects the hips, knees and ankles. Lower extremity limb shortening may be secondary to the bending of the tibia and femur.Patients suffer usually dull, sometimes shooting, or knife-like pains. Their pain in the lower extremities may be associated with transverse cortical infractions, which occur along the convex lateral surface of the femur, or the anterior surface of the tibia. Joint cartilage damages can lead to arthritis.

Angioid streaks of the retina have been observed among patients with Paget's disease. The loss of hearing can be caused by the direct pagetic involvement of the ossicles of the inner ear, or by the effect on the eighth cranial nerve of the pagetic bone, narrowing the auditory foramen. Even more serious neurological complications can result from the overgrowth of the pagetic bone. Compression of the spinal cord with paraplegia can also be observed, mostly if the middorsal spine is involved. Pathological fractures of the vertebrae can also produce spinal lesions. Calcium excretion tends always to be higher if the resorptive phase predominates. This factor may account for the somewhat higher incidence of urinary stones of these patients. Hyperuricemia and clinical gout occur commonly among men with Paget's disease, and calcific periarthritis can also often be found. Osteogenic sarcoma, a form of bone cancer, is a rare complication occuring in less than one percent of these patients.

Diagnosis: by x-ray, alkaline phosphatase level examinations, auditory tests, etc.

Treatment: by administering bisphosphonate etidronate, pamidronate, alendronate, etc and calcitonin. Paget's disease lesions respond often well to steroid therapy.

RFR method: detects and can eliminate the pathogen microorganisms.

The most frequent resonances of:

Coxsackie virus are: 286-290, 294-301 kHz

HPV are: 427-438, 446-447, 452-453, 470-473 kHz

Mycoplasma fermentans are: 442-451, 493-495 kHz

Measles virus are: 350, 364-373, 381-387, 390, 402-407, 450-456, 478, 492, 522-536, 564 kHz

Respiratory Syntitial Virus are: 378-383 kHz

HTLV are: 370-376 kHz

18.25. Adiposis Dolorosa Syndrome (Dercum Disease)

Adiposis dolorosa syndrome (ADS) consists of 4 cardinal **symptoms:**

1. multiple, painful, fatty masses, subcutaneus lipomas;
2. generalized obesity, usually in the menopausal age;
3. asthenia, weakness, and fatigability and
4. mental disturbances, including emotional instability, depression, epilepsy, motor weakness, confusion, dementia and peripherial neuropathy.

Associated conditions are sometimes sleep disturbances and the pickwickian syndrome; slight-to-moderate dryness of eyes and mouth, in spite of normal tear production a gritty feeling in the eyes, irritable bowels; coccygodynia; vulvovaginitis; vulvodynia; carpal tunnel syndrome; Tietze syndrome; chondromalacia patellae; thyroid malfunction (mainly hypothyreosis); trochanteritis; localized tendonitis and fibromyalgia. Adenomas in the pituitary, thyroid and adrenal glands can also be experienced.

Asymmetrical pain of the thighs, knees and the upper extremities does also come about. Pain can be felt in the skeletal system and in the fatty tissues . The pain is temperature- and weather-dependent; decreasing in case of dry heat and high air pressure. Hot baths can have a positive but short-term effect in the relief of pain. Estrogen replacement at menopause does not reduce the pain.

Genetic predisposition: Dercum's disease is believed to be transmitted in an autosomal dominant manner; it is particularly present in the descending line of the female members of the affected families.

Infections are determinative factors in the ADS. The most frequent determinative infections are caused by Mycoplasma species, HTLV, HPV, CMV and certain other viral infections combined with bacterial infections, all of which can influence the symptoms of the ADS.

Diagnosis: symptomatically and by histology.

Treatment: symptomatically, as there is no specific therapy for this illness.

RFR method: detects and can eliminate all present pathogens.

The most frequent resonances are: 310, 314-319, 329, 334, 343-350, 353-356, 362-365, 370-374, 378-386, 402-410, 442-451, 474-475, 493-495, 504, 520, 543-545, 557 kHz

18.26. Weber-Christian Panniculitis

Panniculitis is a group of diseases the hallmark of which is the inflammation of the subcutaneous adipose tissue. Its symptoms are tender subcutaneous nodules and certain systemic signs such as weight loss, fatigue and sometimes fever. Similarly to SLE and scleroderma, panniculitis is a connective tissue disorder.

Weber-Christian panniculitis (also named idiopathic lobular panniculitis) is an inflammatory disorder characterized by a recurring inflammation in the fat layer of the skin. The areas involved manifest themselves as recurrent crops of erythematous, sometimes tender, edematous subcutaneous nodules. The lesions are symmetrically distributed, affecting mostly the thighs and the lower legs. Malaise, fever and arthralgia occur frequently. Nausea, vomiting, abdominal pain, weight loss and hepatomegaly can also come about. Acinous adenocarcinoma, the most common pancreatic neoplasm is often associated with nodular liquefying panniculitis. Weber-Christian panniculitis is caused by a combined viral and mycoplasmal infection developing elevated levels of circulating immune complexes, suggesting an immun-mediated reaction. The similarities between the Weber-Christian disease and the Alpha1-antitrypsin deficiency disorder suggest that an altered regulation of a normal inflammatory process might be involved. Lupus panniculitis, factitial panniculitis, panniculitis associated with a pancreatic disease, histiocytic cytophagic panniculitis and alpha1-antitrypsin deficiency panniculitis differ from the

Weber-Christian disease. Weber-Christian panniculitis is a systemic disease, can involve the lungs, heart, the intestines, spleen, kidney, and the adrenal glands. In case of patients with inflammation involving the visceral organs, serious symptoms and mortality can occur.

In case of patients suffering solely from subcutaneous manifestations, the clinical course is characterized by exacerbations and remissions of the subcutaneous lesions lasting for several years before the disorder subsides.

Diagnosis: By biopsy and histological analysis. Its histology is characterized by a granulation tissue–like capillary proliferation and by septal widening secondary to granulomas and fibrosis, while a mild inflammation, lymphocytic cells and giant cells, neutrophil and eosinophil granulocytes can also be observed. The endothelial cells of the small vessels proliferate and can fill the entire lumen. The capillaries get coiled and swollen. Little or no vasculitis or phlebitis at all can be present.

Differential diagnosis: by distinguishing it from Vilanova disease, tuberculosis, lepra, erythematodes and sarcoidosis.

Treatment: symptomatically, as there is no specific treatment.

RFR method: can detect and eliminate the pathogen microorganisms.

The most frequent resonances are: 310, 314, 318, 343-347, 359-365, 370-374, 428-437, 442-451, 503-513, 543 kHz

18.27. Marfan Syndrome

Marfan syndrome is an inherited disorder affecting the connective tissue and causing abnormalities of the eye, as well as of the cardiovascular and the musculoskeletal system. Its inheritance is carried by a gene named FBN1, which encodes a connective protein called fibrillin-1. Its inheritance occurs in a dominant way, persons, who have inherited one affected FBN1 gene from either of their parents will have Marfan syndrome. Each of the affected parents has a 50% risk of passing the genetic defect on to any of his/her children owing to the autosomal dominant nature of inheritance. 5-10 percent of the Marfan syndrome cases is not inherited but is associated with a de novo genetic mutation in the FBN1 gene on chromosome 15, which mutation is caused by *Mycoplasma fermentans* infection of the pregnant women occuring in the first trimester of their pregnancy.

FBN1 gene encodes the fibrillin-1 glycoprotein, a component of the extracellular matrix. This protein is essential for the proper formation of the extracellular matrix including the biogenesis and the maintenance of elastic fibers. The extracellular matrix is critical for the structural integrity of the connective tissue and serves also as a reservoir for certain growth factors. Elastin fibers can be found throughout the body but are particularly abundant in the aorta, the ligaments and the ciliary zonules of the eyes; so that these areas are the worst affected ones.

Transforming growth factor beta (TGFβ) protein plays an important role in Marfan syndrome. Fibrillin-1 can indirectly bind the latent form of TGFβ and keep it sequestered and unable to exert its biological activity. The mode, how elevated TGFb levels are responsible for the specific pathology of this disease is not proven as yet, though an inflammatory reaction releasing proteases slowly degrading the elastin fibers and other components of the extracellular matrix is known to occur. The importance of TGFb was confirmed by the discovery of a similar syndrome i.e. the Loeys-Dietz syndrome involving the TGFβR2 gene on chromosome 3 producting a receptor protein of TGFβ. Due to the considerable clinical overlap between these two syndromes the Marfan syndrome has often been confused with the Loeys-Dietz syndrome.

The skeleton of Marfan syndrome patients shows typically multiple deformities including arachnodactylia (ie. abnormally long and thin digits), dolichostenomelia (ie. long limbs

relative to trunk length), pectus deformities (ie. pectus excavatum and pectus carinatum) and thoracolumbar scoliosis.
Aortic dilatation, aortic regurgitation (mitral valve prolapse) and aneurysms are the most common clinical findings concerning the cardiovascular system.
Its ocular symptoms include myopia, cataract, glaucoma, retinal detachment and the superior dislocation of the lens.
Marfan syndrome patients have excessively long arms and legs, their arm span being greater than his or her height. Their fingers and toes may be long and slender, with loose joints that can be bent beyond their normal limits. The face may also be long and narrow and he or she can have a noticeable curvature of the spine. Marfan syndrome patients can vary widely in their external signs and in severity; even two patients belonging to the same family may look quite different. Most of the external features of the Marfan syndrome become more pronounced as the patient gets older, so that the diagnosis of the disorder is often easier to establish in case of adults than in case of children. If the patient has but minor outward signs of the disorder, the diagnosis may be missed until the patient develops vision problems or cardiac symptoms.
Marfan syndrome itself does not affect a person's intelligence or his/her ability to learn. Obstructive sleep apnea refers to a partial obstruction of the airway while sleeping, causing irregular breathing and sometimes snoring. Obstructive sleep apnea can also occur, caused by the unusual flexibility of the tissues lining the Marfan syndrome patient's airway.
Diagnosis: there is no objective diagnostic test for Marfan syndrome as yet.
The diagnosis is based on the family history and on the skeletal, ocular and cardiovascular signs.
Treatment: symptomatic.
RFR method: the most common resonant frequency present in this syndrome is that of
Mycoplasma fermentans: 442-451, 493-495 kHz
Other 287-290, 294-301, 313-315, 317-319 kHz

INFECTIONS OF SPECIAL ORGANS

19. BLOOD DISORDERS ASSOCIATED WITH INFECTIONS

19.1. Blood Infections

In case of blood infections viruses, bacteria and parasites remain spreaded in the bloodstream for a while.

19.1.1. Viral Hemorrhagic Fever

Hemorrhagic fever caused by viruses are commonly experienced in tropical areas. These infections can cause mild and debilitating, potentially fatal diseases.

Their early **symptoms** are muscle pain and fever. In severe cases, the most prominent symptom is bleeding or hemorrhaging occuring at the orifices and in the internal organs.

The causative viruses may be the following:

Arenaviridae causing Argentine hemorrhagic fever, Bolivian hemorrhagic fever, Brazilian hemorrhagic fever, Lassa fever, Venezuelan hemorrhagic fever, Guanarito viral, Junin viral, Machupo viral and Sabia viral infections.

Arenaviridae are found all throughout South America, particularly in the Argentine pampas, Bolivia, Venezuela, and rural Brazil near Sao Paulo. *Arenaviridae* are found in West Africa (Lassa) as well. Chronic infections of small field rodents can most frequently infect rural residents and farmers usually in the fall. In Argentina, the illness occurs mostly among agricultural workers. In Bolivia, rodents can invade towns and cause epidemics. The West African illness, Lassa fever, can spread to humans if infected rodents are captured to consumpt, and from person-to-person.

Bunyaviridae: causing Crimean-Congo hemorrhagic fever, Hantavirus pulmonary syndrome, Hemorrhagic fever with renal syndrome, Rift Valley fever, Bayou viral, Black Creek Canal viral, Dobrava/Belgrade viral, Four Corners viral, Hantaan viral, Muleshoe viral, New York viral, Puumala viral, Seoul viral, Sin Nombre viral infections, and *Filoviridae:* causing Ebola, Ebola hemorrhagic fever and Marburg virus disease. These infections are experienced in Africa, the Middle East, the Balkan, southern Russia and western China. Filoviridae (Ebola and Marburg virus) are found in Africa and maybe in the Philippines, too. The vector is unknown, though in case of certain monkey infections aerosol transmission is suspected. It appears that outbreaks of the Ebola disease often follow uncommonly dry periods, when rainfall lasts for an all too long time reaching an unusually high level.

Flaviviridae: causing Dengue fever, Dengue hemorrhagic fever, Dengue shock syndrome, Kyasanur Forest disease, Omsk hemorrhagic fever and yellow fever.

Kyasanur Forest disease follows a tick bite in rural areas of its endemic zone, Karnataka, India. Monkey die-offs may accompany increased virus activity. Omsk HF was observed in western Siberia with unknown vectors and reservoir cycle involving ticks, voles, muskrats, and is possibly water-borne and mosquito transmitted as well.

19.1.1.1. Dengue Disease, Dengue Hemorrhagic Fever and Shock Syndrome

Dengue and dengue hemorrhagic fever (DHF), a generally self-limiting and rarely fatal illness can be caused by one of four closely related, but antigenically distinct virus serotypes of the Flavivirus genus. These serotypes do not provide a cross-protective immunity, so persons living in dengue-endemic areas can have four different dengue infections in their life [redacted]. Important risk factors of DHF include

the strain and serotype of the infecting virus, as well as the age, the immune state and the genetic predisposition of the patient.
One single infective mosquito (i.e. Aedes aegypti) can infect several persons with dengue virus within one day, being thus an effective endemic vektor. The incubation period of the illness lasts for 3 to 14 days, after which, the illness begin with an acute onset of fever accompanied by a lot of nonspecific signs and symptoms. During this febrile period lasting for 2-10 days, dengue viruses can be circulating in the peripheral blood. If the infected person is bitten during this febril days by other A. aegypti mosquitoes, those mosquitoes may become infected and transmit the viruses to other uninfected persons after an incubation period of 8 to 12 days. The disease affects usually older children and adults. The primary pathophysiologic abnormality seen in DHF and DSS means an acute increase in the vascular permeability, leading to the leakage of plasma into the extravascular space, causing hemoconcentration and a decreased blood pressure.
Its symptoms are characterized by fever, frontal headache, retro-orbital pain, nausea and vomiting, joint pain, weakness and rashes. Conjunctivitis, anorexia, altered taste sensations and constipation may also occur. Lymphadenopathy is common. Rashes can also develop, erupting both early and late. Before the onset of fever, facial flushing or erythematous mottling can develop, disappearing 1 to 2 days after the onset of the symptoms and can be followed by a second, scarlatiniform or maculopapular rash. This second rash usually begins on the trunk and then spreads to the face and extremities and its duration is 2 to 3 days. After the temperature of the patient becomes normal, scattered and confluent petechiae and purpura can be often experienced, as well as gum bleeding, epistaxis, menorrhagia and gastrointestinal hemorrhages. In case of adults the convalescent phase is associated with weakness and depression lasting for weeks. Neutropenia followed by lymphocytosis and a mild hepatitis are common alterations in case of dengue fever.
Lacking an early diagnosis and proper management, patients can develop a mild or severe **shock** caused by blood loss. Children if getting a profound shock, are often somnolent, exhibit petechiae on the face and have perioral cyanosis. An early diagnosis, aggressive fluid replacement therapy and careful nursing can decrease the fatality rates. The hemostatic changes involve three factors: vascular changes, thrombocytopenia and coagulation disorders, such as a prolonged partial thromboplastin time, a decreased fibrinogen level and increased levels of fibrinogen degradation products, suggesting a disseminated intravascular coagulation. Patients experiencing a second infection with a heterologous dengue virus serotype have a significantly higher risk of developing DHF and DSS. In case of a secondary infection, the virus can be in complex with antibody, making itself undetectable by most virus isolation techniques.
Diagnosis: by serologic tests, i.e. hemagglutination-inhibition (HI), complement fixation (CF), neutralization tests (NT), IgM capture enzyme-linked immunosorbent assay (MAC-ELISA), and by indirect IgG ELISA.
By dengue virus identification using IFA with serotype-specific monoclonal antibodies. By PCR, hybridization probes.
Vaccination: No effective, safe vaccine has been developed as yet.
Treatment: symptomatically and by infusions. There is no specific therapy concerning this virus.
RFR method: detects and may eliminate the virus! Its resonant frequencies are unknown as yet.

19.1.1.2. Ebola Hemorrhagic Fever

Ebola hemorrhagic fever (Ebola HF) is a severe, often-fatal (50-90%) disease sporadically affecting humans and nonhuman primates (monkeys, gorillas and chimpanzees). The disease is caused by infection with Ebola virus belonging to the Filoviridae family. There are four identified Ebola virus subtypes. Three of the four caused a disease among humans:

Ebola-Zaire, Ebola-Sudan, and Ebola-Ivory Coast. The fourth, Ebola-Reston caused a disease among nonhuman primates.
Its exact origin, location, and natural reservoir is unknown, though it is thought to be zoonotic. Ebola infection may also be spread by contact with personal materials originating from Ebola patients. This illness being life threatening, strict preventive measures (isolation, barrier nursing techniques etc.) must be taken regarding persons suspected to be infected and contact tracing and follow-up must be maid regarding those, who may have been exposed to Ebola virus. Communities affected by Ebola should be well informed as regards the strict safety measures which must be taken.
Its **Symptoms**: are characteristically a sudden onset of fever, intense weakness, muscle pain, headache, sore throat, conjunctivitis, massive hemorrhages often followed by vomiting, diarrhea, rashes, impaired kidneys and liver, and in some cases, internal and external bleeding. Leukopenia, thrombopenia and elevated liver enzymes are commonly experienced.
Diagnosis: by specific laboratory tests on blood, saliva and urine specimens, antibody testing, virus isolation in cell culture, done under maximum biological containment conditions.
Treatment: symptomatically and by ntensive supporting care, rehydration with solutions containing electrolytes, etc.
RFR method: detects and may eliminate the virus!
The resonant frequencies of Ebola virus are not known as yet.

19.1.1.3. Lassa Hemorrhagic Fever (LHF)

Lassa virus is the causative agent of Lassa hemorrhagic fever. This virus belongs to the *arenavirus* group, and can be transmitted to human beings by rodent reservoirs, i.e. by multimammate rats (Mastomys genus), by direct contact with infected tissues or indirectly, by eating food contaminated with excreta, possibly by inhaling aerosol originating from infected rodents and by body fluids from person-to-person. This virus is recognized in Guinea, Liberia, Sierra Leone and Nigeria, though these rodents carrying the virus, can be found all throughout West, Central, and East Africa. The illness develops fairly rapidly but slower then other hemorrhagic fevers, notably Congo hemorrhagic fever.
Symptoms: After an incubation period of 1-3 weeks, the patient complains of chills, fever and malaise, headache, myalgia and arthralgia, weakness, neck pain and sore throat. Early examination reveals fever and flushing of the face and the V area under the neck and is followed by pain in the joints and lower back, and a nonproductive cough. Retrosternal or epigastric pain, rigors, nausea, vomiting, anorexia, diarrhea and abdominal discomfort are also common. Exudative pharyngitis and conjunctival injections may also come to pass. Edema of the face and neck, conjunctival hemorrhages, oral ulcerations, maculopapular rashes, cough, mucosal bleeding, bleeding from the gums, central cyanosis, encephalopathy and shock characterize the most severe cases. Some patients experience adult respiratory distress syndrome. After the first week of illness patients begin to recover, but deteriorate if severly affected. Complications of the illness can be remaining deafness and alopecia.
Diagnosis: by tissue culturing and by complement fixation tests done after the fourteenth day of illness. By direct visualization using electron microscopy, by immunohistochemical techniques, IFA, ELISA, Immunoblot, RT-PCR and Real-Time RT-PCR tests.
Differential diagnosis: by distinguishing it from Yellow fever and other types of Haemorrhagic fevers.
Treatment: by administering ribavirin and according to its symptoms.
RFR method: can detect and eliminate the virus!
The resonant frequencies of the Lassa virus are unknown as yet.

19.1.1.4. Crimean-Congo Hemorrhagic Fever

Crimean-Congo Hemorrhagic Fever was first detected in the Crimea (Russia). At that time it was established by studies in human volunteers that the etiological agent was filterable and that this disease was associated with the bite of the larvae and adult ticks named Hyalomma marginatum. Congo Hemorrhagic Fever was first observed in Africa, its causative virus was found to be serologically indistinguishable from the Russian one, and was similar to those virus strains found in Central Asia and Bulgaria. The virus has been classified as being a Nairovirus of the genus Bunyavirus and is related to Hazara virus isolated from ticks in Pakistan, and to the Nairobi sheep disease virus.

This African virus was isolated from cattle, sheep, goats, hares, hedgehogs and from a number of ticks which parasites them, including Hyalomma, Amblyomma variegatum, Boophilus decoloratus and Rhipicephalus species.

The infection is usually transmitted to man by the bite of a tick carrying the virus and by contact with the infected patient's blood and the blood of infected animals or blood-contaminated objects.

Symptoms: Following an incubation period of 2-7 days the illness begins suddenly with fever, chills, severe muscle pain, headache, vomiting and pain in the epigastric and lumbar regions. A hemorrhagic state develops from the 3rd to the 5th day manifesting itself as petechial hemorrhages and purpura in the skin, and bleeding from the mucous membranes such as epistaxis, hemoptysis, hematemesis, melena and hematuria. At this stage the conjunctivae are injected, the face flushed, the tongue dry and often coated with dry blood. At the beginning the heart beat is slow, but the loss of blood then results in fast and feeble pulse, hypotonia and weak heart sounds, which are all clear signs of an impending shock and vascular collapse. Leukopenia and thrombopenia are commonly present. In case of recovering patients the temperature falls between the 10th to 20th day, the bleeding will stop, their convalescence will be prolonged for 4 weeks or longer. In fatal cases, massive hemorrhages and cardiac arrest will lead to death, usually on the 7-9th days after the onset of the illness.

Diagnosis: by the patient's history of a tick bite and symptomatically. By using a specific Congo virus antiserum in an immunofluorescent test.

Treatment: according to the symptoms as there is no specific therapy as yet.

RFR method: can detect and eliminate the virus!

Its resonant frequencies are not yet known.

19.1.1.5. Rift Valley Hemorrhagic Fever

Rift Valley fever (RVF) is an acute viral disease, affecting domestic animals (cattle, buffalo, sheep, goats and camels) as well as humans. RVF is associated with mosquito-borne epidemics in years with unusually heavy rainfall.

The disease is caused by the RVF virus, belonging to the genus Phlebovirus of the Bunyaviridae family and is generally found in regions of eastern and southern Africa affecting sheep and cattle, exists also in most countries of Sub-Saharan Africa, in Madagascar and even outside Africa in Saudi Arabia and Yemen. Mosquitoes (Aedes vexans, Ae. ochraceus, and Ae. dalzieli) as well as other mosquitoes and blood-sucking insects, f.i. sandflies (Phlebotomus duboscqui) as well as three other mosquito species (i.e. Ae. cumminsii, Ae. circumluteolus, and Ae. mcintoshi) are known to be vectors of this virus. In periods of dry weather, the virus lies dormantly in drought-resistant mosquito eggs all over the African continent. If sleeping outside, the chance of getting ill with RVF is significantly increased.

People can get RVF by being bitten by mosquitoes and other bloodsucking insects, and also if they are in contact with the blood or other body fluids of infected animals and by touching contaminated meat when preparing the food.

Symptoms: following an incubation period of 4 to 6 days, infected persons can either have no symptoms or develop a mild illness associated with fever and liver abnormalities. This illness can also progress to hemorrhagic fever leading to shock, hemorrhages, encephalitis and retinitis with permanent visual loss as well. At the onset of the illness patients experience fever, weakness, back pain, dizziness and an extreme loss of weight, photophobia, and will then recover within one week, though a small percent of the patients will get meningoencephalitis with fever, myalgia, headache, coma and seizures. In extreme cases, hemorrhage and necrosis of the liver, jaundice, hematemesis, melena and petechiae can develop, leading to vascular collapse, shock and even death.
Prophilaxis: by reducing the contact with mosquitoes and other bloodsucking insects by using mosquito repellents and bed nets. By avoiding exposure to blood or tissues of potencially infected animals.
Diagnosis: symptomatically and by laboratory tests. Specimens for virus isolation can be taken from liver, spleen, heparinized blood, serum and brain.
Treatment: symptomatically Interferon and other immune modulatory therapies, convalescent-phase plasma may help.
RFR method: can detect and eliminate the virus!
The resonant frequencies of this virus is not yet known.

19.1.1.6. Marburg Hemorrhagic Fever

Marburg hemorrhagic fever is a rare, severe illness, affecting people and non-human primates. Its causative agent is a zoonotic RNA virus belonging to the *Filovirus* family, and is in that way in relation with the four species of Ebola virus. Although an outbreak occurred in Serbia (Europe), the virus was transmitted by imported monkeys from Uganda. In an other case the patient living in Zimbabwe got ill in Johannesburg, South Africa and transmitted the virus to his traveling companion and a nurse in Marburg. There were outbreaks in Western Kenya and in Durba (DR of Congo).
Symptoms: begin after an incubation period of 5-10 days, with a suddenly developed fever, chills, headache and myalgia. Around the fifth day, maculopapular rashes most prominently on the trunk (chest, back and the stomach region) can occur. Nausea, vomiting, chest pain, sore throat, abdominal pain and diarrhea can also come about. Symptoms become increasingly severe including jaundice, inflammation of the pancreas, severe loss of weight, delirium, shock, liver failure, massive hemorrhagic and multi-organ dysfunctions. While recovering, which can last for a long time and be accompanied by orchitis, recurrent hepatitis, transverse myelitis, uvetis, inflammation of the spinal cord, eyes and the parotid glands. Its fatality rate lies between 23-25%.
Diagnosis: symptomatically and by testing using antigen-capture ELISA, IgM-capture ELISA, PCR and virus isolation as soon as possible.
Differential diagnosis: by distinguishing it from other infectious diseases, such as malaria and typhoid fever.
Treatment: by supportive hospital therapy, by balancing the patient's fluids and electrolytes, maintaining their oxygen status and blood pressure, by replacing blood and clotting factors and treating their eventually concomittant infections.
RFR method: detects and eliminates the virus!
I have had no opportunity to measure this kind of virus.

19.1.1.7. Omsk Hemorrhagic Fever

Omsk Hemorrhagic Fever (OHF) occurs in the western Siberian regions f.i. Omsk, Novosibirsk, Kurgán and Tjumen, affecting mostly muskrat trappers. This illness, caused by a genetically unique zoonotic virus transmitted by the bite of infected ticks, is a rare but severe type of hemorrhagic fevers. Its seasonal occurrence in every area mentioned is coincidental with the activity of its vectors (i.e. Dermacentor reticulatus, D. marginatus and

Ixodes persulcatus), although some data suggest a direct way of transmission from muskrat and virus contaminated water to humans as well.

Symptoms: The incubation period of this disease is usually 3-8 days, following which, suddenly developing fever, chills, headache, nausea, vomiting, pain in the lower and upper extremities, a severe prostration and papulovesicular rashes on the soft palate, cervical lymphadenopathies and conjunctival suffusions can usually come to pass. The central nervous system can also be affected, causing encephalitis. After one or two weeks there can develop in severe cases a typical haemorrhagic fever with gum bleedings, gastrointestinal hemorrhages, hematuria, sometimes cutaneous rashes, leukopenia, thrombocytopenia and lymphocytosis leading to shock, caused by plasma leakage.

Diagnosis: should be established as soon as possible by tests of immunohistochemistry, virus isolation and PCR of blood and tissue specimens, as well as symptomatically.

Differential diagnosis: by distinguishing it from other hemorrhagic fever diseases, and encephalitis of other origin.

Treatment: symptomatically, by giving supportive hospital therapy and an aggressive fluid replacement therapy. A new antiviral drug such as ribavirin can be of help.

RFR method: can detect and eliminate the virus!

The resonant frequencies of the Omsk hemorrhagic fever virus is not known as yet.

19.1.1.8. Argentinian and Bolivian Hemorrhagic Fever

These newly detected hemorrhagic diseases were experienced in Argentinia and Bolivia, caused by the serologically distinct Junin and Machupo viruses isolated from patients and rodents. People can acquire the virus through contact with objects and foodstuff, contaminated with infected rodent urine. The main reservoirs are wild murid rodents, i.e. Calomys laucha and Calomys musculinus, direct human-to-human transmission is also possible.

Symptoms: AHF and BHF usually involve the kidneys, and the cardiovascular system and cause hematological disorders as well. Their incubation period is 7 to 16 days, followed by gradually developing chills, fever, headache, malaise, myalgia, anorexia, nausea and vomiting. A prominent facial flushing and a painless enanthema of the pharynx can also occur. Following this, the disease get worse with dehydration, hypotension, oliguria, bradycardia and hemorrhagic manifestations, including bleeding from the gums, hematemesis, hematuria and melena. Progressive oliguria, the tremor of the tongue and the extremities may develop. Some patients develop agitation, delirium and stupor. From the seventh to tenth day progressive shock, hypothermia, gallop rhythm and gastrointestinal bleeding may occur. Usually pulmonary edema is the cause of death.

Diagnosis: by hematologic and laboratory test systems and symptomatically.

Differential diagnosis: by distinguishing them from other hemorrhagic fevers.

Treatment: symptomatically and by administering newly developed antiviral drugs. AHF and BHF have no specific therapy.

RFR method: can detect and eliminate the virus!

The resonant frequencies of the AHF and BHF viruses are not known as yet.

19.1.2. Bacterial Infections of the Blood

19.1.2.1. Bacteremia and Septic Shock

Bacteremia can be clinically transient, intermittent, or continuous. Many a transient bacteremia results from manipulations of infected or contaminated tissues, f.i. from instrumentation of the genitourinary tract, tonsillectomy, dental procedures and surgical incisions of furuncles or abscesses. In the vast majority of cases the sudden spreading of bacteria into the bloodstream does not produce any symptoms, or if, mostly only rigor and

fever for a short time, after which the bacteria will be promptly removed by the immune system. *Continuous bacteremia* occurs in the first days of typhoid fever, brucellosis and in case of intravascular infections such as endocarditis or endarteritis.
Septic shock is a condition caused by sepsis, in which case the blood pressure falls to life-threateningly low levels. Gram negative enteric bacteria such as *Enterobacteriaceae, Pseudomonas* and their related species are its causative agents, though a septic shock may be associated with Gram positive bacterial infections, f.i. caused by *pneumococci* and *other streptococci*, as well. Bacteremia and sepsis with gram-negative bacilli, f.i. the *Bacteroides species* occur less fulminating than in case of sepsis caused by aerobic Gram negative bacilli [redacted].
The shock syndrome is not due to bloodstream invasion with bacteria per se, but is related to the release of endotoxins, the lipopolysaccharide components of the bacterial cell walls, into the circulation. Endotoxin effects mostly the small blood vessels with sympathetic alpha receptor innervation. The toxin, causing an intense arteriolar and venous spasm, decreases the bloodflow, mostly that of the lungs, splanchnic area and the kidneys, causing permanent anoxia in these tissues and thus local acidosis, promoting the relaxation of the arteriolae, without effecting the venules. An endotoxic active substance, i.e. lipid A, can cause a variety of reactions, and profoundly affects the host's defense. It, first of all, activates the complement system, and leads to the decrease of the C3 complement, inducing thereby a chain-reaction causing vasodilatations, the increase of capillary permeability and local tissue damages. Endotoxin per se, can cause in addition fibrin thrombi in the capillaries, leading to fibrin-platelet aggregates, aggravating thus the anoxia of tissues, and the toxic tissue damages. Owing to the consumption of several clotting factors, a syndrome termed disseminated intravascular coagulation (DIC) can develop.
Symptoms of septic shock are tachycardia, tachypnea, hypotension, chills, fever, nausea, vomiting, diarrhea, pale extremities with peripheral cyanosis, mental shock syndrome, and oliguria, as well. Some patients are hypothermic, and in absence of fever the diagnosis is often false. Jaundice occuring occasionally may signify a biliary infection, intravascular hemolysis, or toxic hepatitis.
Diagnosis: symptomatically, by blood-pressure monitoring, urinanalysis, ECG, x-ray, blood-gas analysis and acid-alkali-balance examinations. The culturing of the primary septic focus may help to establish the right diagnosis. A negative result of blood culturing does not exclude the presence of a septic shock.
Treatment: by supporting the respiration, by vigorous fluid therapy (dextrose-saline, bicarbonate, plasma and dextran) In case of pulmonary edema by giving diuresis together with furosemide. By administering high doses of effective intravenous antibiotics, high doses of corticosteroids, by surgery in order to remove all necrotic tissues, such as the gangrenous tissues of the intestine, etc.
RFR method: should only be used in case of antibiotic resistances of the causative bacteria, after measuring the resonance frequencies in order to eliminate the bacteria together with the clinical treatment mentioned above.

19.1.2.2. Relapsing Fever Diseases

Relapsing fever is the common name of a group of vector borne diseases clinically characterized by cyclic periods of fever and apyrexia. These are caused by spirochetes of the Borrelia genus and have two epidemiological varieties, i.e. the louse-borne and the tick-borne ones. *Borrelia recurrentis* is the causative agent of the louse-borne relapsing fever. The tick-borne relapsing fever can be caused by many a strain of Borrelia f.i. *B. turicatae, B. parkeri, B. hermsii* etc. Rodents and other small animals are the reservoir of the tick vectors of this illness, which ticks belong to the genus Ornithodoros. Human beings can be infected by a bite contaminated with the saliva or coxal fluid of these tick vectors. Louse-borne relapsing fever is transmitted from person to person by scratching the

spirochetes from the body louse into their skin and mucosa. After inoculation borreliae reach the bloodstream, which spirochetemia causes fever and the illness begins. After several days, immobilizing and borrelicidal antibodies will appear, so that the spirochetes will be cleared out of the peripheral blood and the fever will cease. People exposed to the bacteria may not show symptoms usually for 6 days.
Symptoms initially include chills followed by high fever, rigor, anorexia, rapid heartbeat, severe headache, nausea, vomiting, muscle and joint pain, photophobia, cough, tachypnoe, visual impairment, leucopenia or leukocytosis, thrombocytopenia and often delirium as well. Jaundice caused by hepatocellular damages can also develop. Reddish rashes may appear on the trunk, arms and legs. Later on, fever, jaundice, enlarged liver and spleen, carditis and heart failure can be experienced mostly if caused by louse-borne infections. Stiffness of the neck and transient focal neurological signs can be seen. Borrelia can change their antigenity via their vegetative forms and plasmids, which survive the attack of the borrelicidal antibodies produced by the host's immun system. Following a latent period of approximately one week, during which the spirochetes are sequestered in the body, a new antigenic variant originating of them does arise. This reinvasion of the bloodstream will cause fever again and, eventually, a new group of specific antibodies will begin to act, leading to a second critic defervescence. The continued sequential production of new antigenic variants and specific antibodies results in a characteristic relapsing febrile process.
Diagnosis: symptomatically and by blood tests, bacterium culturing and by blood smear examination with phase-contrast or dark-light microscopy.
Differential diagnosis: by distinguishing it from malaria.
Treatment: by administering Doxycyclin.
RFR method: detects and may eliminate the pathologen bacteria!
Its resonant frequencies are: 301, 305, 309, 312-314, 317-319, 327-329, 341, 344, 352-354, 357, 372, 374-388, 404, 407-412, 420-422, 429, 442-453, 494, 510-511, 536, 544-545, 548, 556, 565 kHz

19.1.3. Human Anaplasmosis

Anaplasmosis is a zoonotic illness caused by *Anaplasma phagocytophilum* and *Ehrlichia equi* belonging to the order of Rickettsiales. Their tick vectors are Ixodes scapularis and Ixodes pacificus. *Anaplasma phagocytophilum* is the causative agent of tickborne fever or 'pasture disease' affecting cattle, goats, sheep and wild ruminants and causes also granulocytic ehrlichiosis among cattle, horses and cats, as well as Human Granulocytic anaplasmosis (HGA), (previously known as human granulocytic ehrlichiosis), transmitted to humans by the bite of deer ticks and western black-legged ticks [redacted] All these diseases are characterized by leukopenia and thrombopenia. Most cases occur in the months of spring and summer. The onset of anaplasmosis generally begins within a week after a tick bite.
Affected people are often misdiagnosed because of the non-specific nature of the **symptoms,** such as headache, fever, chills, myalgia and malaise, all of which can be confound with other infectious and non-infectious diseases. Rashes appear but seldom, confusion, hemorrhages and renal failure can also be experienced. Clinical manifestations of HGA can, depending on the patient's age and general health, range from mild to life-threatening state.
Diagnosis: symptomatically, by laboratory examinations with Giemsa staining of the smear, by low white blood cell and platelet counts, by PCR and immunostaining methods.
Treatment: by administering Doxycyclin for 10 to 14 days, but a longer treatment may be needed in case of a coinfection with Lyme disease.
RFR method: detects and may eliminate Anaplasma.

Its most frequent resonances are: 307-311, 335-337, 384-389, 414-425, 490-491, 534-535 kHz
Anaplasma marginale: 385-387, 415-425 kHz

19.1.4. Blood Infections Caused by Protozoa

19.1.4.1. Malaria

Four parasitic protozoa *(Plasmodium ovale, Plasmodium vivax, Plasmodium malariae, Plasmodium falciparum)* of genus Plasmodium can cause human malaria. The most dangerous of them causing severe illnesses and even death is the *P. falciparum* found mostly in the tropics and causes along with *P. vivax* 95% of malarial infections diagnosed worldwide. *P. vivax* causes usually less severe illnesses and is distributed more widely than P. falciparum. About 1.5-3.5 million deaths caused by malaria occur yearly. The majority of these fatal cases affects children younger than 5 years living in rural Sub-Saharan Africa.

All 4 species can be transmitted to human beings through the bite of an infected female mosquito of Anopheles species. These mosquitoes tend to bite only between dusk and dawn. Malaria can be transmitted also via blood transfusion or connatally from the mother to her fetus, although these forms of infection are rare.

Not immune persons living in or traveling to areas of Central and South America, Hispaniola, Sub-Saharan Africa, the Indian subcontinent, Southeast Asia, the Middle East and Oceania run the risk of getting infected with malaria. Infections occur when the vectors, containing plasmodia in their saliva are sucking blood. Entering into the circulating erythrocytes plasmodia feed on hemoglobin and other proteins within the erythrocytes. One brood of parasites becomes dominant and will be responsible for the synchronous occuring of the clinical symptoms of malaria. This protozoan brood replicates inside of the red blood cell, inducing cytolysis while they release toxic metabolic byproducts into the bloodstream; which cause flulike symptoms.

Symptoms: Occure in a cyclic pattern, beginning with shivering and chills lasting for 1-2 hours, which is followed by high fever. Finally, the patients experience excessive diaphoresis and their body temperature drops to normal.

P. falciparum can cause cerebral malaria, pulmonary edema, rapidly developing anemia, jaundice and can damage the kidneys and lead to coma and death. These protozoa metabolize hemoglobin and other red blood cell (RBC) proteins and produce toxic pigments (hemozoin). As they are getting their energy solely from glucose, they can cause hypoglycemia and lactic acidosis. The cytolysis of the erythrocytes, the suppressed hematopoiesis, and the increased clearance of erythrocytes by the spleen will lead to anemia, thrombopenia and hepatosplenomegaly. The RBCs infected with *P. falciparum*, use to form proteinaceous knobs which get bound to the endothelial cells. Due to their adherence these infected cells clump together within the blood vessels in many areas of the body, causing vascular obstructions and damages in the affected tissues.

P. vivax and P. ovale can persist in a dormant form in the liver of infected individuals to emerge at a later time. Thus, an infection caused by these species requires the killing of all dormant protozoan as well as the actively infecting species. If an infection caused by *P. vivax* remains untreated, its paroxysms usually last for 2-3 months with diminishing frequency and intensity, though relapses might occur after a few weeks or even after 5 years. Infections caused by *P. ovale* are usually similar to P. vivax infections, but they are less severe and often resolving even without any treatment.

People infected with *P. malariae* often remain symptomless for a longer period, though later on recrudescent symptoms can often be experienced, and can be associated with nephrotic syndromes, caused by the depositions of antibody-antigen complexes upon the glomeruli.

Diagnosis: symptomatically as well as by smears examined by using 200-300 times magnified oil-immersion and by fluorescent microscopy. One negative smear does not exclude the diagnosis of malaria, so that several more smears should be examined within a period of 36-hours. By PCR, etc.
Treatment: general hospital admission and intensive care unit admission guidelines are to be followed concerning patients with suspected or confirmed *P. falciparum* infections, as well as children, pregnant women and immunodeficient persons.
A reliable, semi-immune, adult patient with a *P. vivax, P. ovale, or P. malariae* infection may be treated on an outpatient basis and should have adequate follow-up care.
By administering chloroquine, or, in case of chloroquine resistancy quinidine, primaquine, mefloquine, etc.
RFR method: detects and can eliminate the protozoa.
The most frequent resonant frequencies of malaria are: 344-346, 364-367, 370-380, 385, 402-404, 410-425, 436-450, 476-482, 520-524, 560-564, 580-590 kHz
This list is not complete yet, as there exist other subspecies with different frequencies.

19.1.4.2. American Trypanosomiasis (Chagas disease)

American trypanosomiasis, also known as Chagas disease, affects millions of people throughout America. *Trypanosoma cruzi*, a protozoan hemoflagellate is the causative agent of this disease. Concerning people, the parasite can cause acute illnesses; though, the infection may prove to be symptomless as well. In some cases, many years after their initial infections, the affected persons experience clinical signs and symptoms caused by damages of the heart and the gastrointestinal tract. This disease is the leading cause of congestive heart failures in areas of Latin America, where it is endemic. During the acute phase of this illness, these protozoa destroy directly the host cells. The pathogenesis of the cardiac and gastrointestinal disorders typical for the chronic phase are not well characterized. The disease is limited to the Western Hemisphere, in temperate, subtropical, and tropical regions. It is prevailing in Mexico, Central America, and South America. Small mammals in the southern and southwestern United States can be the carriers of Trypanosoma cruzi. Infected Triatoma protracta, its insect vector have been found in California, Arizona and New Mexico as well [redacted] Human transmission occurs by contact with the defecated excreta of these insects, which defecate while ingesting human blood, excreting thereby the infective trypanosomes.
Symptoms: The incubation period for Chagas disease is about 1-2 weeks. After the bite of its special insect vector a nodular swelling or chagoma develops at the locus of the entry (mostly on the face or arms) becoming infiltrated with macrophages surrounded by lymphocytes, eosinophils and polymorphonuclear neutrophils, signaling an acute local inflammatory reaction. By lymphatic spreading the parasites will get into the regional lymph nodes, where histiocytes or other inflammatory cells ingest these parasites, which transform into amastigotes. In this amastigote form, protozoa can multiply in the cells of almost every organ and tissue. After their local multiplication, assuming the trypomastigote form, the pathogens invade the bloodstream, infecting all parts of the body. The reticuloendothelial system of the heart, the smooth muscles and the neural cells will be parasitized preferentially. These loci of cellular destruction are accompanied by a marked inflammatory reaction of the host characterized by the local accumulation of polymorphonuclear leukocytes, lymphocytes and plasma cells.
In its acute phase, the heart is the main target organ. The severity of this acute infection can be highly variable, ranging from being asymptomatic to getting severe tissue destructions. In all these cases, the protozoa are forming pseudocysts in the affected cells, each containing hundreds to thousands of amastigotes. Persons, recovering from this acute illness will carry these intracellular parasites. This phase can persist for years, or even for the rest of the patient's life. The myocardium reveals focal myonecrosis, interstitial fibrosis

and lymphocytic infiltration. In this early stage severe myocarditis or meningoencephalitis can lead to the patient's death.
During the **chronic phase** of the illness, the ganglion cells get gradually destroyed. Aneurysm, existing at the apex of the left ventricle is pathognomonic of chronic chagasic cardiopathy. Mural thrombosis can often occur mostly in the presence of atrial fibrillations. Embolization to the brain, lungs, spleen and kidneys can also come about. Disturbance in swallowing, regurgitation, hiccups and abdominal pain are its most common manifestations. Due to the damaged autonomic nervous system there can be abnormalities of secretion, absorption and motility present, affecting mostly the sigmoid colon, causing constipation, extreme dilatation and hypertrophy. Other neurologic symptoms of the central, peripheral and autonomic nervous systems do but rarely occur. Symptoms can include cerebellar disturbances, convulsions and polyneuritis.
These clinical symptoms are mostly experienced among infants, and children younger than 10 years, the younger the patient, the more severe the clinical manifestations.
Intrauterine infection can cause spontaneous abortion or premature delivery. In its acute stage, connatal Chagas disease resembles its acquired disease, including hepatosplenomegaly, jaundice, anemia, fever, edema, and meningoencephalitis with convulsions, hypotonia, hyporeflexia and tremors. Cardiac involvement is rare. Dysphagia can cause aspiration leading even to death occuring within the first few weeks of life. Those surviving, have severe neurologic sequelae with mental deficiency or behavioral and learning disabilities.
Diagnosis in the first 6-12 weeks, by examining motile parasites in blood smears with Giemsa staining or direct wet-mount preparations and by examination of the buffy coat. In case of late infections, by parasites culturing, by serologic tests for anti-T. Cruzi IgG and IgA classes (f.i. by complement fixation, indirect hemagglutination and indirect immunofluorescence, ELISA), by PCR and by xenodiagnosis, etc.
Treatment: symptomatically.
RFR method: detects and eliminates the Trypanosoma! But should only be used together with the antibiotic treatment.
The most frequent resonant frequencies of Trypanosoma cruzi are: 365-375, 442-451, 457-470 kHz

19.1.5. Blood Flukes

Schistosomiasis (bilharziasis) is a group of diseases caused by the three closely related species of digenetic trematodes belonging to the *Schistosomatidae* family, such as *S. mansoni, S. haematobium,* and *S. japonicum.* These blood flukes inhabit the circulatory system of humans and animals living in tropical and subtropical countries. The organs and tissues most frequently affected are the colon, the urinary bladder, liver, lungs, and the central nervous system [see Chapter 9.1.1]
Their resonant frequencies are: 325-330, 336-341, 350-357, 385, 428, 433-445, 473-478, 504-505 kHz

19.1.5.1. Schistosomiasis

Schistosomiasis is ,the second most prevalent tropical disease in the world. If an acute schistosomiasis is not suspected clinically and treated appropriately, it can result in severe illness or even death. The pathophysiology of schistosomiasis is associated with the immune response to the schistosome eggs. The clinical manifestations depend on the species of parasite, intensity of the worm burden, and on the immunity of the person to the parasite. Persons being at risk include those who live or travel in areas where schistosomiasis is endemic as well as those who come into contact with fresh water containing the appropriate type of snail intermediate host. The main forms of human schistosomiasis are caused by 5 species of flatworms belonging to the genus Schistosoma,

within the class trematodes. These 5 species are as follows: *S. haematobium, S. mansoni, S. japonicum, S. intercalatum* and *S. mekongi.* As they live in the vascular system of humans and other vertebrates, these worms are also named blood flukes [redacted]
S haematobium lives in the venous plexus near the urinary bladder and ureters, *S mansoni* lives in the inferior mesenteric vein, and *S japonicum* lives in the superior mesenteric vein of the large and small intestines.
The pathophysiology of this infection correlates with the life cycle of the parasite:
Cercariae: produce allergic dermatitis at the locus of its skin penetration causing thereby senzitization and pruritic papular rashes.
Schistosomula: being transported via blood and lymphatics to the right side of the heart and lungs, can cause cough, fever and eosinophilia.
Adult female worms, after mating in the veins, lay eggs 4-6 weeks following the cercarial penetration. Living in the veins for approximately 3-8 years they lays eggs throughout their whole life.
Eggs: cause Katayama fever and schistosomiasis.
Katayama fever: develops 4-6 weeks after being infected with *S. japonicum* but also with *S. mansoni* at the time of the initial egg release. This fever comes due to the high worm and egg antigen stimuli resulting in immune complex formation and leading to a serum-sickness-like illness. Fever, lethargy and myalgia are the most common symptoms. Less common ones include cough, headache, anorexia and rashes, mimicing acute viral, bacterial, or malarial illnesses.
Schistosomiasis is caused by immunological reactions to Schistosoma eggs trapped in the tissues. Antigens released from the eggs stimulate a granulomatous reaction caused by participating T-cells, macrophages and eosinophils. Symptoms and signs depend on the number and location of the eggs trapped in the tissues. This granulomatous inflammation is initially reversible, but later on collagen deposition and fibrosis will develop in the damaged organ.
Intestinal schistosomiasis caused by *S. mansoni* exists in about 52 countries, including the Caribbean countries (i.e. Saint Lucia, Antigua, Montserrat, Martinique, Guadeloupe, the Dominican Republic, Puerto Rico), the eastern Mediterranean countries, South American countries (i.e. Brazil, Venezuela, Surinam), and in most of the African countries. Other Schistosoma species that can cause intestinal symptoms and diseases include *S. intercalatum, S. japonicum, and S. mekongi. S. intercalatum* is found in 10 countries within the rain forests of central Africa. *S. japonicum* is endemic in 4 countries in the western Pacific region (i.e. China, Philippines, Indonesia and Thailand). *S. mekongi* infections prevail in the Mekong River area of Southeast Asia (i.e. Kampuchea, Laos, Thailand). This intestinal illness leads to portal hypertension and causes gastrointestinal hemorrhages.
Urinary schistosomiasis caused by *S haematobium* can be found in 54 countries in Africa and the eastern Mediterranean. This urinary tract illness can lead to renal failure due to obstructive uropathy, pyelonephritis, and later on to bladder carcinoma
Hepatic schistosomiasis: can cause dyspepsia, flatulence, and, due to splenomegaly, pain in the left hypochondrium, anemia and cor pulmonale, which letter may cause weakness, and shortness of breath. In its later stages, abdominal distention, lower limb edema, hematemesis and melena can occur.
CNS schistosomiasis can cause focal and generalized seizures; headache; and myeloradiculopathy with lower limb and back pain, bladder dysfunction, paresthesia and lower limb weakness.
Diagnosis and differential diagnosis: by stool or urine analysis in order to identify and specify the eggs, by urine analysis and culturing, by blood chemistries, CBC, serology, CT, etc.

Treatment: The first step to take is to cure the disease or at least to minimize its morbidity by administering Praziquantel and Oxamniquine (against S. mansoni). The second step to take is to control the transmission of the infection.
RFR method: detects and eliminates the pathogen!
The most frequent resonant frequencies are: 316-325, 334-337, 352-355, 433-435, 441-444, 471-475 kHz

19.2. Anemia Caused by Infections

Anemia may develop as the result of blood loss, the excessive destruction and inadequate production of red blood cells, or caused by various combinations of these processes. Anemia should never be thought of as a diagnosis in itself, but rather as a manifestation of an underlying disease process. The pathogenesis of the anemia may be caused by many a factor it being but a symptom of many infectious diseases.

19.2.1. Bartonellosis

In case of Bartonellosis large amounts of *Bartonella bacteria* enter the bloodstream, adhere to the erythrocytes and invade the endothelial cells of the capillaries and the lymphocytes. Bartonellosis (Carrion's disease) is an infection caused by *Bartonella bacilliformis*. There occur two well defined clinical forms, i.e. an acute febrile anemia of rapid onset and high mortality, named Oroya fever, and a benign eruptive form with chronic cutaneous lesions, termed verruga peruana. Both may proved to be mild, their asymptomatic cases constitute the greatest epidemiological hazard. This gram-negative bacillus is small, motile, aerobic, pleomorphic and reddish violet by Giemsa-stain. The manifestation of the disease reflects the immune state of the host.
Oroya fever does mostly develop concerning immunosuppressed people [redacted] In case of this illness the life span of the red blood cells is greatly shortend, the presence of the bacteria on the surface of the red blood cells causes their phagocytosis and thus anemia and their destruction in the liver and spleen. Early in the course of the infection also a defective erythropoietic response accentuates this anemia. The initial **symptoms** are a sudden onset of high fever, pain in the bones, joints and muscles. Extreme pallor, weakness and anemia are characteristic.
Verruga peruana, the other clinical form of the disease is characterized by angiomatous skin nodules and can follow the anemic form, though it may even occur in case of patients with no previous symptoms.
Diagnosis: by blood culturing and blood smears with Giemsa stain.
Treatment: by administering Doxycyclin, Chloramphenicol, etc.
The resonant frequencies of Bartonella bacteria are: 308-310, 324, 330-335, 364-372, 374-375, 379-381, 386-390, 402, 478, 480-492, 495, 548, 558-566 kHz

19.2.2. Mycoplasmal Eperythrozoon Anemia

Eperythrozoon anemia is caused by an infection with *Eperythrozoon coccoides, E. ovis, E. suis, E. parvum, E. wenyonii.* This disease is a typical zoonosis, the bacteria parasitize erythrocytes. These haemotrop mollicutes (named also haemoplasmas) belong recently to the Mycoplasma genus. Affected animals can be rats, guinea-pigs, squirrels, hares, rabbits, goats, sheep and pigs. This disease can but seldom spread from animal to human. The blood smear shows reddish violet Eperythrozoon species on the margin of the red blood cells done with Giemsa-staining. The life span of these red blood cells will be greatly shortened and anemia will develop. The **symptoms** include: a low grade fever, anemia and fatigue. Asymptomatic or convalescent carriers of these bacteria may be reservoirs of infection. The manifestation of this disease are thought to reflect the immune state of the

patient. Splenectomy and other immunosuppressed states favour the development of the disease.
Diagnosis: by blood smear with Giemsa-stain.
Treatment: by administering Doxycyclin.
RFR method: detects and may eliminate the pathogen!
The most frequent resonances found in case of this kind of anemia are: 364-370, 375-390, 478, 480-486, 548, 558-566 kHz
This frequency list is not complete, as there are still other variants of bacteria not measured as yet.

19.3. Hemolytic Anemia Syndrome

Hemolysis means the abnormal breakdown of the red blood cells in the blood vessels, or outside of the vessels anywhere in the body. Anemia caused by hemolysis is named hemolytic anemia, which can be acquired or inherited, its severity ranging from relatively harmless to life-threatening. This haemolytic anemia has many a possible cause, its treatment depends on the cause and nature of the hemolysis.
Autoimmune Hemolytic Anemia (AIHA) is a hematologic illness, characterized by the presence of high concentrations of circulating autoantibodies directed against the person's own red blood cells. Autoimmune hemolytic anemia can be primary, or secondary; and can be subclassified according to the type of the autoantibodies playing a role in its pathology.
In case of **primary autoimmune hemolytic anemia**, there is no underlying systemic disease which would explain the presence of autoantibodies, whereas **secondary autoimmune hemolytic anemia** is caused by systemic diseases.
The autoantibodies of AIHA may belong to the IgG, IgM, or, rarely, to IgA immuneglobulin families; and may be warm reacting or cold reacting. AIHA syndromes associated with cold-reacting autoantibodies include **cold agglutinin disease** and **Paroxysmal Cold Hemoglobinuria**. In the latter case the symptoms are caused by IgG type autoantibodies. AIHA can be associated also with warm antibodies, with systemic lupus erythematosus (SLE), and is a characteristic component of Evans' syndrome, a rare autoimmune illness characterized by antiplatelet and hemolytic antibodies. *Mycoplasma and HTLV* play an important role in the pathogenesis of AIHA.
Cold agglutinin disease, a clinical form of AIHA is characterized by autoantibodies binding red blood cells only at a low temperature. These cold-reacting autoantibodies are mostly of IgM type, which getting bound to the erythrocyte membranes cause a premature erythrocyte destruction, i.e. hemolysis.
Cold agglutinin associated antigens can be i antigens, I antigens, Pr antigens, and rarely sialylated polysaccharides. The specificity of cold agglutinins of anti-I and anti-i are strikingly similar to one another concerning the structure of the antigen-binding site. The I antigen is not activated before birth. Anti-i autoantibodies predominantly agglutinate the RBCs of newborns, while anti-I autoantibodies predominantly agglutinate the RBCs of adults.
In case of primary cold agglutinin diseases, the target is the I antigen. In case of secondary cold agglutinin diseases, the target may be the I or i antigen. Less common target antigens of RBC include Pr, Gd, Fl, Vo, Li, Sa, Lud, M, N, Me, Om, D, Sdx, and P. Cold agglutinins attach to the RBCs in the cooler peripheral circulation and will be dissociated from the RBCs as the blood returns to the warmer central circulation.
The hemolysis occurs due to complement fixation. The complement fixation and that of the autoantibody occurs within the intravascular space if the blood temperature drops below the thermal maximum of the antibody. This can occur if the antibody has a high thermal maximum or if the patient is exposed to a colder temperature. The fixation of the complement components to the RBC membranes can result in extravascular or intravascular hemolysis.

Extravascular hemolysis occurs when the complement activation and fixation to the RBC membrane is insufficient to trigger the activation of the membrane attack complex of the complement system. C3b and C4b present on the surface of RBCs interact with the receptors on the phagocytes of the lungs, liver and spleen, in consequence the RBC will be phagocytized. The liver is the predominant locus of hemolysis.
Intravascular hemolysis occurs if the complement fixation to the red blood cell membranes is of a sufficient amount to activate the membrane attack complex, causing thus the lysis of the RBCs, hemoglobinemia, hemoglobinuria and hemosiderinuria.
The primary, i.e. idiopathic form of this Cold agglutinin disease is usually caused by monoclonal cold-reacting antibodies, the illness is chronic and affects genetically predisposed people of more than 50 years.
The secondary cold agglutinin disease may be associated either with monoclonal or with polyclonal cold-reacting autoantibodies, is predominantly caused by infections and lymphoproliferative disorders and can be associated with systemic autoimmune diseases as well. These cold reacting antibodies are usually of IgM type, occasionally of IgG, and rarely of IgA type. The polyclonal antibodies have heavy chains and light chains as well, the monoclonal ones have mostly only one single type of light chain.
Different combined chronic infections can cause secondary cold agglutinin diseases with *monoclonal antibodies,* affecting mostly adults. Secondary monoclonal cold agglutinin diseases affecting children are induced by different infections and are associated with B-cell lymphoproliferative diseases, such as acute B-lymphoblastic leukemia, other B-cell neoplasms, Waldenström macroglobulinemia, lymphoma, chronic lymphoid leukemia, myeloma and can be associated even with nonhematologic neoplasms.
Secondary *polyclonal* cold agglutinin diseases are usually acute, transient, postinfectious, affect mostly children and young adults and are caused by Mycoplasma, such as *Mycoplasma pneumoniae, M. genitalium, M. fermentans and M. penetrans*, viral infections, caused f.i. by *EBV, HTLV, CMV, mumps, varicella, rubella, adenovirus, HIV, influenza, HCV* and bacterial infections, f.i. *Legionnaire disease, syphilis, listeriosis*, and can be caused by *Escherichia coli* and *corynebacterium*, parasitic infections such as *malaria* and *trypanosomiasis*.
An inherited hemolytic anemia can be associated

with genetic alterations of the RBC membrane including Hereditary spherocytosis, Hereditary elliptocytosis,

with genetic disfunctioning metabolism of the RBC (i.e. enzyme defects) including Glucose-6-phosphate dehydrogenase deficiency (G6PD or favism), Pyruvate kinase deficiency,

with genetic alterations of the hemoglobin, including Sickle cell anemia and Thalassaemia

An other form of acquired AIHA is the drug induced one.
A high dose of penicillin can induce immune mediated hemolysis via the hapten mechanism, in which antibodies are targeted against the complex of penicillin-RBC. The complement system will be activated by the attached antibody leading to the decomposition of red blood cells by the spleen.
Symptoms: In case of AIHA the clinical findings reflect the presence of anemia, hemolysis and RBC agglutination, as well as the presence of a co-existing disease. Anemia causes pallor, in more severe cases dyspnoe, tachycadia, fatigue, signs of a congestive heart failure and even shock.
Hemolysis causes jaundice, splenomegaly, sometimes fever and the cyanosis of the skin. Acrocyanosis of the fingertips, feet, earlobes and the nose can often be experienced. Fever, lymphadenopathy and rashes may be caused also by an associated illness. Exposure to cold may significantly worsen the anemia.

Laboratory findings: Mean corpuscular volume (MCV) is elevated due to reticulocytosis and agglutination of the RBCs. Smear usually shows spherocytosis, polychromasia and RBC agglutinations. Transient cold agglutinin disease is characterized by a moderately elevated cold agglutinin titer (1: 1,000-20,000) and by polyclonal cold agglutinins.
Anti-I autoantibodies can be experienced in case of mycoplasma infections, while anti-i in case of EBV infections. The autoantibodies appear 2-3 weeks after the onset of the symptoms and disappear within 2-3 months. Serology of mycoplasma, EBV, HTLV, or CMV, and of other infectious agents may be positive, depending on the associated causative illness.
Treatment: Viral infections, such as EBV, CMV and mumps, are usually self-limiting. Mycoplasma infections, systemic autoimmune diseases and lymphoproliferative diseases has to be treated. Prednisone therapy is seldom effective. Patients having a cold agglutinin disease, characterized by a low cold agglutinin titer and high thermal amplitude, may respond to prednisone therapy. Plasmapheresis can transiently reduce the autoantibody level. Rituximab (anti-CD20 monoclonal antibody) depletes B-lymphocytes and, interferes thereby with the production of cold agglutinin.
RFR method: detects and may eliminate the infectious agents. If they are already eliminated, the immune-process can be suppressed by administering corticosteroids. The elimination of the infectious causative agent of hemolytic anemia and immune-autoimmune anemia is of adventage if using RFR method.
The most frequently found resonances are those of the
Mycoplasma: 307-308, 321-324, 337-344, 342-350, 440-451, 491-495 kHz
EBV: 372-383, 518-519 kHz
CMV: 408-410, 530-536 kHz
HTLV-1 and HTLV-2: 311-314, 320-324, 330-331, 370-376, 406, 432-435, 493-504 kHz
Escherichia coli: 354-356, 392-393 kHz
Corynebacterium: 316-318, 340-344, 348, 356, 372, 383-389, 396-399, 402, 409-410, 460, 476, 492, 505, 576-578 kHz

19.4. Anemia Caused by Antiphospholipid Syndrome

Antiphospholipid syndrome (APS) is characterized by a wide variety of hemocytopenic and vaso-occlusive manifestations and is associated with antibodies directed against the negatively charged phospholipids. The features of APS include hemolytic anemia, thrombocytopenia, venous and arterial occlusions, livedo reticularis, pulmonary manifestations, recurrent fetal loss, neurological manifestations such as stroke, transverse myelitis, Guillain-Barré syndrome; and a positive Coombs test, anticardiolipin antibodies, or lupus anticoagulant activity. Primary antiphospholipid syndrome is caused by a *M. penetrans* infection, APS and *M. penetrans* infections can cause hemolytic anemia, and the antigens of M. penetrans adsorbed in the tissues of the host can become superantigens provoking thus autoimmune processes. See also Chapter 12.1.9.

19.5. Plummer-Vinson Syndrome

Plummer-Vinson syndrome (named also **Paterson-Brown-Kelly syndrome** or **sideropenic dysphagia)** is characterized by the triad of dysphagia, glossitis and iron-deficient hypochromic anemia. A crescentic fold indenting the anterior part of the cricopharingeal area, mucosal atrophy and inflammation of the hypopharynx are frequent signs of this disorder. Patients suffering from Plummer-Vinson syndrome often complain of a burning sensation in the tongue and the oral mucosa, while the atrophy of the lingual papillae results in a smooth, shiny red tongue at its dorsum. This syndrome is also associated with koilonychia, glossitis, cheilitis, angular stomatitis and splenomegaly. The cause of this syndrome is a combined infection with *Mycoplasma fermentans* (autoimmune

factor), *Epstein-Barr Virus*, *Cytomegalovirus* while also genetic factors and nutritional deficiencies play an important role.
Scandinavian women are at a higher risk than men, particularly in their middle age. Among these patients the **risk** of developing esophageal **squamous cell carcinoma** is increased (if in an immunocompromised state caused by *mycoplasma* and *HPV* infection); so that this syndrome is considered to be a premalignant state.
Plummer-Vinson Syndrome is often associated with autoimmune conditions, such as rheumatoid arthritis, pernicious anemia, celiac disease and thyroiditis. The basic causative factor of this autoimmune process is mycoplasmal infection.
Diagnosis: by serial contrasted gastrointestinal radiography, by upper gastrointestinal endoscopy, by blood tests showing a hypochromic microcytic anemia that is consistent with iron-deficiency anemia. By the biopsy of the involved mucosa.
Treatment: by iron replacement against anemia, by mechanical dilation.
RFR method: can detect and eliminate the virus and the mycoplasma
The most frequent resonances of Mycoplasma fermentans are: 442-461, 493-495 kHz.
The most frequent resonances of Cytomagalovirus are: 408-410, 530-536 kHz
The most frequent resonances of Epstein-Barr Virus are: 372-383, 518-519 kHz
The most frequent resonances of HPV are: 402-410, 540-545 kHz
Prevention of developing squamous cell carcinoma: by detecting and eliminating HPV viruses.
The primer goals of the therapy are to reduce morbidity and to prevent complications and the development of squamous cell carcinoma. Patients generally respond well to the treatment. Iron supplementation can usually heal anemia and tongue.

19.6. Infectious Hemochromatosis Syndrome

Hemochromatosis syndrome (HS) is an iron-storage disease, characterized pathologically by an excessive deposition of iron in the parenchymal tissues, and, clinically, by hepatomegaly. Liver insufficiency, skin pigmentation, diabetes, arthropathy, cardiac disease and hypogonadism can also come about in case of this illness. The involved organs are the liver, heart, pancreas, the pituitary gland, joints and skin. This hemochromatosis syndrome can be caused by certain combined infections or/ and can be a common, in an autosomal recessive way inherited genetic disorder, affecting whites and causing liver damages.
In case of healthy adults, the losses are balanced by the absorption of sufficient (1-2 mg) dietary iron.
In case of **hereditary hemochromatosis**, a dysregulation of the intestinal iron absorption is present, wherein the iron absorption does continuously occur even in case of a substantial elevation of body iron stores. **Infectious Hemochromatosis Syndrome** is caused by certain pathogen group of Coxsackie and/or ECHO viruses (f.i. by those causing heart damages, i.e. *Coxsackie viruses A 1-9* and *16, B1-5, ECHO viruses 1-19, 22, 25, 30* and by that causing diabetes; *Coxsackie virus B4*) in this case these infections causing hemolytic damage of the erythrocytes are associated with a co-infection by *Mycoplasma fermentans* causing arthropathy. The hereditary form of hemochromatosis is a genetic disorder inherited in an autosomal recessive way. Its specific gene, which is mutated in the vast majority of patients suffering hereditary hemochromatosis, is named HFE, is linked to the HLA-A region on the chromosome 6. This mutation causes an enhanced accumulation of iron in the peripheral tissues.
Hepcidin, a human antimicrobial peptide synthesized in the liver, plays an important role in the down-regulation of iron release by enterocytes and macrophages. In case of adult-onset HFE-related diseases its amount is small. Excessive iron amount is hazardous, as it produces free radical formations which can lead to the rapid formation of damaging reactive oxygen metabolites and thus to cell injury and fibrosis.

Symptoms: include usually fatigue, impotence and arthralgia, while later on, skin bronzing or hyperpigmentation caused by iron and melanin deposits, can be experienced. Patients may be asymptomatic, too and can be only diagnosed if their elevated serum iron levels are notable. The classic triad of cirrhosis, diabetes mellitus and skin pigmentation is characteristic to the late phase of this disease. The progressive iron accumulation in the pancreas can often cause diabetes mellitus. The damages developing due to the progressive iron deposition in the liver parenchyma may progress to hepatocellular carcinoma, which is the most common cause of death among patients with hereditary or infectious hemochromatosis. Cardiomyopathy affecting mostly younger patients may be present with a congestive heart failure and arrhythmias. Hypogonadism, experienced in case of this illness, occuring due to the iron deposition of the pituitary gland is the most common endocrine abnormality causing decreased libido and impotence in men. Amenorrhea can occur among affected women but is less frequent than the hypogonadism of men. Arthropathy develops due to iron accumulation in the joint tissues.
Hemachromatosis can be idiopathic, erythropoetic (f.i. in case of porhyria cutanea tarda), alcohol-toxic and infectious.
Diagnosis: by quantitative examinations of serum iron levels, liver biopsy, by transferritin saturation test. By ultrasound, CT, MRI.
Treatment: symptomatically, and, in severe cases, by administering Deferoxamine mesylate (Desferal)
RFR method: detects and may eliminate the pathogen microorganisms in case of infectious hemochromatosis.
The most frequent resonances are: 307-308, 317-319, 322, 357,.361-365, 369-376, 397-399, 404-411, 419-426, 431-433, 442-444, 447-452, 457, 472, 480-489, 493-495, 512-519, 526, 532, 552, 559-560, 577 kHz

19.7. Polycythemia

In case of polycythemia there is a net increase in the total number of blood cells, primarily in the red blood cells of the body. The overproduction of red blood cells may be caused by a primary process working in the bone marrow (i.e. in case of so-called myeloproliferative syndromes), or may be a reaction provoked by chronically low oxygen levels, or, rarely, by a malignancy.
Primary polycythemia (named often polycythemia vera (PV), polycythemia rubra vera (PRV), or erythremia), occurs if excessive amounts of red blood cells are produced as a result of an abnormality of the bone marrow. This disorder is often accompanied by the production of an extraordinarily great amount of white blood cells and platelets. Polycythemia vera belongs to the myeloproliferative diseases.
Secondary polycythemia can be associated with renal and liver tumors, with specific infections of the erythroid formation system, with hypoxia caused by heart and lung diseases, and with endocrine abnormalities, including pheochromocytoma and adrenal adenoma causing the Cushing's syndrome. Athletes and bodybuilders using anabolic steroids, or erythropoietin (EPO) may develop a secondary polycythemia.
Polycythemia vera is a stem cell disorder, characterized as a panhyperplastic, malignant, and neoplastic bone marrow disorder [redacted].

20. EYE DISORDERS ASSOCIATED WITH INFECTIONS

A variety of viruses, bacteria, fungi and parasite can cause infections, inflammations and tissue damages concerning the eyes. Disorders of the eyes can develop associated with systemic illnesses including various types of allergy, psoriasis, etc.

20.1. Inflammations of the Eyes in General

The different eye tissues can get infected by viruses, bacteria, fungi and protozoa, and get inflamed also owing to allergic reactions as well as to autoimmune processes.
The most frequent viral pathogens attacking the eyes are *Adenoviruses, Cytomegaloviruses and Herpes Simplex Virus-1 and 2.*
The most frequent bacterial pathogens of the eyes are the species of *Staphylococcus, Streptococcus, Pneumococcus, Gonococcus, Pseudomonas, Chlamydia and Borrelia B. sensu lato.* Inflammations of the eyes can as a secondary process be experienced in case of *Syphilis* and *Tuberculosis.*
Toxoplasma and *Acanthamoeba* are the most important protozoa, while *Onchocerca* and *Toxocara* are worms causing inflammation of the eyes.
Histoplasma and other fungi, such as *Candida and Aspergillus* may cause slowly growing ulcers of the eyes. *Rhinosporidiosis* can produce small, tumor-like masses usually in the nose and nasopharynx, but sometimes in the eyes as well. An endosporulating fungus seen in the tissues can not be cultured on laboratory media.
The eyes can get inflamed as a consequence of allergic reactions to molds, animal danders, pollens, etc., can get irritated by the wind, dust, smoke and other types of chemicals of air pollution, too.
The pathogens mentioned above cause inflammations in different parts of the eyes.
Conjunctivitis is the inflammation of the conjunctiva.
It can develop due to infections (caused by bacteria, viruses, parasites and fungi);and due to irritation (such as the lack of tear fluid), caused by an early type allergy, toxins (irritants like smoke, dust, etc.) and by systemic diseases (f.i. Stevens-Johnson syndrome).
Trachoma is a chronic infection of the conjunctiva caused by different serotypes of *Chlamydia trachomatis.* This infection is still one of the most important causes of visual loss in some countries.
Inclusion conjunctivitis is an acute inflammation caused by *Chlamydia* affecting sexually active adults and their newborn offsprings.
Epidemic keratoconjunctivitis typically causing epidemics; appears most often due to *adenovirus types 8 and 19* infections, though minor forms of epidemic keratoconjunctivitis can be caused by other adenovirus types as well. This infection can produce a follicular conjunctivitis from moderate to severe, healing spontaneously within 10 to 18 days. Its incubation lasts for 4 to 14 days. Severe cases can be complicated with conjunctival membrane formation, conjunctival scarring and with subconjunctival hemorrhages. Mild systemic symptoms, such as low-grade fever, headache and malaise may occur. The preauricular lymph nodes at the side of the affected eye are usually enlarged.
An adenoviral spreading might occur in case of manipulating f.i. foreign-body removal, and examination made by an ophthalmologist. The virus can be transmitted by the fingers, instruments and solutions as well.
Vernal keratoconjunctivitis, a recurrent inflammation of the conjunctiva affecting mostly both eyes, can damage the surface of the cornea as well. The latter illness is caused typically by allergy, mostly occuring in spring and summer. Allergic conditions can worsen due to bowel parasitic ascariasis.

Keratoconjunctivitis sicca is a chronic dryness of the eyes leading to the dehydration of the conjunctiva and cornea. This disorder can also be associated with rheumatoid arthritis, systemic lupus erythematosus and Sjögren's Syndrome. Whether it accompanies these diseases or occurs alone, the dry eyes are most commonly experenced among middle aged women.

Purulent conjunctivitis can be a manifestation of *Pseudomonas* infection mostly affecting premature infants.

Episcleritis is the inflammation of a layer of connective tissue between the sclera and the conjunctiva. This inflammation usually affects only a small part of the eyeball causing a yellow, slightly raised alteration.

Scleritis is a deep, painful inflammation of the sclera showing a purplish discoloration. Scleritis accompanies usually rheumatoid arthritis and other immune-mediated disorders. In severe cases this inflammation may lead to the perforation of the eyeball and the loss of the eye.

A corneal ulcer develops generally due to infections.

A corneal *herpes simplex infection* resembles at the beginning a mild bacterial infection, the eyes are slightly painful, watery, red and sensitive to light. The vision can be hazy due to corneal swelling. In case of this viral infection minor changes of the cornea develop if not treated. Rarely, the virus deeply penetrates the cornea, destroying its surface. The infection may recur, further damaging the surface of the cornea. *Herpes Simplex Virus* can cause neovascularization of the cornea, impairing the vision.

Pseudomonas and *Proteus* infections cause more severe forms of corneal ulceration, usually following a traumatic abrasion and may lead to panophthalmitis and the destruction of the globe. Contact lenses or lens cleaning fluids contaminated with Pseudomonas bacteria may be sources of eye infections. Repeated irritative inflammations and infections of the cornea may result in ulceration, scarring, and even in the loss of vision.

Herpes Zoster Virus growing in the nerves may spread to the eye. This unilateral infection causes pain, redness, and the swelling of the eyelid. The infected cornea can become swollen, severely damaged and scarred. **Peripheral ulcerative keratitis** is an inflammation and ulceration of the cornea often occuring among people with autoimmune connective tissue diseases, f.i. rheumatoid arthritis. Similar alteration can be experienced in case of infections caused by *Chlamydia* and *Borrelia B. sensu lato* species, which alterations occur probably due to autoimmune processes generated by these pathogens.

Uveitis is a common inflammation of the iris, the ciliary body and/or the choroidea.

Glaucoma is a disorder in case of which the pressure in the eyeball increases, damaging the optic nerve leading thus to visual loss. If the outflow channels are open, the disorder is named open angle glaucoma. If the root of the iris blocks the channels, the disorder is a closed angle glaucoma. In case of an infection also the inflammatory cells may block the channels.

Endophthalmitis is an inflammation involving all inner layers of the eye.

Retinitis pigmentosa (RP) is a rare, inherited disorder characterized by a progressive loss of photoreceptors and of the function of the retinal epithelium. In this case a diffuse, usually bilateral and symmetrical retinal dystrophy will occur. Although rods as well as cones of the retina are involved, the damage of the rod system is prominent. Retinitis pigmentosa can exist as an isolated disorder inherited in an autosomal dominant, autosomal recessive, or X-linked manner, or, may occur in association with certain systemic disorders. The gradual degeneration of the light sensing rod cells lead to poor vision in the dark. Its first symptoms often begin already in childhood. Over time, a progressive loss of the peripheral vision also comes about. In the later stages of this disease, the affected person has solely a so-named tunnel vision.

Symptoms of **macular degeneration** are f.i. a certain kind of blurred vision, in case of which the normally straight lines look crooked and the patient solely sees a dark or empty

space in the center of the vision. The exact cause of this destructive eye disease is not yet known, but it is a fact that a new vessel formation in the macular area, nutritional deficiencies and genetical anomalies can all lead to macular degeneration.

Optic neuritis is an acute or subacute inflammatory demyelinating process affecting the optic nerve. Its most frequent type among adults is **retrobulbar neuritis**, in case of which the optic disc is normal. This inflammative disorder is frequently associated with Multiple Sclerosis. Several other conditions can also trigger retrobulbar neuritis such as viral infections, immunizations, syphilis, borreliosis, temporal arteritis, poisoning by chemicals, tumors spread to the optic nerve, allergic reactions, autoimmune reactions, etc.

The infection of the eye with parasitic larva is sporadic, in which case the parasitic larva is spread via the bloodstream.

Acute granulomatous uveitis may prove to be the initial manifestation of *sarcoidosis*. This ocular disease may cause severe visual impairments and blindness with corneal and lenticular opacities and secondary glaucoma.

Lacrimal gland enlargement, conjunctival infiltrations and keratoconjunctivitis sicca may all be present in case of **Sjögren's Syndrome** as well as. exophthalmos, retinal lesions with vasculitis, producing papillitis and periphlebitis.

Recurrent ocular inflammations are common features of **Behçet's Syndrome**, manifested by anterior and posterior uveitis, conjunctivitis, optic neuritis and retinal arteriitis. Though cataract, glaucoma, and even blindness may develop, the former mentioned inflammations can prove to be reversible.

(In regard to other ocular infections, see the special Chapters.)

Treatment: by administering effective antibiotics, or acyclovir, trifloruridine, vidarabine, idoxuridine and in case of severe, destructive inflammations corticosteroids as well.

RFR method: detects and eliminates the pathogen microorganisms!

The most frequent resonances are those of the

Cytomegalovirus: 305, 349, 406-412, 512, 530-536, 548 kHz

Adenovirus: 370-387, 390-392, 393, 394-400, 402, 523, 534 kHz

Epstein-Barr Virus: 318, 342-347, 352, 374-382, 403, 422, 424, 451, 476-480, 491, 516-519, 528, 560 kHz

Herpes Simplex Virus-1: 290-294, 302, 336, 344-346, 347-350, 357-358, 370-377, 381, 397-400, 413, 431-433, 438, 449, 463, 478-482 kHz

Herpes Simplex Virus-2: 301, 337-340, 352-365, 374-376, 380, 396, 403, 413, 425, 434, 450-459, 474, 493-496, 500, 540-552, 568 kHz

Herpes Zoster Virus: 310, 337-339, 348, 372, 383, 396-398, 409-410, 416-421, 460-461, 467, 477, 540-541, 555 kHz

other Herpes frequencies: 366-377, 383-385, 413, 434, 540-554 kHz

Staphylococcus group are: 294, 308, 323-329, 345, 347, 367, 376-381, 388, 401-402, 421, 434, 448-453, 458, 463, 465, 482, 484, 486, 490-491, 504, 511, 517, 542, 552, 556-557, 563-568, 576 kHz

Streptococcus: 313-321, 340, 349-351, 356-362, 374-376, 381-387, 391, 397-403, 408, 410, 418, 432-437, 442-444, 447-454, 464 472-478, 508, 516, 542 kHz

Pseudomonas group: 323-325, 330-336, 351, 356-361, 374, 380, 396, 401, 414, 438, 446-447, 496, 512, 579 kHz

Proteus group: 320-329, 333-339, 345-352, 408-416, 426, 516, 522-529, 535 kHz

Pneumococcus: 320, 360-372, 397, 410, 433, 472-480 kHz

Gonococcus: 307, 330-337, 364 kHz

Borrelia Burgdorferi sensu lato: 372-388 kHz

Borrelia B. s. l. have different plasmids, the most well-known are: 302, 432 kHz

Their other frequencies can be: 341, 374, 389, 399, 401-409, 429, 442, 452-453, 508-511, 548, 556, 565 kHz

Chlamydia: 316-319, 374-386, 429, 440-444, 480-482, 566 kHz

Toxoplasma: 394-396, 436, 444 kHz
Histoplasma: 298-306, 315, 375-384, 434 kHz
Mycoplasma: 307-308, 321-324, 337-350, 442-451, 493-495 kHz
Glaucoma associated virus: 372, 403, 409, 450, 467-468 kHz
Moraxella: 296-297, 394-398, 514-518 kHz

20.2. Cytomegaloviral Retinitis

CMV infects the majority of the adult population. In case of immunocompetent hosts, its infection is generally asymptomatic or limited to a mononucleosis like syndrome. Similar to many other herpes viruses, CMV remains latently in the host and may reactivate, if the immunity of its host gets compromised.
In case of immunocompromised persons, the primary infection or the reactivation of the latent virus can cause opportunistic infections in several organs of the host, including the skin (rashes), the lungs (pneumonitis); the gastrointestinal tract (colitis, esophagitis); the peripheral nerves (radiculopathy and myelopathy); the brain (meningoencephalitis) and the eye (retinitis, optic neuritis).
CMV infection of the eyes causes most commonly a necrotizing retinitis. An untreated CMV retinitis leads to visual loss and blindness. CMV retinitis is the leading cause of visual loss among AIDS patients.
This severe visual loss primarily occurs from the direct spreading of retinitis into the posterior pole, affecting the central vision, or from the retinal detachment secondary to multiple retinal breaks in the peripheral, necrotic retina. The symptoms vary according to the locus of retinal involvement. Active CMV retinitis is usually found in conjunction with immunosuppressed states caused f.i. by AIDS, leukemia, or chemotherapy.
Fine, stellate keratitic precipitates observed on the corneal endothelium are characteristic of CMV. Uveitis may be present in the anterior chamber. CMV retinitis can lead to necrosis, scarring and atrophy. The infected cells lyse, leaving a necrotic area of full-thickness and releasing virus particles that infect the adjacent retinal cells. The most frequent co-infections are caused by *HTLV 1-6, Herpes Simplex Viruses, Mycoplasma fermentans* or other mycoplasmas.
Diagnosis: by PCR (and based on HIV test and CD4 count), and symptomatically.
Treatment: CMV treatment with gancyclovir implant intravitreal, and other antiviral drugs in order to prevent viral replications. By vitreoretinal surgery, laser photocoagulation, etc., combined with the treating of HIV and AIDS.
RFR method: detects and may eliminate both virus infections (i.e. CMV and HIV).
The most frequent resonances of CMV are: 305-306, 322-326, 349, 408-410, 417, 453, 518-519, 530-536, 548-550, 566 kHz
The most frequent resonances of HIV are: 290, 312-318, 350, 360-367, 372, 397-399, 401-409, 416, 425-428, 450-455, 460-464, 458-559, 476 kHz
Regarding other HTLVs see their special Chapter.
The most frequent resonances of Mycoplasma fermentans are: 442-451 kHz
Regarding Herpes Simplex Viruses, see their special Chapter.
There may be many different frequencies present in this disease. RFR method is not an alternative therapy concerning CMV retinitis. Combine the conventional therapy with this method!

20.3. Stye

Stye is usually a *Staphylococcus aureus* infection of one or more glands of the edge of the eyelid or of the skin under it. Styes occur sometimes simultaneously with blepharitis or resulting from it. Pain, redness, tenderness and swelling can be experienced on this very

small area, usually at the edge of the eyelid. This stye rarely ruptures by itself, one may have to open it in order to drain the developed pus.
Diagnosis: symptomatically
Treatment: by topical treatment or by administering effective antibiotics.
RFR method: detects and eliminates the bacteria, which are usually staphylococci.
The most frequent resonances of Staphylococcus aureus are: 329, 372, 376-384, 402, 434, 482, 491, 537, 557, 567 kHz
As to the other frequencies of Staphylococci see its special Chapter.

20.4. Mysterious Conjunctivitis, Optic Neuritis and Visual Loss

Borreliosis (named also Lyme disease) is caused by the spirochetes *Borrelia Burgdorferi sensu lato* [redacted] Involvement of the eye is not uncommon in case of borreliosis. The eyes can be affected in many a different way. In the early stage of the disease, persons may have conjunctivitis. In this case, the eyes are red, itchy, causing uncomfortable feeling and there can be a whitish discharge, too. Unlike many forms of conjunctivitis, this type is not contagious. If not recognized, borreliosis can get chronic. Lyme borreliosis can cause a variety of ocular manifestations, which develop mainly in the late stage of the disease. Severe periodic ocular pain and photophobia are characteristic symptoms of Lyme borreliosis. Blurred vision and eye pain, caused by keratitis and iritis can also be experienced. Uveitis associated with photophobia, macular edema, retinal vasculitis, neuroretinitis and choroiditis with retinal detachment may develop. Neuro-ophthalmic manifestations such as optic neuropathy and other cranial neuropathies, f.i. abducens-nerve palsy and paresis of the seventh nerve are often come about. Unilateral blindness caused by panophthalmitis has been reported as well. Ocular symptoms can include floaters (i.e. spots in front of the eyes). Inflammation of the optic nerve, i.e. optic neuritis can also occur, leading to visual loss. Disturbed vision can result from the inflammation of the brain as well.
Persons who develop Bell's palsy may be unable to blink or close their eyes. This could lead to the drying-out of the cornea and result in an infection or cause a hole in the cornea, which can endanger the vision if not promptly treated. Chronic inflammation may cause glaucoma-like symptoms and blindness. *Adenovirus and Herpes virus* frequently coinfect the eyes in these cases of borreliosis.
Diagnosis and treatment of Borreliosis, see Chapter 6.20.3. In case of more severe ocular damages local and systemic corticosteroid treatments, as well as special ophthalmologic managements may prove to be essential.
RFR method: in case of Borreliosis (see Chapter 6.20.3.), Adenoviruses (see Chapter 5.2.3.), Herpes viruses (see Chapter 5.2.4.)

20.5. Toxoplasma Infections of the Eyes

Toxoplasma gondii is an obligate, intracellular parasite responsible for zoonotic infections of mammals and men. More over, it is the most common cause of intraocular inflammations all over the world. Cats are its definitive hosts becoming infected by eating contaminated raw meat, wild birds and mice. The 3 life forms of the protozoan, present solely in cats, are tachyzoites, bradyzoites and sporozoites. Mammals and humans can only be infected by tachyzoites and bradyzoites. The prevalence of serum antibodies against toxoplasmosis varies throughout the world and depends on eating habits, hygiene, and climate. Toxoplasmosis appears to be more prevalent in hot, humid climates. Toxoplasmosis may be connatal or otherwise acquired.

If a pregnant susceptible woman acquires primary toxoplasmosis, there can occur a transplacental transmission of the parasite to the fetus. Human toxoplasmosis can be aquired by ingesting tissue cysts in contaminated raw or undercooked beef, lamb and pork, by ingesting oocysts in the soil, milk, water and vegetables, by inhaling oocysts, by contaminated blood transfusion, organ transplants and by an accidental inoculation acquired in the laboratory. [redacted]
An acute infection is characterized by tachyzoites that invade and proliferate in almost any type of mammalian cells, except those of nonnucleated erythrocytes. Tachyzoites entering the host's cells reproduce via endodyogeny, by which process, 2 daughter tachyzoites are formed within the parent parasite, which when the the daughter cells are released will be destroyed by the host's cell. When this protozoon reaches the eye via the bloodstream, depending on the host's immune state a clinical, or subclinical focus of infection will develop in the retina. In case of the immune response of the host, the tachyzoites convert into bradyzoites, i.e. into cyst forms. These cysts are extremely resistant to the host's defense, so that a chronic latent infection does begin.
Toxoplasmosis is typically classified as follows:
Connatal toxoplasmosis, acquired toxoplasmosis, toxoplasmosis in case of immunocompromised hosts and ocular toxoplasmosis.
Connatal toxoplasmosis can develop if a susceptible woman becomes infected during her pregnancy and thus T gondii will be transmitted on a transplacental way to the the fetus. A chronic maternal infection will not associate with connatal disease.
This connatal disease is characterized by the classic clinical triad of chorioretinitis, cerebral calcifications and convulsions. Acutely severe and fatal infections, occuring but seldom, can cause hydrocephaly, microcephaly, organomegaly, jaundice, rashes, fever and psychomotor retardation. Antitoxoplasma immunoglobulin M (IgM) antibodies are present in 75% of infants with connatal toxoplasmosis.
If patients with active retinochoroiditis have preexisting chorioretinal scars, their ocular illness may be caused by a secondarily reactivated connatal infection.
Acquired toxoplasmosis can occur on all way of transmission mentioned above but one, i.e. the connatal way. These acquired infections are is usually subclinical and asymptomatic. In 10-20% of the cases patients develop a flulike illness characterized by fever, lymphadenopathy, malaise, myalgias, maculopapular skin rashes sparing the palms and soles. In case of immunocompetent persons the disease is benign and self-limited. Elderly patients acquiring toxoplasmosis are at a greater risk of developing a severe retinochoroiditis, due to their weakend cellular immune functions caused by their aging.
Ocular toxoplasmosis is more commonly associated with acquired infections than previously believed. In case of a subclinical infection no funduscopic changes can be observed, despite of the fact that the cyst is present in the seemingly normal retina. Whenever the host's immune defense weakens for some reason, the cyst wall may rupture, releasing protozoa into the retina, so that the inflammatory process will start again. A clinically active lesion will heal with a chorioretinal scar. The cyst often remains inactive within or adjacent to the scar.
Ocular symptoms include blurred vision, floaty points, pain, red eyes, metamorphopsia, and photophobia. The hallmark of the disease is a necrotizing retinochoroiditis, which may be primary or recurrent. In case of primary ocular toxoplasmosis, a unilateral focus of necrotizing retinitis is present. The area of necrosis usually involves the inner layers of the retina showing a whitish fluffy lesion surrounded by retinal edema.
The retina is the primary site for the multiplying parasites, the choroid and the sclera can be the loci of a contiguous inflammation. If the optic nerve will be involved by toxoplasmosis, its typical manifestations are optic neuritis or papillitis associated with edema (named Jensen disease).

Toxoplasmosis of immunocompromised hosts. The host's immune function plays an important role in the pathogenicity of toxoplasmosis. Immunocompromised patients often develop life-threatening pneumonitis; myocarditis; encephalitis; and an atypical, sight-threatening, severe multifocal, bilateral and progressing necrotizing retinochoroiditis.
Posterior vitreous detachment is usually to experience, patients may develop precipitates of inflammatory cells named vitreous precipitates. Thick vitreous strands and membranes can require vitrectomy. The hypersensitive reaction against the toxoplasma antigens can cause retinal vasculitis and granulomatous or nongranulomatous anterior uveitis as well. These ocular lesions often heal with punched-out scars. In case of reactivation of live cysts an active necrotizing retinitis will newly develop adjacent to the old scars.
The diagnosis: symptomatically and by the clinical appearance of the fundus lesion, by serology using ELISA, indirect fluorescent antibody, indirect hemagglutination, complement fixation tests, etc.
Treatment: by administering effective antibiotics (f.i. Clindamycin, Azithromycin, Sulfadiazine etc.) corticosteroids use to decrease inflammatory processes, is to be given together with antitoxoplasmatic drugs. By cryotherapy, surgery, etc.
RFR method: detects and may eliminate the Toxoplasma.
Its most frequent resonant frequencies are: 393-400, 430-464 kHz

20.6. Dry Eye Syndrome of Infectious Origin

Chronic Keratoconjunctivitis Sicca (CKCS) is either an inflammatory or a non inflammatory ocular disease. CKCS is often associated with connective tissue and other immune-autoimmune diseases, co-existing most commonly with rheumatoid arthritis), SLE, Graves' disease, Banti's syndrome, Guillain-Barré syndrome, systemic sclerosis and Lyme disease, which infection can trigger various autoimmune diseases. CKCS can or not be associated with Sjögren's Syndrome (SS). Primary Sjögren's Syndrome is an autoimmune disease, affecting mostly the lacrimal and the salivatory glands and is thereby characterized by aqueous tear deficiency (ATD) and xerostomia. This tear deficiency usually includes decreased tear production, excessive tear evaporation and an abnormality in the mucin or lipid components of the tear film. Xerostomia means dry mouth and lips. In case of a Secondary Sjögren's Syndrome these eye and mouth symptoms are associated with a connective tissue disease f.i. with reumatoid arthritis, systemic lupus erythematodes and progressive systemic sclerosis. It being an autoimmune disease serum antibodies are often present, which are mostly ANA and SS antibodies, of which SSB/La is the most specific for this illness. SSA/RO is usually associated with other autoimmune disorders as well. Patients with Sjögren's Syndrome are mostly women. There exists a genetic predisposition concerning Sjögren's Syndrome associated with CKCS showing a high prevalence of HLA-B8 haplotype. This predisposition can lead to chronic inflammatory processes, caused by the production of autoantibodies, including antinuclear antibodies (ANA), rheumatoid factors, SS-specific antibodies, and by inflammatory cytokines and focal infiltrations of $CD4^+$T cells of the lacrimal and salivary glands, causing glandular degeneration and induction of apoptosis in them. Various proinflammatory cytokines may cause cellular destructions, including IL-1, IL-6, IL-8, TGF-beta, TNF-alpha and RANTES, being all enhanced present in patients with KCS. The amount of tear proteins, such as lysozyme, lactoferrin, lipocalin and phospholipase A2, is decreased in case of CKCS. Sjögren's Syndrome may develop into Hodgkin's lymphome.
The symptoms of CKCS include: ocular irritation, dry sensation, burning, itching, pain, foreign body sensation, photophobia and blurred vision. Chronic infectious corneal ulceration in case of dry eye syndrome can cause blindness. Other complications include punctate epithelial defects, corneal neovascularization and corneal scarring.
CKCS may develop in case of many an immune-autoimmune disease such as systemic lupus erythematosus, polymyositis and dermatomyositis, rheumatoid arthritis, systemic

lupus erythematosus, progressive systemic sclerosis (scleroderma), autoimmune primary biliary cirrhosis, autoimmune interstitial nephritis, polyarthritis nodosa, Hashimoto thyroiditis, Wegener granulomatosis and Borreliosis. Secondary coinfections caused by herpes viruses and/or adenoviruses and by bacteria, f.i. staphylococci and/or streptococci can often come about. In case of CKCS the natural defense mechanism of the eyes becomes weak.

Diagnosis: symptomatically, by conjunctival cytology, by serology for circulating autoantibodies, including ANA and SS antibodies. By using rose bengal and fluorescein staining to evaluate epitheliopathy. By Schirmer test to measure the aqueous tear production, by the biopsy of a lacrimal gland or minor salivary gland, etc.

Treatment: in case of secondary CKCS, by treating the causative disease. The aim of the therapy is to reduce morbidity, and to prevent complications. By applying artificial tears, opthalmic drops, etc.

RFR method: detects the pathogen triggering the autoimmune inflammatous disease, (see the special Chapter) and eliminates all causative pathogens, thus stopping the autoimmune process together with the administering of corticosteroids. According to our RFR measures there are many a kind of viruses and bacteria to be found in case of this disease. The most frequently observed pathogens being HTLV, EBV, CMV, Mycoplasmas and Borrelia B.s.l. species, while the secondary infections are caused mostly by HSV and adenovirus species. When the associated autoimmune/immune disease is healed, CKCS can heal by itself.

20.7. Macular Degeneration

Macular degeneration is a condition in which the macula, the central and the most vital area of the retina, is degenerating. Macular degeneration is the leading cause of vision loss and blindness among people over 65, in the case of which it is named age-related macular degeneration (ARMD). The disease affects the macula, the light-sensitive part of the retina, responsible for the sharp, direct vision needed for reading and driving. Especially the central vision will get gradually damaged.

Macular degeneration has a dry (atrophic) and a wet (exudative) form. Its dry form is much more common than the wet one. The wet form of this disease often leads to a more serious vision loss.

Macular degeneration is more common among white people and females. This illness can occur as the side effect of drugs, can be related to aging and to familiar predisposition (owing to a hereditary viral infection).

Stargardt's disease, known also as **Juvenile Macular Degeneration**, is an autosomal recessive retinal illness characterized by a juvenile-onset macular dystrophy, alterations of the peripheral retina and a subretinal deposition of a certain lipofuscin-like material.

Age-related macular degeneration (ARMD) is the most common cause of irreversible vision loss in the developed world. It is associated with the presence of drusen, early in the disease without any visual loss, it often progresses to retinal atrophy and to central retinal degeneration associated with the loss of the central vision.

Dry ARMD is, in most cases, an inherited autosomal dominant disease which appears to be in connection with nutrition and environmental factors such as by infections with *Adenoviruses* and/or with *Herpes Simplex Virus-1*. This atrophic, dry form can result from the aging and thinning of the macular tissues, depositing pigment in the macula. The disease is characterized by the degeneration of the retina and the choroid in the posterior pole owing either to a retinal atrophy or to a retinal pigment epithelium detachment. The atrophy is generally preceded by (or, in some cases coincident with) the presence of yellow extracellular deposits adjacent to the basal surface of the retinal pigment epithelium called drusen. The retinal pigment epithelium degeneration is accompanied by the loss of the overlying photoreceptors and the underlying choroidal perfusion. If persons of the above said age suffer from loss of visual acuity, visual field, or other visual functions, the

condition is often classified as AMD. Drusen are composed of vitronectin (a multifunctional plasma and extracellular matrix protein), lipids, inflammation-related proteins, amyloid associated proteins, as well as of other poorly characterized substances. While drusen was thought to be the result of accumulated waste material from subretinal tissues, nowadays it is suggested that the accumulation is owing to of an inflammation of viral-origin in the subretinal space.

Wet ARMD: The neovascular or exudative ARMD, i.e. the "wet" form of an advanced ARMD, causes the loss of vision due to an abnormal blood vessel growth (choroidal neovascularization) in the choriocapillaris, through the Bruch's membrane, leading ultimately to blood and protein leakage below the macula. If left untreated, the bleeding, the inflammatory capillary leakage and the scarring can eventually cause an irreversible damage to the photoreceptors and a rapid vision loss.

The Cystoid macular edema (CME) is a painless condition characterized by the swelling or thickening of the central retina (macula) and is usually associated with blurred or distorted vision. CME is a relatively common phenomenon frequently associated with certain various conditions, such as cataract surgery, age-related macular degeneration (ARMD), uveitis, eye injury, diabetes mellitus, retinal vein occlusion, and drug toxicity.

Macular degeneration can develop also as a side effect of toxic drugs, f.i. chloroquine, phenothiazine, occuring due to hapten formation.

The **etiology** of the ARMD is multicausal, including coexisting viral infections affecting immune competent persons. The most frequent causative viruses are *Herpes viruses* and *Adenoviruses* often found together. The viral inflammation can cause the release of prostaglandins, the breakdown of the blood-retinal barrier, vasodilation, increased capillary permeability owing to the damage of tight endothelial junctions in the retinal capillaries, and a decreased removal of fluid by the retinal pigment epithelium. Enzyme phospholipase can be inhibited by steroids and thus can block the formation of prostaglandins and their effects. The cyclooxygenase pathway can specifically be inhibited by aspirin and nonsteroidal anti-inflammatory drugs.

Systemic diseases, such as diabetes mellitus, chronic renal failure, hypertension, etc can promote the development of macular degeneration. The risk to develop ARMD is determined by alterations in 3 specific genes, i.e. the CFH gene on chromosome 1, the BF (complement factor B) gene and the *C2* (complement component 2) gene on chromosome 6 and the LOC gene on chromosome 10.

The symptoms of MD can develop slowly or suddenly. The most frequent symptoms are central scotomas, trouble discerning colors, loss in contrast sensitivity, specifically to distinguish dark ones from dark ones and light ones from light ones.

If a person sees straight lines look wavy and if his vision is fuzzy and if there are shadowy areas in his central vision, he might experience the early signs of ARMD.

Persons with nonexudative macular degeneration may be asymptomatic or may notice a gradual loss of their central vision, whereas those with exudative macular degeneration often notice a rapid onset of painless vision loss.

Diagnosis: by ophthalmoscopy, by fluorescein angiography. By Amsler Grid eye test. The early established diagnosis is very important in order to prevent irreversible processes and blindness.

Treatment: There is no specific cure for macular degeneration, though a therapy may be able to delay its progression and even improve the vision. Researchers suggest that antioxidant vitamins, such as beta-carotene (vitamin A) and vitamin C and E can protect the macula from damages. The consuming of omega-3 fatty acids has been correlated with a reduced progression of early ARMD, and the eating of low glycemic index foods has been correlated with a reduced progression of advanced ARMD.

The use of LASER photocoagulation in order to destroy or to seal new blood vessels and to prevent leakage can be of help to wet AMD patients.

RFR method: detects and eliminates the viruses!
The most frequent resonances are: 290-294, 392-394, 467-476 kHz
As to the resonant frequencies of Herpes Simplex Viruses, see [redacted]
As to the resonant frequencies of Adenoviruses, see [redacted]
There can often be found other resonant frequencies, too, f.i. those of Mycoplasma species.
The elimination of *Adenoviruses and HSV-1* is able to prevent the progression of macular degeneration.

20.8. Retinal detachment

Retinal detachment occurs when subretinal fluid accumulates in the potential space between the neurosensory retina and the underlying retinal pigment epithelium (RPE). Depending on the mechanism of the subretinal fluid accumulation, retinal detachments have traditionally been classified into rhegmatogenous, tractional, and exudative kinds.
The retinal detachment refers to the separation of the inner layers of the retina from the underlying retinal pigment epithelium (choroid). Choroid is a vascular membrane containing large branched pigment cells sandwiched between the retina and sclera. The separation of the sensory retina from the underlying RPE can be caused f.i. by fluid seeping from the vitreous cavity (**rhegmatogenous retinal detachment**), by exudation of certain materials from the retinal vessels into the subretinal space (**exudative, serous, or secondary retinal detachment**, f.i. in case of hypertension, diabetic microangiopathy, central retinal venous occlusion, vasculitis and papilledema) and by some injury, inflammation or neovascularization, when a fibrovascular tissue is pulling the sensory retina away from the retinal pigment epithelium (**tractional retinal detachment**):
Certain familial disorders, such as Stickler syndrome, Marfan syndrome, Homocystinuria and Ehlers-Danlos syndrome, are associated with rhegmatogenous retinal detachment.
Certain **inflammatory processes and infections,** f.i. in case of acute retinal necrosis syndrome, *Adenoviral* and/or *Herpes* viral, and/or *CMV retinitis* among *HTLV* or *Mycoplasma* infected patients, or in case of ocular *toxoplasmosis* can lead to retinal detachment, too.
Symptoms: Photopsia refers to the perception of a flashing light by the patient. It probably occurs due to the mechanical stimulation of vitreoretinal traction on the retina. It may be induced by eye movements and appears to be more noticeable in dim illumination. Patients often see a black curtain (visual field defect) once the subretinal fluid extends posterior to the equator. Dark shadows observed in the patient's visual field are opacities floating in the vitreous cavity. A ring-shaped floater is the so-called Weiss ring, a remnant of the hyaloid that was attached to the edges of the optic disc.
Cobwebs caused by the condensation of the collagen fibers can also be seen by the patients. Detectable small spots usually indicate an acute bleeding caused by the rupture of a retinal vessel during an acute posterior vitreous detachment. If the macula becomes detached (in case of extension of the subretinal fluid into the macula), the patient experiences the dropping of his visual acuity.
Diagnosis: symptomatically, by indirect ophthalmoscopy and by direct funduscopy.
Treatment: f.i. by cryopexy, laser photocoagulation, scleral buckle surgery, vitrectomy and ignipuncture
Prevention: by treating the basic disease, by eliminating the infective viruses.
RFR method: can detect and eliminate the causative pathogenic agents.
The most frequent resonances of Adenoviruses are: 371-387, 393-394 kHz
The most frequent resonances of Herpes Simplex Virus are: 290-294 kHz
The most frequent resonances of CMV are: 408-411, 530-536 kHz
The most frequent resonances of Toxoplasma are: 393-396, 436-445 kHz
The most frequent resonances of Mycoplasma fermentans: 442-451 kHz
The most frequent resonances of Herpes Zoster Virus are: 416-420, 544-545 kHz

The frequencies of other pathogens not identified as yet, are: 370-374, 403, 468 kHz

20.9. Glaucoma

Glaucoma is an ocular disorder, in case of which the pressure in the eyeball increases, damaging the optic nerve and leading to loss of vision.

The anterior and posterior chambers of the eye are filled with a thin fluid, the aqueous humor. This fluid is produced in the posterior chamber, passes through the pupil into the anterior chamber, and then leaves the eye through the outflow channels. If an inflammatory process blocks these channels, the intraocular pressure will be increased.

Although a raised intraocular pressure is a significant risk factor for the development of glaucoma, there is no set threshold for intraocular pressure to cause glaucoma. Some person may get a nerve damage at a relatively low pressure, while others may have high eye pressure for years, nevertheless they do never get damaged. Untreated glaucoma leads to the permanent damage of the optic nerve and a resultant visual field loss, which can lead to blindness. However, glaucoma is a nonspecific term used for several different ocular diseases that ultimately result in an increased intraocular pressure as well in a decreased visual acuity. The ocular blood flow is also supposed to be involved in the pathogenesis of glaucoma. Fluctuations of the blood flow are more harmful in the glaucomatous optic neuropathy than steady reductions.

The most important *risk factors* of glaucoma include: age, an elevated eye pressure, family history of glaucoma, African or Spanish-American ancestry; farsightedness or nearsightedness; former eye injuries, a thinner central corneal thickness; systemic health problems including diabetes, migraine headaches and poor circulation.

Primary open-angle glaucoma, its most common form, develops gradually and slowly, giving no warning signs. In its case the entrances to the drainage canals are clear and work correctly, the clogging occurs inside the drainage canals. Most of the affected people have no symptoms for many years, until their vision is damaged. If this open angle glaucoma is not diagnosed and treated, it can lead to a gradual loss of vision, though it usually responds well to medication, especially if diagnosed and treated in time.

The other type, i.e. the **angle closure glaucoma** (also known as acute glaucoma or narrow angle glaucoma) is more rare and differs from the open angle glaucoma. In case of an open angle glaucoma there is a reduced flow through the trabecular meshwork, while in case of an angle closure glaucoma, the iris is pushed forward against the trabecular meshwork blocking the out flow of the fluid. In case of an angle closure glaucoma the intraocular pressure usually grows very fast. This happens if the drainage canals get blocked or covered. The iris and cornea are not as wide and open, as they should be. The outer edge of the iris bunches up over the drainage canals, when the pupil gets enlarged too much or too quickly, f.i. when entering a dark room. The attack of an acute angle-closure glaucoma means an ocular emergency, the symptoms of which, in order to save the person's vision, needs immediate treatment. A quick diagnosis, an immediate intervention can significantly influence the outcome of this illness and the patient's morbidity.

Secondary glaucoma can occur as the result of an eye injury, inflammation and tumor or in advanced cases of cataract and diabetes. Certain drugs such as steroids can also cause it. This form of glaucoma may be mild or severe. Its treatment will depend on the fact, whether it is an open angle or an angle closure glaucoma.

Glaucoma is a multietiologic disease of the eyes. The characteristic angle closure can often develop due to ocular inflammations. Many a bacterial or viral infection f.i. caused by *Herpes Simplex Virus, Adenovirus, Mycoplasma, Borrelia B.s.l.,* as well as autoimmune processes can cause the ocular inflammations mentioned above.

Symptoms of angle closure glaucoma may be headache, eye pain, nausea, rainbows around lights at night, and a blurred vision.

Diagnosis: symptomatically and by tonometry, gonioscopy, optical coherence tomography, scanning laser polarimetry, etc.
Treatment: symptomatically. By suppressing the ocular inflammation by applying topical beta-blockers and corticosteroids in order to reverse the process of angle closure and to reduce the damage of the optic nerve. By laser iridotomy. By applying pilocarpine in order to relieve a pupillary block. By reducing the intraocular pressure suppressing the aqueous humor production, by eliminating the pupillary block and by reversing the inflammation with acetazolamide, methazolamide, timolol, carteolol, etc.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances are: 290-294, 299-302, 344-345, 370-374, 378-388, 393, 401-410, 442-452, 466-470 kHz
One should not treat an acute attack of glaucoma with RFR method. There are several medications to be used in order to get a quick decrease of the pressure in the eyes of persons suffering from an acute attack.

20.10. Cataract

Cataract is a vision-impairing disease characterized by a gradual, progressive thickening of the lens. It typically progresses slowly, can cause vision loss and, if untreated, even blindness. Nowadays it is one of the leading causes of blindness all over the world, which is most unfortunate, as this age-related visual morbidity could be, if treated, reversible. Early detection, close monitoring and a surgical intervention in due time is necessary in the management of senile cataract.
Connatal cataracts are diagnosed usually already at birth. If in case of an infant a cataract is undetected, there may ensue a permanent visual loss. Concerning vision, not all cataracts are significant. If a lenticular opacity is present in the visual axis, it is significant as it can lead to blindness. If a cataract present in the anterior portion of the lens or that in the periphery is small, no visual loss can come about.
The most common etiology of connatal cataracts includes intrauterine infections, metabolic disorders and can be genetically transmitted as well. One third of the pediatric cataracts are sporadic and not associated with any systemic or ocular diseases. There may, however, be spontaneous mutations present, which, concerning the patient's offspring can cause a cataract formation. The most frequent mode of transmission occurs in an autosomal dominant way with complete penetrance. This type can be a total cataract, polar cataract, lamellar cataract and nuclear opacity. All closely related members of the family must be examined.
Cataracts of infectious origin are mostly caused by *rubella,* but can also be caused by *chicken pox, EBV, CMV, HSV, VZV, Poliomyelitis virus, Influenza virus, Borrelia B.s.l., Treponema pallidum and Toxoplasma gondii.*
Senile cataracts can be classified into 3 main types, i.e. those of the nuclear cataract, cortical cataract and the posterior subcapsular cataract. In case of age-related cataract forms there can different risk factors be associated with different cataract types. Cortical and posterior subcapsular cataract types are closely related to environmental stresses, such as ultraviolet (UV) radiation exposure, diabetes, drugs (f.i. corticosteroids) and to autoimmune processes and diseases as well. The nuclear type cataract correlates significantly with calcitonin and milk intake and with *nanobacterial* infections.
The pathogenesis of senile cataracts is multifactorial and not fully understood yet. As the lens is aging, its weight and thickness will increase, while its accommodative power will decrease. During this process, termed nuclear sclerosis, its new cortical layers are added to it in a concentric pattern and the central nucleus gets compressed and hard.
Some multiple mechanisms of the senile cataracts can lead to the progressive loss of the transparency of the lens. The lens epithelium undergoes age-related changes, f.i. a decrease in the density of its epithelial cells and an aberrant differentiation of the lens fiber cells.

Another involved mechanism is the conversion of the soluble, low-molecular weight cytoplasmic lens proteins into soluble, high molecular weight aggregates and into insoluble membrane-protein matrices, causing scattered light rays and a reduced transparency of the lenses. Nutrition, involving glucose, trace minerals and vitamins as well, plays likewise a role in the development of this disorder.
Senile cataracts can be associated with a lot of systemic illnesses, including cholelithiasis, allergy, borreliosis, pneumonia, coronary diseases and heart insufficiency, hypotension, hypertension, mental retardation and diabetes.
Diagnosis: by ophthalmoscope, and other ophthalmologic examinations. Nuclear cataracts are characterized by the homogeneity of the lens nucleus with the loss of cellular laminations, while cortical cataracts typically manifest themselves with a hydropic swelling of the lens fibers, with globules of eosinophilic material (Morgagnian globules) seen in slitlike spaces between the lens fibers. A posterior subcapsular cataract is associated with a posterior migration of the lens epithelial cells in the posterior subcapsular area and with an aberrant enlargement of the epithelial cells.
Treatment: by surgery, by applying phenylephrine and corticosteroids.
RFR method: detects and eliminates the pathogen microorganisms.
The most frequent resonances are: 291-293, 299-303, 333, 341-345, 370-376, 396-397, 401-403, 408-415, 422-423, 442-451, 468-469, 507-508, 539-540, 559-566 kHz

20.11. Von Hippel-Lindau Disease

Von Hippel-Lindau disease (VHL) is characterized by benign congenital capillary angiomatous hamartomas of the retina, the optic nerve as well as of various organs. This disease has a hereditary, autosomal dominant form as well as a non hereditary one, and is usually manifested in the second to third decade of the patient's life. Its responsible gene is to be found on the chromosome 3 (3p25-p26), behaving as a typical tumor suppressor gene.
As long as one copy of the VHL gene is producing functional VHL protein in each cell, there will no tumors develop. If a mutation occurs in the second copy of the VHL gene during a person's life, no functioning VHL proteins will be produced by the cells. The lack of this protein allows the development of tumors characteristic concerning the von Hippel-Lindau disease. This hereditary chromosomal defect does predispose this disease and a viral infection will manifest it. Immunosuppressant agents, f.i. HTLV, Mycoplasma species, etc. play an important role in the development of this illness causing damages of the cellular immune response. The viral infection may stay dormant for a few years.
Symptoms: Retinal capillary hemangiomas, usually supplied by large dilated feeder vessels, may be present in any part of the retina. Vitreous hemorrhage, secondary iris neovascularization with glaucoma and cataract formation may follow. Systemic manifestations can occur years after the retinal hemangiomas are noted. Angiomatous hamartomas of the CNS, such as hemangioblastomas in the cerebellum and other organs of the body may also come about.
Diagnosis: by testing of vanillyl-mandelic acid levels in the urine, by genetic analysis, CT, MRI, ultrasound and Doppler sonography.
Treatment: by surgery, f.i. by argon laser photocoagulation, cryotherapy, fluid drainage, scleral buckling, penetrating diathermy, vitreous surgery, endodiathermy.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances are: 450-451, 455-460, 469, 491-492, 530, 557-558 kHz

21. EAR DISORDERS ASSOCIATED WITH INFECTIONS

21.1. Inflammations of the Ear in General

Infectious ear diseases are inflammations of the ears caused by viral, bacterial and fungal infections.

Acute otitis media can be a bacterial, viral or fungal infection of the middle ear, mostly affecting young children. This infection develops usually as a complication of common cold. Viruses and bacteria from the throat can reach the middle ear via the Eustachian tube, or, occasionally, via the bloodstream. A viral otitis media is usually followed by a bacterial one.

Symptoms: Its first symptom is a persistent, severe earache. A temporary hearing loss may come about. Further symptoms include nausea, vomiting, diarrhea, headache, vertigo, chills and fever. If the eardrum ruptures, a discharge from the ear may at first be bloody, then change into a clear fluid and finally to pus. Serious complications such as mastoiditis, petrositis, labyrinthitis, meningitis and brain abscess may also come to pass.

Secretory otitis media is an infection, in which case fluid caused by acute otitis media accumulates in the middle ear.

Acute mastoiditis is a bacterial infection of the mastoid process, the prominent bone behind the ear. Its symptoms appear usually a few weeks after an acute otitis media when the spreading infection destroys the inner part of the mastoid process, where an abscess may develop.

Symptoms include fever, pain around and within the ear and a creamy, profuse discharge from the ear. The pain tends to be persistent and thrombing. An inadequately treated mastoiditis can cause deafness, sepsis, meningitis, a brain abscess and even lead to death.

Chronic otitis media is a long lasting infection which can cause a permanent hole perforation of the eardrum. It is often the result of a too short treatment.

Herpes Zoster of the ear such as the Ramsay Hunt syndrome is an infection of the auditory nerve caused by *Herpes Zoster Virus*, inducing severe ear pain, hearing loss and vertigo. Small fluid filled blisters form on the skin of the outer ear and in the ear canal. If the facial nerve is compressed due to local inflammation and edema, the muscles of the affected side of the face can become temporarily or permanently paralyzed. The loss of hearing may even become permanent, though the hearing may partially or completely return. The vertigo can last for a few days or for several weeks.

Sudden deafness is a form of a severe loss of hearing, usually affecting only one ear, develops in a few hours. It is usually caused by a viral disease such as *mumps, measles, influenza, chickenpox,* or *infectious mononucleosis*.

Diagnosis: symptomatically, by pathogen culturing.

Treatment: by administering effective antimicrobial drugs, even intravenously if needed. Abscess formation may need surgical treatment.

RFR method: detects and eliminates the causative viruses, fungi and bacteria and should be used only after the beginning of the antibiotic treatment. RFR method has a very good effect in case of viral, fungal, or antibiotic polyresistant bacterial infections.

The most frequent resonances found in case of ear infections are: 318-321, 326, 356, 368-372, 383, 396, 401-412, 437, 450, 492, 503-504, 530-536 kHz

The resonant frequencies of Cytomegaloviruses are: 305, 349, 406-412, 512, 534, 548 kHz

The resonant frequencies of Adenoviruses are: 333-336, 340, 370-387, 390-392, 393, 394-400, 402, 523, 534, 560-570 kHz

The resonant frequencies of EBV are: 318, 342-347, 352, 372-383, 403, 422-424, 451, 476-480, 491-493, 516-519, 520-528, 560 kHz
The resonant frequencies of HSV1 are: 290-294, 307, 328, 331-339, 344-346, 357-358, 370-372, 396-403, 413, 431-433, 438, 449, 476, 478, 480-482 kHz
The resonant frequencies of HSV2 are: 300-301, 337-340, 352-365, 366-368, 374-376, 380, 396-397, 403, 413, 425, 434, 450-459, 474, 496, 540 kHz
The resonant frequencies of HZV are: 310, 337-339, 372, 396-398, 410, 416-421, 460, 467, 477, 544, 555 kHz
Other Herpes frequencies are: 366-377, 383-385, 413, 434, 540-544 kHz
The resonant frequencies of Staphylococci are: 294, 308, 323-329, 345, 347, 367, 376-381, 388, 401-402, 421, 434, 448-453, 458, 463, 465, 482, 484, 486, 490-491, 504, 511, 517, 542, 552, 556-557, 563-568, 576 kHz
The resonant frequencies of Streptococci are: 290-294, 317, 320, 330, 340, 349-352, 360-372, 397, 410, 433, 466, 472-480, 515, 550, 567 kHz
The resonant frequencies of the Pseudomonas group are: 323-326, 330-336, 351-361, 364-367, 372-374, 377-380, 388-397, 401, 414, 438, 446-447, 496, 512, 579 kHz
The resonant frequencies of Gonococci are: 307, 330-337, 364 kHz
The resonant frequencies of Borrelia B.s.l. bacteria s[redacted]
The resonant frequencies of Chlamydia are: 316-319, 374-386, 429, 440, 444, 480-483, 566 kHz
The resonant frequencies of atypical Chlamydia forms are: 516-566 kHz
The resonant frequencies of Toxoplasma are: 394-396, 436, 444 kHz
The resonant frequencies of Histoplasma are: 298-306, 315, 375-384, 434 kHz
The resonant frequencies of Mycoplasma are: 320-325, 337-352, 397, 442-451, 499 kHz
The resonant frequencies of the Candida group are: 293, 295, 297, 332, 345, 352-359, 372, 380-390, 396-397, 403, 410, 440-453, 520, 554-559, 572-586 kHz
The resonant frequencies often found in case of ear wax are: 318, 326, 368, 383, 488 kHz

21.2. Otitis Media

Otitis media (OM) is an acute or chronical inflammation of the middle ear affecting mostly children. This inflammation often begins after infections causing a sore throat, common cold and other respiratory or breathing problems, and is spreading to the middle ear. Otitis media can be caused by primary viral or bacterial infections or can be secondary, due to combined infections with other bacteria or fungi. Viruses most often causing an acute otitis media are *Respiratory Syncytial Viruses (RSV), influenza viruses, parainfluenza viruses, rhinoviruses and adenoviruses.*

An acute otitis media implies a rapid onset of the disease associated with abnormal otoscopic findings of the tympanic membrane, such as opacity, bulging, erythema and effusion of the middle ear. There is many a reason why children are more likely suffering from otitis media than adults. Children, due to their still developing immune system, have more trouble in the fighting of infections. The Eustachian tubes of children are small passageways connecting the upper part of the throat to the middle ear. It is shorter and straighter in case of children than in case of adults. It can lead to otitis media in several ways. This tube is usually closed, but opens regularly to ventilate or replenish the air in the middle ear. This tube also equalizes the air pressure of the middle ear in response to air pressure changes of the environment. An eustachian tube blocked by swelling of its lining, or plugged with mucus, cannot open nor ventilate the middle ear. The lack of ventilation may lead to fluid accumulation produced by the tissue lining of the middle ear. If the eustachian tube remains plugged, the fluid cannot drain and will collect in the normally air-filled middle ear.

The adenoids of children are larger than those of adults. These adenoids are composed mostly of lymphocytes helping to fight infections. They are positioned near to the eustachian tubes. If enlarged, they can interfere with the opening of the eustachian tube. In addition, adenoids themselves may become infected, which infection may spread into the eustachian tubes. Bacteria reach the middle ear through the lining or the passageway of the eustachian tube and cause inflammation and swelling of the lining of the middle ear, blocking off the eustachian tube. This inflammation can lead to the formation of pus, a thick yellowish-white fluid in the middle ear.
Symptoms: If this fluid increases, the child will have trouble in hearing, and will experience severe ear pain. These symptoms can be accompanied by one or more of the following symptoms: fever, recent onset of anorexia, irritability, vomiting and diarrhea. Too much fluid can put pressure on the eardrum and tear it, leading to otorrhea.
A persisting fluid in the middle ear and a chronic otitis media can cause a permanent hearing impairment at a time that is critical concerning the child's speech and language development and can lead to speech and language disabilities. If not treated adequately, serious complications may occur such as a chronic suppurative inflammation of the middle ear leading to a definitive loss of hearing (conductive and sensorineural), cholesteatoma, tympanosclerosis, mastoiditis, petrositis, labyrinthitis, facial paralysis, cholesterol granuloma and infectious eczematoid dermatitis. Intracranial complications, such as meningitis, subdural empyema, brain abscess, extradural abscess, lateral sinus thrombosis and otitic hydrocephalus can also come to pass.
Prevention: a child who is prone to have otitis media should avoid the contact with sick playmates and environmental tobacco smoke. By vaccination.
Diagnosis: symptomatically, by otoscopy, labor examinations and bacterial culturing, by serology, by tests with tympanometry, by CT, MRI.
Treatment: by administering effective antibiotics, f.i. high-dose amoxicillin/clavulanate, cefuroxime, etc. By surgery
RFR method: detects and may eliminate viruses, bacteria and fungi!
The most frequent viruses found in these cases are: RSVs, Influenza viruses, Parainfluenza viruses, Rhinoviruses, Herpes viruses, Coxsackie viruses and Adenoviruses. As to their frequencies see the special Chapters.
The most frequent bacteria are: Pneumococci, Streptococci, Staphylococci, Pseudomonas, Klebsiella and Haemophilius influenzae. As to their frequencies see the special Chapters.
Other possible causative agents can be Chlamydia, Mycoplasma, as to their frequencies see the special Chapters.
The most frequent resonances found in case of otitis media are: 258-268, 313-318, 320-322, 360-368, 372-382, 396, 398-412, 448-460, 492, 530-534 kHz

21.3. Otosclerosis

Otosclerosis is an illness in case of which the bone surrounding the middle and inner ear grows excessively, immobilizing the stirrup so that it can not transmit sound properly. The cause of this illness is multifactorial, as it is hereditarily determinated and characterized by a chronic inflammatory process. Otosclerosis may be caused by *Coxsackie viruses* and *nanobacteria.*
Diagnosis: by special auditory laboratory examinations.
Treatment: by surgery
Prevention: By RFR method.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances present in otosclerosis are: 290-293, 310, 324, 348, 371-375, 380-383, 396-398, 401-403, 409, 416-420, 442-451, 467, 474-478, 486, 544, 555, 560-568 kHz

The RFR method can only inhibit the development of the otosclerotic process, but has no effect on an established otosclerosis.

21.4. Meniere's Syndrome

The classic Meniere's disease is a disorder of the inner ear affecting hearing and balance and is characterized by recurrent attacks of disabling dizziness, tinnitus and by a progressive loss of hearing. It affects usually one ear and is caused by an increased endolymphatic pressure of the affected inner ear. The latter symptoms may be absent during the initial attacks of vertigo, but they appear as the disease progresses and increase in severity at the time of an acute attack. In case of a milder form of these syndromes the patients may complain rather of head discomfort and difficultly in concentration than of vertigo and they may be considered to be neurotic. The progressive pathological changes of this Meniere's disease probably occur due to the pressure of the endolymphatic system which leads to the degeneration of the delicate vestibular and cochlear hair cells. Another disorder of the labyrinthine function is characterized by the occurrence of paroxysmal vertigo and nystagmus in case of certain critical positions of the head.

There can be many other causes of aural vertigo, such as purulent labyrinthitis complicating meningitis, serous labyrinthitis due to infection of the middle ear, toxic labyrinthitis due to drug intoxication, f.i. to streptomycin and salicylates, motion sickness, trauma and hemorrhaging into the internal ear.

The etiology of *viral infections* affecting the cranial nerves can always be suspected in case of acute palsies of the facial, trigeminal and auditory nerves associated with Meniere's signs, especially if the symptoms are bilateral, involve several nerves and are associated with pleocytosis. Actually, the only proved virus causing such cases is the Herpes Zoster Virus, so that the searching for this virus in case of Bell's palsy and vestibular neuritis can be rewarding. Since perceptive deafness, vertigo and other cranial nerve palsies can often be experienced in case of parainfectiosus encephalomyelitis caused by *varicella, measles, rubeola, mumps, and scarlet fever* and also in case of the Landry-Guillain-Barre-Strohl syndrome, an allergic immune process must be considered concerning their pathogenesis.

Treatment: by applying anticholinergic drugs, by administering antihistamines, barbiturates, diazepam, betahistine, lipoflavonoids, diuretics, corticosteroids, long lasting acyclovir therapy in case of infections with species of the herpes virus family, etc. Several surgical procedures such as vestibular neurectomy, chemical labyrinthectomy may help in severe cases of vertigo.

RFR method: detects and eliminates the causative virus in the nerves.

The most frequently found resonances are: 290-295, 336, 345-350, 372, 396, 400-410, 416-421, 438, 447-451, 474-476, 530-536, 544-548, 578 kHz

21.5. Tinnitus and Deafness

Tinnitus and deafness are frequent symptoms indicating a disease of the ear, the auditory nerve and its central nervous connections.

Tinnitus, i.e. a ringing in the ears, is a subjective phenomenon and may be felt as a buzzing, whistling, hissing or roaring sound. It is a very common symptom among adults. Low-pitched tonal, vibratory clicks, pops, roaring are signs of the disease of the middle ear and the Eustachian tube. High-pitched tonal, nonvibratory tinnitus occurs in case of diseases of the cochlea and the eighth cranial nerve. A severe, prolonged tinnitus combined with normal hearing is very rare. Tinnitus can be a symptom of almost every ear disorder, including ear infections, blocked ear canal, blocked Eustachian tube, otosclerosis, tumors of the middle ear, Meniere's disease, loss of hearing, and can be a side effect of drugs (f.i. aspirin, streptomycin, neomycin), can be caused by anemia, heart and blood vessel disorders (f.i. hypertension, arteriosclerosis), hypothyreosis, brain tumors, etc.

Deafness is a frequent symptom with two types:
1. The sensorineural one, which is a neural deafness developing due to cochlear diseases or the interruption of nerve fibers, and
2. Conduction deafness, caused by diseases of the middle ear, f.i. otosclerosis and chronic otitis, or by the occlusion of the external auditory canal or the Eustachian tube.
Diagnosis: by auditory laboratory tests, Békési audiometry, etc.
Differential diagnosis: by distinguishing it from Treacher-Collins syndrome, Engelman dysplasia, Pendred's disease, Hallgren's disease, Alström syndrome, Refsum's disease, Waardenburg's disease, Meniere's disease, etc.
Treatment: in case of infections by administering effective antiviral and antibiotic drugs. By surgery, by hearing aids.
RFR method: detects and eliminates the pathogen microorganisms
Nanobacteria and Chlamydia species can lead to otosclerosis.
The resonant frequencies of Nanobacteria are: 294-298, 305-310, 320-328, 336-345, 372-375, 395-397, 424-442, 466-476, 480-486, 556-568 kHz
The resonant frequencies of Chlamydia are: 316-319, 420-430, 440, 443-445, 476-485, 497, 562-568 kHz
Other resonant frequencies often found in case of otosclerosis are: 375-387, 424-442, 466-476, 548-568 kHz
Coxsackie viruses and Herpes Simplex Virus-1 can cause inflammation and damages of the auditory nerve and of its nerval centrum.
The resonant frequencies of Coxsackie viruses are: 298-305 kHz as to its other frequencies [redacted]
The resonant frequencies of Herpes Simplex Virus-1 are: 290-294, 344-346 kHz, as to its other frequencies [redacted]
The most frequent resonances of other, often found, non identified pathogens are: 330-338, 347-349, 397-398, 400-403, 408-411, 448-451, 478 kHz
In case of an infectious secondary tinnitus and deafness the symptom is combined with other otologic diseases, f.i. with Meniere's disease, etc. The concerning frequencies see their Chapters.

21.6. Vestibular Neuritis and Labyrinthitis

Vestibular neuritis (VN) can cause a single paroxysmal attack of *vertigo*, as well as series of attacks and even a persistent disorder ceasing in more than two weeks. It may be associated with *nausea, vomiting* and with previously occuring infections of the upper respiratory tract. Unlike labyrinthitis, it has generally no auditory symptoms. This disorder attacks people in the fourth and fifth decades of their life mostly in spring and the early summer. It is caused by the inflammation of the vestibular nerve, connecting the inner ear to the brain. In case of vestibular neuritis, the dizziness felt is attributed to a *viral infection* of the vestibular nerve. The vestibular nerve carries information about head movements from the inner ear to the brain. If one of the two vestibular nerves gets infected, an imbalance between the two sides will come about, leading to vertigo.
Vestibular neuronitis is an other term used for the same clinical syndrome. These various terms for the same clinical syndrome probably reflect our lack of ability to localize the site of the lesion. The term „neuritis" implies damages of the nerve, while „neuronitis" means damages of the sensory neurons of the vestibular ganglion. There are actually evidences for both and even for viral damages to the vestibular nuclei within the brainstem, a second potential „neuronitis". As the vestibular neurons are distinct from the cochlear ones within the brainstem, it makes it more evident, that this type of neuronitis does not cause hearing symptoms. Vestibular neuronitis may be associated with eye nystagmus.

In case of vestibular neuritis, the causative virus is thought to be usually a member of the herpes family i.e. *H. simplex viruses, HZV* and other viruses such as *measles, mumps, EBV, Coxsackie* and *ECHO viruses.*

Labyrinthitis can cause the combination of the symptoms of vestibular neuritis, with the addition of hearing symptoms. It may be caused by a pathological process affecting the whole inner ear or the 8th nerve. Labyrinthitis is always attributed to infections, being an infectious inflammatory disorder of the inner ear or labyrinth affecting one or both ears. Bacteria and viruses can cause acute or chronic inflammation of the labyrinth due to their local or systemic infections.

Suppurative or bacterial labyrinthitis exists but very rarely, though it can occur in case of an acute or chronic otitis media and is almost always associated with cholesteatoma. A profound loss of hearing, severe vertigo, ataxia, nausea and vomiting are common symptoms of bacterial labyrinthitis.

Serous labyrinthitis can occur due to bacterial toxins and other inflammatory mediators getting into the inner ear. The anatomic relationship between the labyrinth, middle ear, mastoid, and subarachnoid space is essential to understand the pathophysiology of labyrinthitis. The labyrinth is composed of an outer osseous framework surrounding a delicate membranous network that contains the peripheral sensory organs (utricle, saccule, semicircular canals, and cochlea) for balance and hearing. The symptoms of labyrinthitis occur if infectious microorganisms or inflammatory mediators invade the membranous labyrinth and damage the vestibular and auditory end organs. The labyrinth maintains connections with the central nervous system and the subarachnoid space by way of the internal auditory canal and the cochlear aqueduct. Bacteria may get to the membranous labyrinth by these pathways or due to connatal or acquired defects of the bony labyrinth. Viruses typically spread to the labyrinthine structures hematogenously or by way of the above mentioned preformed pathways.

Viral labyrinthitis is characterized by the sudden unilateral loss of vestibular function and hearing. The acute onset of a severe vertigo, frequently associated with nausea and vomiting, is characteristic for this disorder. Vertigo eventually resolves after several days or weeks; however, unsteadiness and positional vertigo may persist for several months. A loss of hearing of any degree and type, though it mostly affects higher frequencies, is also present and may be the first symptom. A spontaneous nystagmus towards the unaffected side with diminished or absent caloric responses in the affected ear can be usually experienced.

Ramsay Hunt syndrome, i.e. Herpes Zoster oticus is a unique form of viral labyrinthitis. This disorder is caused by the reactivation of a latent VZV infection occurring years after the primary infection. Deep, burning, auricular pain are its beginning signs, followed a few days later, by a vesicular rash in the external auditory canal and concha. Vertigo, loss of hearing and unilateral weakness of the facial muscles can also be exprienced. Some patients will improve others will suffer a permanent loss of hearing and a persistent reduction of caloric responses.

In case of labyrinthitis the causative agents are usually *viruses,* although, rarely, labyrinthitis can be the result of a bacterial middle ear infection f.i. in case of *borreliosis.* Viral infections can cause both congenital and acquired loss of hearing. *Rubella, Coxsackie virus and Cytomegalovirus* are the best-recognized viral causes of prenatal loss of hearing. Loss of hearing got due to viruses in the postnatal period is usually owing to *mumps* or *measles.*

Vestibular neuritis and labyrinthitis are seldom painful, but if, the patient should be treated at once, as there may be a treatable *B. fragilis* or *Klebsiella* bacterial infection or/and a *herpes viral infection* present.

The symptoms of vestibular neuritis and labyrinthitis are dizziness or vertigo, disequilibrium or imbalance, nausea and vomiting. After a few days, these symptoms can be provoked only by sudden movements, mostly by the sudden turning of the head.
Diagnosis: symptomatically, by ENG, audiogram, nystagmus examination, VEMP. By blood tests for diabetes, thyroid disorders, immune vascular diseases, autoimmun diseases. By serologic tests and PCR examinations in order to find treatable causative illnesses such as syphilis, borreliosis and other bacterial or viral infections. By MRI scan in order to exclude any reasonable possibility of a stroke or brain tumor, etc.
Differential diagnosis: Meniere's disease.
Treatment: symptomatically, by administering meclizine, lorazepam, phenergan, compazine, diazepam, etc. By administering antibiotics (amoxicillin) in case of the evidence of a middle ear infection (f.i. ear pain, fluid, redness or pus behind the eardrum) and broad-spectrum antibiotics or combination therapy with CNS penetration, if needed. By administering antiviral drugs (acyclovir, famciclovir and valacyclovir) in order to shorten the duration of viral shedding in persons with Herpes Zoster oticus and, if started early in the clinical course to prevent some auditory and vestibular damages.
RFR method: detects and may eliminate the pathological microorganisms.
RFR method is able to eliminate the causative viruses.
The most frequent resonances are: 287-290, 295-303, 313-321, 344-345, 360-363, 369, 372-387, 395-405, 408-410, 416-420, 442-445, 450, 471-473, 526, 576-586 kHz
In case of viral labyrinthitis the RFR method may be lifesaving. In case of a concomittant bacterial infection given, the administration of effective antibiotics is at first needed and must be then followed by RFR method. In case of an antibiotic resistant bacterial infection the RFR method is of great value. In case of a neuroborreliosis the treatment will last for a very long time, in order to eliminate borrelia present in the brain and in all other parts of the body.

21.7. Autoimmune Inner Ear Disease

Autoimmune inner ear disease (AIED) implies a direct attack of the person's immune system against the *Coxsackie viral antigens*. This virus or/and its antigen are absorbed to the cochleovestibular system changing the antigenity of this tissue, provoking an immune-mediated response in the inner ear. This autoimmune disorder is characterized by a progressive sensorineural loss of hearing and/or dizziness caused by attacking antibodies and immune cells in both inner ears. This reduction of hearing is mostly accompanied by tinnitus (i.e. ringing, hissing, roaring) present for more than a few months. Its bilateral episodes of the loss of hearing and tinnitus may resemble Meniere's disease, but these are accompanied by auto-antibodies in the serum. Half of the patients with AIED suffer from imbalance symptoms as well.
AIED is a clinical diagnosis based on its distinct clinical course, immune test results and its response to the treatment. The most important diagnostic sign is the improvement of hearing in case of administering immunosuppressants. In case of AIED there are combined co-infections caused by species of Coxsackie virus, HTLV, Herpes virus and Mycoplasma. There are several other ways leading to autoimmune damages in the inner ear, f.i. in case of Ankylosing spondylitis, SLE, Sjögren's Syndrome, Cogan's disease, Ulcerative colitis, Wegener's granulomatosis, Rheumatoid Arthritis and scleroderma which all can cause, or be associated with AIED. In case of Bechet's disease, an other multisystem disease, there commonly occur audiovestibular problems.
Genetic factors controlling or being associated with certain responses of the immune system may increase or otherwise be associated with increased susceptibility to certain hearing disorders, f.i. to Meniere's disease. This loss of hearing is probably associated with HLA-DRB1*04, DQA1 03 and 05 haplotypes.

Diagnosis: is based on the patient's history, physical examinations, blood tests, and the results of hearing and vestibular tests. By ABR testing and otoacoustic emmission tests.
Treatment: by administering immunosuppressive drugs, such as corticosteroids, cyclophosphamide, methotrexate, azathioprine and biological response modifier drugs, f.i. etanercept.
RFR method: detects and may eliminate all causative pathogen microorganisms.
The most frequent resonances are: 287-288, 290-303, 306-324, 442-451, 576-580 kHz

22. DISEASES OF THE MOUTH, TEETH AND FACE, ASSOCIATED WITH INFECTIONS

The oral mucosa may be damaged by local bacterial, viral and fungal infections and injuries. Systemic diseases f.i. diabetes mellitus, AIDS, autoimmune disorders and leukemia can damage the mouth as well.

22.1. Mouth Infections

22.1.1. Thrush

Candida albicans resides normally on the mucous membranes in small numbers and can be sometimes cultured from the oral mucosa and feces of healthy persons, too. In case of debilitated patients, the fungus can grow and produce white patches on the buccal mucosa, the tonsils, cheeks, gums, tongue, and initiate a mild inflammatory reaction. These patches can easily be removed leaving a reddened surface. Though usually self-limited, the disease may become chronic and spread to other mucosal surfaces and to intertriginous areas of the groins, the breasts, the armpits and the umbilicus. Chronic moniliasis of the oral mucous membranes can induce chronic, hyperplastic changes resembling leukoplakia. In case of pregnancy and diabetes mellitus, Candida albicans frequently causes a mild superficial infection of the mucous membraines of the mouth.

Symptoms: The creamy white patches typical of thrush cling to the tongue and to the sides of the mouth, are often painful causing a burning sensation. These patches can be scraped off easily. Thrush is not unusual among otherwise healthy infants, but in case of adults it may signal an impaired immunity, caused possibly f.i. by diabetes mellitus or AIDS. The use of antibiotics while killing off bacteria increases the chance of getting thrush. This disease does often occur together with myeloproliferative and hematologic diseases, intensive courses of immunosuppressive drugs and broad-spectrum antibiotics.

Diagnosis: symptomatically and by microscopic examinations.

Treatment: by administering nystatin, myconazol, fluconazol etc. and by applying local antifungal prepatations.

RFR method: detects and may eliminate the pathogen fungi.

The resonant frequencies 420-434, 476-478 kHz are often effective, there are **other Candida frequencies as well, f.i.: 293, 295, 297, 332, 345, 352-359, 372, 380-390, 396-397, 403, 410, 440-453, 520, 554-559, 572-586 kHz**

22.1.2. Aphthous Stomatitis (Canker Sores)

Canker sores are often occuring as small, painful sores appearing inside the mouth being round white spots with a red border. These sores exist almost always on soft, nonkeratinized or poorly keratinized surfaces of the oral mucosa, gingiva, soft plate or the ventral surface of the tongue. Canker sores are classically sorted into 3 clinical forms: i.e. into minor, major, and herpetiform ones.

Small canker sores often appear in groups of two or five; disappear generally by themselves within a few days and do not leave scars. They are painful, shallow, recurrent ulcerations measuring from 3 mm to smaller than 1 cm in diameter, occuring on the labial and buccal mucosa and on the floor of the mouth.

Larger sores, formerly known as periadenitis mucosa necrotica recurrens, are less common; may be irregularly shaped, oval ulcers 1-3 cm in diameter, can take many weeks to heal, leaving frequently scars and severe distortions of the oral and pharyngeal mucosa. The pain lasts for 3 to 10 days, worsens if the tongue rubs the sore or, if the person eats hot or spicy food.
Herpetiform sores are its smallest forms, measuring 1-3 mm in diameter, tend to occur in clusters, and may be small and localized, or distributed on the soft mucosa of the oral cavity. More severe canker sores can cause fever and swollen lymphnodes at the neck.
A recurrent aphthous stomatitis is a T-cell mediated localized destruction of the oral mucosa, in which case a cytotoxic reaction of lymphocytes and monocytes in the oral epithelium seems to cause the ulceration, the mechanism of which is not yet cleared.
Aphthous stomatitis is frequently associated with systemic diseases, such as Behçet Syndrome and Inflammatory Bowel Diseases. Behçet Syndrome is strongly associated with the HLA-B51 haplotype. Genetic predisposition, immune dysregulation, physical or emotional stress, allergy and different microbial infections can all play a role in the development of this syndrome. These lesions may be associated with HIV infection as well. Canker sores can be caused by spirochete bacteria, though viral infections caused f.i. by HSV, other herpes viruses, VZV, CMV, EBV may promote their developing as well.
Diagnosis: symptomatically, by biopsy, by culturing bacteria and fungi.
Treatment: symptomatically and by applicating clorhexidine gluconate, betadine, diluted hydrogen peroxide, lidocaine, benzocaine. By administering antibiotics (Doxycyclin), anti-inflammatory drugs and immunomodulatory agents.
RFR method: detects and eliminates these microorganisms!
The most frequent resonant frequencies in case of aphthous stomatitis are: 290-294, 302-303, 321-324, 330-332, 344-351, 372-378, 380-381, 391, 400, 408-414, 416-420, 425, 433-434, 442-454, 468, 471-476, 483-491, 498, 502-504, 509, 534, 547, 569 kHz
The development of aphthous stomatitis is influenced and iniciated by *Mycoplasma orale* living in the oral cave, determining the local immune response.
The frequencies of Mycoplasma orale are 450-454, 485-488, those of Mycoplasma hominis are 502-504 kHz, those of Mycoplasma pneumoniae are 321-324 kHz, all measured and found in the saliva of aphthous patients. Mycoplama is the etiological agent of oral ulcerations.
Behçet's Syndrome, Sjögren's Syndrome and Inflammatory Bowel Diseases are systemic diseases associated with *Mycoplama fermentans* infections.
Regarding certain immune response mechanisms, see the special Chapter of Mycoplasma.
Mycoplasma species can change the immune response to viral infections and develop an oral ulcerative process.

22.1.3. Oral Herpes Infections

Primary oral herpes infection, i.e. **primary herpetic gingivostomatitis** is the first infection caused by *Herpes Simplex Virus,* characterized by rapidly developing, painful sores on the gums and other parts of the mouth. Secondary herpes infections such as the **recurrent herpes labialis** is a local reactivation of this virus producing a cold sore. Primary herpes infection affecting infants causes a general gum inflammation and an extensive mouth soreness. The child may have fever, swollen lymph nodes at the neck, and feel discomfort. Most cases are mild and go unrecognized. The soreness may be anywhere in the mouth but they always include the gums. People escaping oral herpes in childhood but getting it as adults will usually have more severe symptoms. Unlike the original infection the later flare ups usually produce cold sores i.e. fever blisters. These flare-ups are often triggered on the lips by sunburn, cold, fever, food allergy, mouth injury, etc. For a day or two before a blister appears, the person may feel tingling or discomfort at the spot where the blister will erupt. Sores in the mouth start as small blisters quickly running together forming a painful,

red sore. The symptoms of a herpes simplex infection depend on the response of the immune system. AIDS, chemotherapy, radiation therapy, immunosuppressive drugs can weaken the immune defense, so that the virus can spread all over the body and cause even a fatal brain infection.
Treatment: symptomatically. By administering acyclovir in case of immune deficiency. Corticosteroids are contraindicated as they may facilitate the infection to spread.
RFR method: detects and eliminates the herpes virus!
The most frequent resonant frequencies in case of Herpes labialis are: 290-294, 344-346, 352-353, 372, 402, 440-452 kHz

22.1.4. Hand-Foot-and-Mouth Disease

Hand-foot-and-mouth disease (HFMD) is caused by viruses belonging to the *Picornaviridae*: This viral infection is characterized by fever, a typical rash mostly appearing on the palms, hands, soles of the feet and inside the mouth. It should not be confused with foot and mouth disease affecting cattles, sheep, swine and immunocompromised people. HFMD is caused by several members of the *Enterovirus* family. Its most common cause is *Coxsackie virus A-16*; the less frequent one being *Human Enterovirus 71*. This group of RNA enteroviruses include also *Coxsackie viruses A5, A9, A10, B1* and *B3*, though *Herpes Simplex Viruses* (*HSV*) can also cause these symptoms.
The illness can be transmitted via the fecal-oral and the oral-oral way, or less likely via respiratory droplets. The virus is typically dispersed within the gastrointestinal tract, in the buccal mucosa or the ileum. After an incubation period of 72 hours, viremia will occur spreading through the nearby lymph nodes.
Infants and children are those mostly affected by this HFMD, which is slightly contagious, typically occuring in small epidemics in nursery schools or kindergardens, during the months of summer and autumn. This illness usually promises full recovery, though the *HEV71* infections can cause severe complications and even death. More severe complications may affect the CNS and the cardiopulmonary system, too. These sequelae are dysphagia, limb weakness, cardiopulmonary failures, and can lead even to death, mostly due to pulmonary hemorrhages and edema. Though common enteroviruses may cause aseptic meningitis and encephalitis, HFMD is usually not associated with meningitis.
Symptoms of the HFMD are low grade fever, malaise, abdominal pain, upper respiratory symptoms, painful oral lesions starting as pink macules and papules progressing into vesicles with a surrounding erythema, sores with blisters on palms and soles, as well as on the buttocks of small children and infants, mouth ulcers also included. Not all these symptoms can be experienced in every case of HFMD.
Diagnosis: Symptomatically. By virus culturing, by serology.
Differential diagnosis: by distinguishing it from atypical HSV infections using Tzanck cell smear examinations.
Treatment: symptomatically as there is no specific treatment.
RFR method: detects and may eliminate the causative viruses and, if present, can treat the immunocompromised state as well.
The most frequent viral resonances found in HFMD are: 307, 310-319, 321, 324-331, 342-350, 375-386 kHz
The resonant frequencies of HSV are: 291-293, 344-345 kHz
The resonant frequencies of Mycoplasma fermentans are: 440-451 kHz
The resonant frequencies of Epstein-Barr Virus are: 372-383, 518-519 kHz

22.2. Dental Infections

The principal cause of the loss of teeth of people under fourty is **dental caries**, characterized by bacteria-induced progressive destructions of the mineral and organic components of the outer enamel and its underlying dentin. If a carious infection progresses unnoticed, an eventually developed infection of the dental pulp can give rise to acute pulpitis. The most common manifestation of a periapical disease is periapical granuloma, a localized mass of chronic granulation tissues slowly expanding at the expense of the surrounding alveolar bone. This chronic periapical granuloma can cause symptoms or remain asymptomatic. A persistent untreated periapical granuloma may result in a periapical cyst or an abscess. An acute periapical abscess may extend into the surrounding bone marrow and cause osteomyelitis. More frequently, the abscess perforates the cortical plate, and spreads through various tissue spaces, giving rise to cellulitis and bacteremia, or discharges into the oral cavity and the maxillary sinus, leading to secondary illnesses. Ludwig's angina, a serious cellulitis of the tissues of the floor of the mouth affecting adults, originates usually from an infected mandible molar tooth infection.
Diagnosis: symptomatically, by x-ray, and macroscopic examinations.
Treatment: conservatively, or surgical, together with antibiotic defence.
RFR method: can be used only in order to prevent the disease. Detects and eliminates the pathogens in the mouth.

22.2.1. Pulpitis

Pulpitis is a very painful inflammation of the tooth pulp, the innermost part of the tooth containing the nerves and blood supply. The most common cause of pulpitis is tooth decay, the second frequent one being injury. Severe inflammations can destroy the pulp. An increased pressure may push the pulp through the end of the root to the jawbone and the surrounding tissues. To determine whether the pulp is healthy enough to be saved, a dentist can perform certain tests.
Treatment: the inflammation can cease if its cause is treated by the dentist.
RFR method: detects and eliminates the pathogen microorganisms!
The most frequent resonant frequencies of dental infections are: 307, 313-318, 325-330, 332, 340, 348-350, 358-364, 372-388, 395-404, 408-412, 425, 450-452, 460, 476, 530-533, 559, kHz
The most frequent resonant frequencies of tooth decay are: 293-297, 326-331, 367-375, 383-388, 396, 400-404 kHz
The most frequent resonant frequencies of tooth plaques are: 294-298, 305-311, 340-344, 378-387, 424-436, 466-476, 556-568 kHz

22.2.2. Periapical Abscess and Granuloma

A periapical abscess is a collection of pus, originating usually from a dental infection, spreading from a tooth to the surrounding tissues. The person's immune system attacks with a number of white blood cells this infection, the pus contains these cells, bacteria and dead tissues as well.
This pus drains first into the gum, so that the gum near the root of the tooth will get swollen. Depending on the location of the tooth, the pus may drain into the skin, the mouth, throat, or the skull. A periapical granuloma contains chronic granulomatous tissues and may cause dental cavities, missing teeth, sensitive teeth, teeth grinding and tooth pain as well.
Treatment: by dental surgery, by administering antibiotics, and by local applications.
RFR method: should not be used for treating!
The most frequently found pathogen is Streptococcus pyogenes, the most frequent resonances of which are: 369-375 kHz

22.3. Periodontal Infections and Parodontopathy

Periodontal infections inflame and destroy their surrounding structures supporting the teeth, i.e. primarily the gums, the outer layer of the tooth root, then the bone. Periodontal diseases are caused mainly by coinfections of bacteria with viruses and fungi. General immune problems, associated f.i. with diabetes mellitus, leukemia and immunosuppressive processes such as AIDS, etc., as well as with smoking, being all predisposing factors for the development of these infections.

22.3.1. Gingivitis

Gingivitis is the inflammation of the gingiva. The inflamed gums are red, swollen and bleed easily. Gingivitis is a very common disorder, can develop at any time after a person's dentition. It can be the result of inadequate brushing and flossing which allows plaque to remain along the gum line of the teeth. Plaque is the main cause of gingivitis, though also other factors can worsen this inflammation, f.i. pregnancy, puberty and birth control drugs. **Some drugs** such as cyclosporin, phenitoin, nifedipin etc. can often cause the overgrowth of the gums, leading to chronic gingivitis, in which development first a simple gingivitis will be experienced. **Acute and chronic herpetic gingivostomatitis** is a painful viral infection mostly caused by HSV1.

Desquamative gingivitis is a poorly understandable, painful disease occuring mostly among postmenopausal women. The process is often allergic and related to autoimmune diseases.

Gingivitis can be associated also with **leukemia,** other tumors and hematologic malformations.

Trench mouth (named also Vincent's infection, Vincent's angina, Vincent's stomatitis), is a noncontagious, acute, necrotizing ulcerative gingivitis affecting mostly young adults, in which case the gums are painful, fever and fatigue are also characteristic of it. Trench mouth is may be an allergy/autoimmune mediated illness. Poor oral hygiene, nutritional deficiency, heavy smoking and debilitating diseases may be predisposing factors of this disease. *Fusobacterium fusiforme* combined with the spirochetes *Borrelia vincentii* and *Treponema denticola* are the possible infective agents of this inflammation. The gums bleed easily on pressure, eating and swallowing cause pain and a bed smell of the breath can be sensed. Papillary and marginal gingival necrosis and ulceration are characteristic. The lymph nodes under the jaw are enlarged, a chronic low grade fever may also come to pass.

This acute necrotizing ulcerative gingivitis differs from acute herpetic gingivostomatitis, with which it is frequently mistaken. Trench mouth patients respond rapidly to penicillin or broad-spectrum antibiotics, while in case of this disease high fever and malaise but seldom develop. Risk factors, f.i. smoking, poor oral hygiene, diphenylhydantoin therapy and exposition to cadmium may play a role in its severity.

Treatment: administering antibiotics can help and instead of brushing the teeth for the first few days of the treatment, topically 1% hydrogen peroxid solution should be applied several times a day.

RFR method: detects the pathogen microorganisms and eliminates them!

The most frequent resonant frequencies found in case of gingivitis are: 307, 325-327, 330-340, 350, 371-374, 383, 388, 396-404, 408-412, 416-426, 450-453, 476-478, 530-533, 559 kHz

22.3.2. Periodontitis, Parodontopathy

Peridontitis with pyorrhea can occur if the gingivitis extends to the supporting structures of the tooth. The pockets of the gingiva get deeper, the plaques harden into dental calculus (tartar) and more plaques will accumulate on their top. Plaques get down to the root of the

teeth and, eventually, destroy the bone supporting the tooth. Without this support, the tooth gets loose and falls out. Though local infections caused by bacteria together with viruses and fungi can be experienced, paradontopathy is a typical immune disease. Many a medical condition, including diabetes mellitus, Down's syndrome, Crohn's disease, severe leukocytopenia and AIDS can predispose a person to develop periodontitis. Concerning people with AIDS parodontopathy progresses quickly.
Chronic destructive periodontitis is responsible for the loss of more teeth than dental caries does. This process begins as a chronic marginal inflammation of the gingivae, spreading slowly and involving the underlying alveolar bone and the periodontal ligament. The alveolar bone can get absorbed, resulting in the loss of the periodontal ligament fiber attachment binding the tooth to the bone. The separation of the soft tissue from the tooth surface results in poket formation, the inner side of which bleeds readily when touched and when chewing. Pus exudating from the gingival margin is termed pyorrhea. With the continueing loss of alveolar bone, the involved teeth will become loose. An occluded deep periodontal pocket leads to a periodontal abscess. Chronic periodontitis is caused by several *bacterial, viral and fungal* infections, resulting in the accumulation of visible adherent masses of pathogens, which masses will develop into a mineralized bacterial plaque (produced by *nanobacteria*), resulting in calculus. This process is influenced by the immune modulating effects of *Mycoplasma salivarium* species, so that the host's response to this infection can not be specific enough. All these facts are causing a local autoimmune disease at the end.
Diagnosis: symptomatically, by bacterial culturing, PCR, etc.
Treatment: by administering effective antibiotics, metylsalycilate, local antiseptic preparations and surgery.
RFR method: detects and may eliminate the pathogens found in the mouth, including those with tooth decay and tooth plaque frequencies. In case of RFR examination the application of special oral elctrodes is advisable.
The most frequent resonances are: 290-297, 305-310, 326-331, 340-345, 353-358, 360-388, 396-403, 408-426, 434-437, 485, 556-568 kHz
The resonance frequencies of Mycoplasma salivarium are: 387-389, 426-430, 461-463, 518, 570-572 kHz and Mycoplasma fermentans 442-451 kHz

22.3.3. Oral Ulcer

Oral or mouth ulcer is the name of an open sore inside the mouth affecting either its mucous membrane or the epithelium of the lips and its surroundings. These oral ulcers may be caused by physical or chemical trauma, infections, medical intervention and medication, by cancerous processes and may be the symptoms of systemic diseases. In certain cases they are caused by an overreaction of the person's immune system. Once formed, the ulcer may persist owing to inflammation and/or secondary infection.
Factors appearing to provoke oral ulcers are stress, fatigue, illness, injury caused by accidental biting of the mouth when eating, hormonal changes, menstruation, sudden weight loss, food allergies and deficiencies in vitamin B12, iron and folic acid.

22.3.3.1. Oral Ulcers Caused by Viruses

The most common oral ulcer types are *aphthous ulcers (canker sores)* and *cold sores* [redacted]
Infections caused by *Herpes Zoster Virus (Shingles), Varicella Zoster Virus (Chickenpox), Coxsackie A and B viruses and ECHO virus subtypes* can all be associated with oral ulcerations. A *HIV*-caused immunodeficient state allows opportunistic infections or neoplasms to proliferate.

22.3.3.2. Oral Ulcers Caused by Bacteria

Bacterial infections leading to ulceration of the mouth can be caused by *Mycobacterium tuberculosis* and *Treponema pallidum (syphilis)*. Opportunistic pathogens combined with otherwise nonpathogen bacteria, such as *aerobic Streptococci, Neisseria, Actinomyces, Spirochetes, Mycoplasma and Bacteroides species* can prolong and reactivate ulcerative processes, cause ulcers in case of persons in immunodeficient state.

22.3.3.3. Oral Ulcers Caused by Fungi

Fungal infections caused by f.i. *Coccidioides immitis* (valley fever), *Cryptococcus neoformans* (cryptococcosis), *Blastomyces dermatitidis* (Blastomycosis) can develop oral ulcerations.

22.3.3.4. Oral Ulcers Caused by Protozoa

Entamoeba histolytica, this parasitic protozoan can cause mouth ulcers due to the formation of cysts.

22.3.3.5. Oral Ulcers in Case of Immune Compromised States

In case of immunodeficiency caused by various different disease processes aphthous and other oral ulcers are common symptoms.

Aphthous ulcers are thought to occur, if the body becomes aware of and attacks chemicals not exactly recognized. The presence of unrecognized molecules provoke reactions of local lymphocytes, which start a reaction leading to the development of a local oral ulcer. Repeated occurings of mouth ulcers can indicate immunodeficiency, signaling low levels of immunoglobulins in the oral mucous membranes. Chemotherapy, infections with HIV, other HTLVs and EBV can all cause immunodeficiency and thus oral ulcers. Autoimmune diseases can also cause oral ulcerations. Pemphigoid of the mucous membranes, an autoimmune disorder affecting the epithelial basement membrane, causes desquamation and ulceration of the oral mucosa.

Contact with allergens can lead to the ulceration of the buccal mucosa as well.

Oral cancers, f.i. Squamous Cell Carcinoma can be ulcerated as its center loses its blood supply and gets necrotic.

Systemic illnesses and disorders associated with oral ulcers are f.i. Behçet's disease, bullous pemphigoid, celiac disease, Crohn's disease, oral lichen planus, SLE, neutropenia, candidiasis and ulcerative colitis.

The symptoms of oral sores include fever, feeling of general discomfort, uneasiness, tiredness, very sore mouth with no desire to eat and a bed smell of the breath.

Treatment: depending on the causative factors, f.i. by administering antibiotics, acyclovir, choline salicylate, euthymol, corticosteroids, etc.

RFR method: detects and may eliminate the viral, bacterial and fungal pathogens.

The most frequent frequencies in case of Cold sores are: 291-293, 344-345 kHz

The most frequent frequencies of VZV are: 416-420, 544-545 kHz

The most frequent frequencies of EBV are: 372-383, 518-519 kHz

As to other pathogens see their special Chapters.

Only the RFR method is able to eliminate all viruses.

22.4. Sinusitis and Infections Affecting the Face

Acute sinusitis is most frequently caused by *Streptococcus pneumoniae, Streptococcus pyogenes, Staphylococcus aureus, Klebsiella pneumoniae and Haemophilus influenzae*, rarely together with *Candida species*. Other bacteria may also be involved in case of patients receiving immunosuppressive therapy, those who take long-lasting antibiotics, or

those with penetrating trauma, or vasculitis. The most common predisposing factor of **acute purulent sinusitis** leading to the obstruction of the drainage of the paranasal sinuses is the viral infection of the upper respiratory tract.
Its **symptoms** are: local pain, tenderness and low-grade fever. Sinus infections can be caused by a large number of pathogen viruses, including *Adenovirus, Influenza and Parainfluenza virus, Rhinovirus, Herpes viruses* and others. Acute sinusitis usually heals when the viral disease subsides. In a number of cases, an invasion by anaerobic pyogenic bacteria supervenes and is responsible for the development of purulent sinusitis. **Wegener's granulomatosis** may produce the clinical picture of an acute or chronic sinusitis. Bacterial superinfection can frequently occur among patients suffering from this granulomatous illness, and are often examined only after an infection had developed, thus the underlying granulomatous lesions will often remain overlooked. Recurrent or prolongated episodes of sinusitis, that are refractory to antimicrobial therapy, or that relapse soon after the treatment is discontinued, must be thoroughly investigated concerning the presence of allergy and noninfectious obstructing lesions, f.i. developmental or traumatic anatomical pathology, nasal septal elevation, papilloma and malignant tumor. Bacterial meningitis can be a rare complication of purulent frontal sinusitis, and may be associated with cranial osteomyelitis and a subdural brain abscess. The diagnosis of acute purulent sinusitis is usually made when its symptoms (such as fever, chills, pain and tenderness of the involved sinuses, nasal obstruction and recurrent headache changing in intensity depending on the position of the head) are present. The isolation of the pathogenic organisms obtained from nasal secretions may help to establish the diagnosis.
Chronic sinusitis is caused by persistent infections. This illness develops in case of viral and bacterial infections, which pathogen attack causes fever, chills, pain and tenderness of the involved sinuses, nasal obstruction and recurrent headaches changing in intensity depending on position. Complications may occur due to intracranial spreading of the infection from the sinuses via the diploic veins. An usual form of chronic sinusitis in association with bronchiectasis and situs inversus is the Kartagener's syndrome.
Allergic rhinitis generally occurs among atopic individuals, persons with a family history of a similar or related symptom-complex i.e. eczematous dermatitis, urticaria and asthma. Allergic rhinitis causes sneezing, rhinorrhea, obstruction of the nasal passages, conjunctival and pharyngeal itching and lacrimation. It often occurs seasonally owing to its relation to airborne pollens, though it can have other causes as well. The hypersensitive state can be caused by an *ascaris* infection, which state enhances the possibility of an allergic attack provoked by pollen and other allergens, f.i. cat glycoproteins.
Diagnosis: symptomatically, by x-ray and by isolating the pathogens.
Treatment: by administering effective antibiotics, antifungal drugs and surgical drainage.
RFR method: detects and eliminates the viruses, bacteria and fungi!
The most frequent resonances are: 290-298, 307-315, 317-319, 326, 332-337, 349, 364, 372-374, 378-387, 393, 398-406, 408, 410-426, 442-444, 448-450, 460, 486-528, 536, 560-572 kHz

22.5. Pharyngitis

Pharyngitis is an inflammation of the pharynx and tonsils caused by irritations or infections mostly of viral and bacterial origin. Its other causes can be allergy, trauma, toxins and neoplasma. Pharyngitis can be caused by a variety of microorganisms.
Viral pharingitis is often be caused by the so called *cold viruses, flu (influenza virus), adenoviruses, mononucleosis, HIV, etc.*
Bacterial pharyngitis is often caused by *Group A streptococcus* and sometimes by *Corynebacterium, Arcanobacterium, Neisseria gonorrhoeae, Chlamydia pneumoniae*, and

others. Most bacterial and viral cases of pharyngitis are clinically indistinguishable, they can however, show certain characteristics, f.i.:
A *beta-hemolytic streptococcal (GABHS)* infection occurs mostly among children aged 4-7 years. A **sudden onset** is consistent with group A beta-hemolytic streptococcal infections. Pharyngitis following several days of coughing or rhinorrhea is more consistent with a viral etiology.
Headache is consistent with *GABHS* and *mycoplasma infections*. **Cough** is usually not associated with GABHS infections.
Vomiting is often associated with *GABHS* infections but may be present in other types of pharyngitis as well.
A patient's history telling about recent **orogenital contact** suggests the possibility of the presence of *gonococcal* pharyngitis.
A patient's history telling about rheumatic fever is important regarding his treatment.
Fever is usually absent or low-grade in case of viral pharyngitis, but this is not specific enough to differentiate viral and bacterial etiologies. Fever can be about 38-40 C in case of infections caused by *Coxsackie virus A, Coxsackie virus B, Herpes Simplex Virus, GABHS, HIV-1, EBV and CMV.*
Concomitant symptoms include **conjunctivitis**, may be seen in association with *adenoviruses,* while **scleral icterus** in case of *infectious mononucleosis.*
Rhinorrhea has usually a *viral* cause.
Tonsillopharyngeal/palatal petechiae are seen in case of *GABHS* infections and infectious *mononucleosis.*
A tonsillopharyngeal exudate may be experienced in case of *streptococcal* infections, *mononucleosis* and occasionally in case of *Mycoplasma pneumoniae, Chlamydia pneumoniae, Arcanobacterium haemolyticum, adenoviral and herpes viral* infections. Exudates do thus not differentiate viral and bacterial causes.
Oropharyngeal vesicular lesions are seen in case of *Coxsackie viral* and *herpes viral* infections. Concomitant vesicles on hands and feet are associated with Coxsackie virus (hand-foot-and-mouth disease).
Lymphadenopathy, i.e. tender anterior cervical lymphnodes are consistent with *streptococcal* infections, while generalized adenopathy is present with *infectious mononucleosis* and with an acute lymphoglandular syndrome of *HIV* infection.
Cardiovascular: Murmurs should be documented in case of an acute episode of pharyngitis to monitor for potential rheumatic fever.
Pulmonary: Pharyngitis and lower respiratory tract infections are more consistent with *Mycoplasma pneumoniae* and *Chlamydia pneumoniae*, particularly if a persistent nonproductive cough is present.
Hepatosplenomegaly can be found in case of an *infectious mononucleosis.*
Group C, G, and F streptococcal infections may be clinically indistinguishable from GABHS infections but do not cause the immunologic sequelae of GABHS infections. They may be associated with food-borne outbreaks. Group C streptococci is said to cause meningitis, endocarditis and subdural empyemas as well.
Arcanobacterium (former Corynebacterium) haemolyticum infections are more common among young adults and are very similar to GABHS infections, including the similar scarlatiniform **rash.** Patients are often coughing. Occasional outbreaks have been reported.
Mycoplasma pneumoniae affecting young adults causes headache, pharyngitis and lower respiratory symptoms. Approximately 75% of the patients have a cough, not present in case of ABHS infections.
Chlamydia pneumoniae has a clinical picture similar to that of Mycoplasma pneumoniae. Pharyngitis usually precedes pulmonary infection by about 1-3 weeks.

Neisseria gonorrhoeae is a rare cause of pharyngitis. A careful patient's history is important since this infection usually follows an orogenital contact. It may be associated with urethritis and a severe systemic infection as well.
Corynebacterium diphtheriae can occur in areas, where vaccination is not obligatorily prescribed. Its foul smelling, gray-white pharyngeal membrane may result in airway obstruction.
Unusual bacteria which could be present with pharyngitis include *Borrelia B.s.l., Francisella tularensis, Yersinia species* and *Corynebacterium ulcerans.*
Adenovirus: The distinguishing feature of an adenoviral infection is conjunctivitis associated with pharyngitis (pharyngoconjunctival fever). It is commonly occuring among children under 3 years.
Herpes Simplex Virus: Vesicular lesions (herpangina), present especially among young children, while concerning older patients, its pharyngitis may be indistinguishable from GABHS infection.
Coxsackie viruses A and B: These infections can have symptoms similar to those of herpes simplex and may develop vesicles as well. If the enanthems are whitish and nodular, the symptom is named lymphonodular pharyngitis. Coxsackie A16 may cause Hand-foot-and-mouth disease, which causes oropharyngeal ulcers of 4 to 8-mm as well as vesicles on hands and feet, and, occasionally, on the buttocks. The oropharyngeal ulcers and vesicles cease within one week.
Epstein-Barr Virus (EBV): Clinically known as infectious mononucleosis, is extremely difficult to be distinguished from GABHS infection. Exudative pharyngitis is prominent. Distinctive features include retrocervical or generalized lymphadenopathy and hepatosplenomegaly. Atypical lymphocytes can be seen on peripheral blood smears. *CMV:* The symptoms of CMV infection are similar to those of infectious mononucleosis. It concerns sexually active patients, causes high fever and significant malaise. Pharyngitis is not its characteristic complaint.
HIV-1: can be associated with pharyngeal edema and erythema, aphthous ulcers, with no exudates. Fever, myalgia, and lymphadenopathy are also present.
Diagnosis: symptomatically. By GABHS rapid antigen detection tests, by bacterial culturing, by complete blood count, erythrocyte sedimentation rates, by x-ray. By serology, PCR etc.
Treatment: symptomatically, by administering antibiotics in order to decrease the duration of the illness and the infective period, to provide symptomatic relief and to decrease the incidence of relapses and complications (f.i. rheumatic fever). Antifungal and antiviral drugs are used in certain cases.
RFR method: detects and may eliminate the pathogen microorganisms!
In case of bacterial pharyngitis the RFR method is supporting the antibiotics. In case of viral pharyngitis RFR is the main therapy.
As to the resonance frequencies of the bacteria see their special Chapters.
As to the frequencies of Arcanobacterium and other
As to the resonant frequencies of viruses
The RFR method of viral infections should be used according to the lifecycle of the causative virus.

22.6. Bell's Palsy

Bell's palsy is a disease of the seventh cranial nerve, characterised by a sudden weakness or paralysis of the related innervated muscles of the face, on one or two of its
10.38.).
The path of the facial nerve is very complicated, which may be the reason of its vulnerability to injuries. The two bundles of the facial nerve leave the brain at the

cerebellopontine angle, traverse the posterior cranial fossa, dive into the internal acoustic meatus, pass through the facial canal in the temporal bone, then turn sharply backwards, pass behind the middle ear and exit the cranium at the stylomastoid foramen. From here, the facial nerve bisects the parotid gland, their terminal branches burst out from the parotid plexus innervating the muscles forming the facial expressions. Inflammatory, demyelinating, ischemic and compressive processes may impair the neural conduction at the stylomastoid foramen, which is a unique anatomic locus.

The etiology of Bell's palsy remains unclear, though it may be influenced by vascular, infectious, genetic and immunologic factors. Patients with other diseases sometimes suffer from peripheral facial nerve palsy, too.

Its dominant cause seems to be a viral infection triggering a non-adequate immunological response, resulting in damages to the facial nerve. Pathogens causing such infections are mostly the HSV1; HSV2; other HHVs; VZV; EBV, CMV, HIV, Influenza B virus; Adenoviruses; Coxsackie viruses; Hepatitis A, B and C viruses; ECHO viruses; the rubella virus, Mycoplasma pneumoniae; Mycoplasma fermentans and Borrelia B. sensu lato.

Bell's palsy may develop in association with polyneuritis, acute and chronic otitis, infectious mononucleosis, Herpes Zoster infection, HIV infection, temporal bone fracture, facial trauma, parotid tumors, leukemic meningitis, leprosy, sarcoidosis, cholesteatoma, aneurysm, cerebral tumor, Melkersson-Rosenthal syndrome, middle ear surgery and osteomyelitis.

Diagnosis: symptomatically, by complex laboratory, neurological and ophthalmic examinations.

Treatment: depending on its cause, by administering antiviral drugs, corticosteroids, begun at once if the orgin of the illness is unknown, by eye care and symptomatically.

RFR method: should be used immediately, at the very start of the inflammatory process of the facial nerve, until no irreversible damages do develop.

The most frequent resonances of Herpes Simplex Virus-1 are: 290-294, 344-346 kHz

The most frequent resonances of Herpes Simplex Virus-2 are: 352-365, 413, 425 kHz

The most frequent resonances of Herpes Zoster Virus are: 416-421 kHz

The most frequent resonances of HIVs virus are: 349, 365, 424, 460 kHz

The most frequent resonances of Coxsackie virus are: 287-291, 294-303 kHz

The most frequent resonances of Cytomegalovirus are: 305-306, 345-350, 406-412, 530-536 kHz

The most frequent resonances of Epstein-Barr Virus are: 337-340, 342-347, 370-384, 397, 422, 438, 518 kHz

The most frequent resonances of ECHO viruses are: 316-319, 395-405, 471 kHz

The most frequent resonances of Rubella virus are: 372, 402, 440, 450-451, 468, 520-530 kHz

The most frequent resonances of Mycoplasma pneumoniae are: 321-324 kHz

The most frequent resonances of Mycoplasma fermentans are: 442-444, 447-451, 493-495 kHz

The most frequent resonances of Borrelia Burgdorferi sensu lato are: 378-382, the frequencies of its antibiotic-resistant species are: 382-390 kHz

As to the frequencies of other causative microorganisms see their special Chapters.

23. IMMUNE DISORDERS

Immune disorders are diverse conditions in which the immune system doesn't function adequately, the immune response is not effective, or not specific enough, so that among the patients infections occur and recur more frequently, are unusually severe and last longer than usual. These disorders can be manifested by way of allergic processes, various immunodeficient response mechanisms and autoimmune reactions as well.
Immunodeficient states may come about already at the birth of the persons, which states are the connatal immunodeficiency disorders, or may develop later on. Immunodeficiency occuring later in life is termed acquired immunodeficiency and is usually caused by viral infections. The acquired immunodeficiency disorders are much more common than congenital ones. Some infections cause only minor impairments of the immune system, while others may damage and even destroy the body's ability to fight infections. *HIV* infection f.i. can result in an acquired immunodeficiency syndrome (AIDS). The virus attacks and destroys the T helper white blood cells, which normally concer viral and fungal infections. Many other different factors can impair a person's immune system.
Certain viruses, bacteria and fungi f.i. *Chickenpox, Smallpox, Cytomegalovirus, German measles, Epstein-Barr Virus, Measles virus, Tuberculosis*, severe other bacterial and fungal infections can cause immunodeficiency for a short or longer time. The human pathogen members of the *Mycoplasma genus* cause a special immunodeficiency. Immunodeficiency is the cause of some hematologic diseases and malformations, f.i. of agranulocytosis, aplastic anemia, leukemia, lymphoma, myelofibrosis, myeloma multiplex, histiocytosis, all cancer, which are all consequences of viral infections. Immunodeficiency can be caused by a stronger worm infection as well.
An impaired immunity can result in severe, persistent, recurring and complicated bacterial infections, f.i. sinus infections, chronic ear infections and chronic bronchitis which all follow the sore throat and head cold of infants. In case of immunodeficiency a bronchitis can progress to pneumonia, the skin and the mucous membranes lining the mouth, eyes and genitalia are all susceptible to infection. Thrush, a fungal infection causing ulcers and inflammation of the mouth is often experienced in case of impaired immunity. Chronic mucocutaneous candidiasis can develop and persist due to the poor functioning white blood cells of infants and adults. The fungus can cause infections of the mucous membranes, as well as the scalp, skin and nails. A lot of women develop candidiasis in vagina.
Conjunctivitis and other inflammations of the eyes, hair loss, eczematous exanthems and plaques of enlarged, broken capillaries under the skin can also be signs of an immunodeficiency syndrome. Infections in the gastrointestinal tract can cause diarrhea, extreme wind colic and weight loss and in absence of the friendly bacterial bowel flora irritable bowel syndrome and Crohn's disease may develop.
Chronic fatigue syndrome (CFS) is usually associated with immunodeficiency. It is characterized by debilitating fatigue, often accompanied with inability to concentrate, a low grade fever and the swelling of the lymph nodes.
Autoimmune disorders, including failure of the adrenal glands, Addison's disease, Immune thyroiditis, Rheumatoid Arthritis, SLE, all develop due to immunedeficiency. Tendency to get diarrhea is common, and food may not be well absorbed from the gastrointestinal tract.
Immunodeficiency disorders not caused in a hereditary and metabolic way can occur due to chemicals and treatments suppressing the immune system (f.i. corticosteroids, surgery) trauma, alcohol, etc.

The most common types of congenital/connatal immunodeficiency disorders are:

X-linked agammaglobulinemia
Selective antibody deficiency

Common variable immunodeficiency
Severe combined immunodeficiency
Wiskott-Aldrich syndrome
Ataxia telangiectasia
Hyper-IgE syndrome
Chronic granulomatous disease
Transient hypogammaglobulinemia of infancy
DiGeorge anomaly
Chronic mucocutaneous candidiasis

23.1. Immune Deficiency Syndromes in General

Immunodeficiency syndromes, wether congenital, acquired due to infections or in a iatrogenic way, are characterized by an unusual susceptibility to infections and sometimes, to autoimmune diseases and lymphoreticular malignancies as well. The type of the infection often provides the first clue concerning the nature of the present immunological defect. Immunological functions are mediated by two developmentally divergent, but functionally interacting families of the lymphocytes. The activities of these B and T-lymphocytes and of their products are closely integrated in the host's defense system together with the functions of other cells of the reticuloendothelial system. Fixed macrophages and wandering monocytes play an important role in the trapping and processing of foreign antigens, they will become effector cells, activated by lymphokin products of T-lymphocytes. The antimicrobial activity of the polymorphonuclear leucocytes is directed and made specific by antibodies in concert with the products of the complement system. The interaction of basophils and tissue mast cells works together with IgE antibodies in the causing of immediate hypersensivity.

Moderate doses of an immunogen initiate usually an immune response, whereas an excessive dose will be followed by a state of tolerance and further on by a specific nonreactivity to ahy of the subsequent doses of this immunogen, though unrelated immunogens in appropriate doses will provoke immune responses. Tolerance can be brought about more easily in case of neonates than in case of adults, is maintained by the persistence of the immunogen, and can be manifested by the impairment of humoral or cellular immunity.

The mechanisms of tolerance are diverse and may involve the deletion of a clone of antigen-committed immunocompetent cells, which may occur if a large amount of antigens or antigen-antibody complexes bypasses the macrophage and interacts in a deleterious way directly with the lymphocyte provoking the production of active suppressive T cells. This can be shown, if the cells of a tolerant person is transplanted to a normal one, which cells will not allow a specific normal immune response to develop. In case of transplantation and tumors the presence of blocking antibodies leads to the inactivation of the immunocompetent lymphocytes developing owing to antigen-antibody complexes and/or to the direct, antibody- caused blockade of the cellular antigens.

It is well known that different viruses, bacteria, Mycoplasma species and other agents may cause a reduced natural immunoreactivity. Diminished immune response can often be experienced several years before the development of a severe chronic disease.

The immonological response is controlled by many a regulatory factor. Interleukin-2 (IL-2) is the most important of them. It can be produced by most of the CD4+helper/inducer cells, by a smaller population of CD8+suppressor/cytotoxic cells, and by special NK cells. Effected by IL-2, cytotoxic T cells, B lymphocytes, NKs, lymphokines-activated killers and a considerable part of monocytes will multiply and be differentiated. The immune response is regulated by the interaction of lymphocytes communicating by way of the release of soluble mediators, the interleukins. IL-2 amplifies the effector phase of immunity.

The IL-2 production of normal human lymphocytes is regulated by monocytes, PGE2 and polyamine levels. A monocytic cell-line product inhibits the IL-2 production. The reaction of polyamine oxidase with poliamines yields H_2O_2 mediating IL-2 suppression. Immunosuppressive PGE2 inhibits the mitosis of T lympocytes, the production of lymphokines, the IL-2 synthesis, the ADCC and NK activity, the immunoglobulin production of B cells, the expression of HLA-DR antigens and the antigen presentation of macrophages. It is also indirectly immunosuppressive as it increases the activity of T suppressor cells.

An immune deficiency disease cannot be healed by immunostimulant medications. Nowadays, immunological researches focus on the regulating mechanisms of the immune response. The effect of IL-2 treatment is limited by downregulation, so that the efficiency of the therapy could be increased by substances inhibiting the downregulation.

Immune deficiency diseases can be cured by eliminating the pathogen microorganisms, such as *Mycoplasma, Chlamydia, EBV, Human T-cell Lymphotropic Viruses and Human B-cell Lymphotropic Viruses* (see the special Chapters).

The autoimmune types of the immune deficiency syndromes can be healed by eliminating microorganisms stimulating autoimmunity, such as *Chlamydia and Borrelia Burgdorferi sensu lato species, Human T-cell Lymphotropic Virus-1,* and others (see the special Chapters).

After eliminating the pathogen agent the autoimmune process should be stopped by administering corticosteroids.

23.2. Connatal Immune Deficiency Syndromes

23.2.1. Severe Combined Immunodeficiency (SCID)

Severe combined immunodeficiency syndrome is the most serious among the immunodeficiency disorders and is characterized by a significant functional impairment of the humoral and cell-mediated immunity. In case of this syndrome, the B lymphocytes and the antibodies are deficient, the T-lymphocytes are deficient or nonfunctioning as well and therefore unable to fight infections effectively. Several different defects of the immune system can result in the severe combined immunodeficiency disease, including the deficiency of the enzyme adenosine deaminase. Reticular dysgenesis, associated with aleukocytosis, is a very rare form of SCID. Most infants suffering from a severe combined immunodeficiency get pneumonia, thrush, diarrhea and other chronic infections. This illness is usually congenital, may be inherited either as an X-linked or an autosomal recessive-linked defect, though it may occur sporadically as well. Affected infants rarely survive longer than one year. This syndrome is associated with a diversity of developmental defects of immunocompetent cells, some of which are related to specific enzymatic abnormalities. Swiss-type agammaglobulinemia, the classic example of SCID, a disorder inherited in an autosomal recessive way is characterized by severe lymphopenia, involving both T and B cells. SCID patients are secondarily infected with some viruses, fungi and bacteria. In case of a healthy person the immunogens initiate the host's immune responses f.i. the cellular processing of the immunogen materials; the producing of effective substancies, the controlling of mechanisms and consequences both beneficial and detrimental ones, etc. SCID patients have no immune response, they need continous clinical control. Unfortunately, antibiotic treatments often lead to generalized fungal infections among SCID patients.

If a pregnant woman suffers from a viral infection inhibiting the development of the immune system of her embryo, other hemopoetic cell lines may also fail to develop in her embryo.

Some children with autosomal recessive SCID are **deficient in adenosine deaminase** (ADA), an enzyme involved in the nucleic acid metabolism. In case of this enzyme deficiency substances toxic to immature lymphocytes will accumulate in the infant leading to a severely compromised or completely lacking immune system.
Diagnosis: by difficult laboratory test systems and symptomatically.
Treatment: by transplantation of histocompatible bone marrow, by umbilical cord blood transplantation from sibling donors. Antibiotics, IVIG and IL-2 therapy are helpful but do not cure.
RFR method: The cause of SCID syndrome can be a congenital HTLV or HBLV or/and mycoplasmal infection got via the placenta. The eliminating of these viruses present in a woman before her pregnancy can be the only protection against the development of SCID syndrome in her fetus.
The infant needs a continuous control! The pathogen resonances should be detected and the pathogens should be eliminated by their resonances.
The friendly bacterial flora plays an important role and must be continuously controlled.
Human T-cell Lymphotropic Viruses and Mycoplasma species are the most frequent pathogens of this syndrome.
The resonant frequencies of HTLV-1 are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz
The resonant frequencies of HTLV-2 are: 314, 320-324, 370-376, 493-501 kHz
The resonant frequencies of HTLV-3 are: 307, 312, 320-324, 338-340, 365-367, 397-400, 416, 428, 435, 453-455, 484, 526-530 kHz
The resonant frequencies of HTLV-4 are: 297, 454, 540-545 kHz
The resonant frequencies of HTLV-5 are: 297-298, 315, 320-340, 354, 439, 480-482, 523, 544-545 kHz
The resonant frequencies of HTLV-6 are: 359, 374-376, 382-383, 474-476, 570-578 kHz
The resonant frequencies of Mycoplasma fermentans are: 312-315, 329-331, 352-355, 361, 371, 404, 442-451, 493-495, 518, 520-524 kHz
The resonant frequencies of Mycoplasma pneumoniae and M. pulmonis are: 306-309, 321-324, 337-344, 346-353, 369, 397, 499 kHz
The resonant frequencies of other Mycoplasma species are: 339, 345-350, 363, 367, 377, 398-400, 404, 410, 424, 442, 470, 494-496, 534-535, 546-550 kHz
The summarized most frequently found resonances of SCID are: 297-299, 311-315, 321, 324, 330, 339-341, 365, 370-374, 382, 397, 408-411, 416, 428, 432-433, 442-452, 453-455, 482-483, 487-490, 493-497, 518-519, 523, 526-530, 568, 574 kHz

23.2.2. Wiskott-Aldrich Syndrome

Wiskott-Aldrich syndrome is an X-linked genetically determinated disease affecting only boys and causing skin eczema, thrombocytopenia, and a combined cellular and humoral immunodeficiency, with deficient B and T lymphocytes leading to repeated infections. These patients have an increased risk for getting autoimmune diseases and hematologic malignancies.
Genetic predisposition: Several mutations in the *WASP* gene, consisting of 12 exons and encoding 502 amino acids, are identified in patients with Wiskott-Aldrich syndrome. WASP gene is an important transcription factor of lymphocyte and platelet functions. (Mutations of this *WASP* gene may be responsible for X-linked thrombocytopenia and myelodysplasia as well.) The Wiskott-Aldrich syndrome protein (WASp) is a key-regulator of the actin polymerization in hematopoietic cells. The expression of this gene in dendritic cells regulates the ability to traffic to secondary lymphoid organs and to activate naive T cells in the lymph nodes. WASp mutations cause a severe *immunodeficiency*

characterized by a defective initiation of the primary immune response and the *autoimmunity*.
CD43, a sialylated glycoprotein, which is the component of the T-cell activation pathway binding the intercellular adhesion molecule-1 (ICAM-1), is not correctly expressed by lymphocytes and platelets in WAS patients. The antibody response to polysaccharides and the cellular immunity is defective in this disease, the serum concentration of IgM being decreased, while that of IgA and IgG is normal. The immune system of these patients is unable to produce antibodies against polysaccharide antigens, though their responses to protein antigens are normal. The Wiscott-Aldrich syndrome possibly reflects the primary defect of B lymphocytes.
This lymphocyte deficiency makes the affected children susceptible to infections caused by antibiotics dependent or antibiotics resistant *Staphylococcus aureus* bacteria, *Gram-negative bacilli,* viruses such as *HTLV 1-6, HBLV, HSV* and *Mycoplasma, f.i. M. fermentans* and *fungi as well.* There often occur serious infections caused by encapsulated pathogens causing life-threatening complications, including pneumonia, respiratory tract abscesses, meningitis and sepsis. Infections with Pneumocystis carinii and several different *secondary viral infections* can develop and cause trouble.
Symptoms: In Wiskott-Aldrich syndrome thrombocytopenia and platelet dysfunction are often present already at birth, so that the affected boys often bleed and bloody diarrhea may be the first symptom during the first weeks or months of their life. Hematuria, epistaxis and cutaneous petechiae, eczema like lesions and purpura can be experienced. *Recurrent bacterial, mycoplasmal and viral* infections begin in infancy after the placentally transmitted maternal antibody levels get diminished. Superficial and deep skin infections such as impetigo, cellulitis, furuncles, abscesses are common. Otitis media, sinusitis and mucous membrane damages such as sinonasal infections, pharyngitis, thrush, etc can also come about.
IgE mediated asthma, bronchial or pulmonary infections, neurological symptoms, f.i. meningitis, CNS lymphoma, intracranial bleeding can develop. The signs of a possible malignancy, f.i. adenopathy and hepatosplenomegaly can sometimes be observed. Hemolytic anemia, nephritic syndrome can also occur among affected patients whether treated or not with transfer factor.
These infants rarely survive their childhood, the cause of their death being usually infection, bleeding, or being associated with developed malignancies, primarily lymphoreticular tumors and leukemia.
Diagnosis: by immunological testing, by culturing bacteria and fungi, by serology of Mycoplasma and viruses. By WASP gene examinations.
Differential diagnosis: by distinguishing it from other immunodeficiency disorders.
Treatment: symptomatically by administering antibiotics and IVIG, though bone marrow transplantation offers better results.
RFR method: detects and can eliminate the pathogen microorganisms!
The most frequent resonances are: 291-294, 344-345, 358-361, 365, 370-381, 440-451, 493-497, 526-530, 574 kHz
Having measured children with Wiscott-Aldrich syndrome I have found many a resonant frequency.

23.2.3. Hyper-IgE Syndrome (Job-Buckley Syndrome)

Hyper-IgE syndrome (HIES, named also Job-Bucley syndrome) is an immunodeficiency disorder characterized by very high levels of IgE antibodies in the blood.
HIES affects many an organ, including the skeleton, the connective tissue and the dentition. It is a primary immunodeficiency of infectious-allergic etiology, mostly with an autosomal dominant inheritance pattern. Autosomal dominant-HIES (AD-HIES) can be inherited with various expressivities, the possibly affected locus being in chromosome arm

4q. In contrast, patients with autosomal recessive-HIES (AR-HIES) are free of a skeletal or dental involvement and do not develop cystic lung diseases, but are susceptible to viral infections, such as a severe Molluscum contagiosum infection and can develop severe neurological complications. In some studies it is supposed that this disease can be linked to mutations in the STAT3 gene, though the exact etiology is not yet clear.
Besides the immune defects affecting the IgE synthesis, defects of cell-mediated immunity with decreased Th1 responses have also been reported. These defects are a decreased or absent delayed-type hypersensitivity in some patients with HIES and a decreased lymphoproliferative response to *HTLV,* Staphylococcus aureus, Candida *species, Mycoplasma species* (such as *M. pneumoniae, M. fermentans, M. penetrans*) and rarely to tetanus antigens as well. There exist some reported data concerning the decrease of the CD8+ T cells and of the CD45RO+ memory T cells. An abnormal neutrophil chemotaxis due to a decreased production of interferon gamma is also thought to cause some symptoms.
The symptoms of this heterogeneous group of HIES patients are usually characterized by recurrent infections, unusual eczema-like skin rashes, severe lung infections resulting sometimes in pneumatoceles, abscesses and allergy, causing f.i. eczema, nasal stuffiness and asthma, as well. The recurrent infections can affect the skin, the lungs, the joints and other organs. Pulmonary pneumatoceles are thin-walled, air-filled cysts developing within the lung parenchyma, can be single emphysematous lesions and multiple, thin-walled, air-filled, cystlike cavities. They occur most often as sequelae of an acute pneumonia, commonly caused by *Staphylococcus aureus*. Pneumatocele formation, however, can occur also caused by other pathogen agents including *Adenovirus, Coxsackie virus, Streptococcus pneumoniae, Haemophilus influenzae, Escherichia coli group, Pseudomonas aeruginosa, A streptococci, Serratia marcescens, Klebsiella pneumoniae, Mycoplasma fermentans, Aspergillus species, HTLV* and *Mycobacterium tuberculosis*. Pneumatoceles can generally be observed on the chest radiograph soon after the development of pneumonia. This infection is the major cause of morbidity; approximately 80% of patients have pneumatoceles secondary to pneumonia, and a same percentage of patients have chronic mucocutaneous and ungual candidiasis.
Patients having an autosomal dominant form of the disease have weak bones getting often broken, and have sometimes, by not loosing their primary teeth, two sets of teeth simultaneously.
Infant patients with AR-HIES have frequently complications caused by *VZV* and *HSVs*. While the mortality caused by acute pneumonia or/and CNS infections of HIES patients is significantly high, the mortality associated with pneumatoceles is low.
Diagnosis: symptomatically, by making immunodeficiency tests and by measuring the IgE levels, which, as the hallmark of this illness, are usually 10 times higher than normal. Eosinophilia can also be often found.
Treatment: by continually or intermittently administering antibiotics in order to cure staphylococcal and/or other infections. By giving high-dose iv. gamma-globulin preparations. A profilactic skin care is advisable.
RFR method: detects and can eliminate the pathogen microorganisms!
The first step to take is to eliminate the Mycoplasma species. Use RFR method combined with antibiotics! Antibiotic resistant staphylococci are often to be found in these cases, but the RFR method is effective also against them.
The most frequent resonances are: 287-302, 307-308, 313-324, 326-327, 331-333, 337, 446-450, 352-363, 370-374, 376-381, 393, 395-405, 409-410, 416-420, 432, 442-451, 485-490, 493-495 kHz

23.2.4. DiGeorge Syndrome

DiGeorge syndrome (named also 22q11 deletion syndrome, congenital thymic hypoplasia, or third and fourth pharyngeal pouch syndrome) occurs due to abnormal fetal developments. This condition of the fetus develops usually caused by a viral infection got during the pregnancy of its mother. Newborn infants have abnormal thymus glands and T lymphocyte functions. Their lymphocyte counts may be normal, but virtually all lymphocytes are B cells. The specific antibody responses of affected patients are usually impaired even with normal concentrations of immunoglobulins. (Nezelof syndrome is a similar, but in an autosomal recessive way inherited congenital immunodeficient condition, a form of thymic dysplasia).
Infants with DiGeorge syndrome usually have congenital cardiac defects, particularly involving the great vessels, hypocalcemic tetani due to the failures of the parathyroid development, and the absence of the thymus. Other associated abnormalities can include abnormal ears, shortened philtrum, and hypertelorism. Having no parathyroid glands, their blood calcium levels are low and they often develop seizures shortly after birth. In time of the pregnancy when infect the virus DiGeorge syndrome could be prevented by healing the viral infection before its mother's pregnancy. Already developed syndromes can not be healed.
Diagnosis: symptomatically and by complex examinations
Treatment: by transplants of fetal thymus. Symptomatically
RFR method: detected HTLV infections should be treated before the planned pregnancy!
The most frequent resonances are: 291, 312-318, 324, 328, 349, 359-365, 371-374, 382, 397, 424, 428-432, 454, 493, 545 kHz

23.2.5. Omenn Syndrome

Omenn syndrome is a hereditary disorder in an autosomal recessive manner and is characterized by a severe immunodeficient state. The expansion of an oligoclonal population of *HTLV* infected and antigen-stimulated T helper2 cells results in the production of elevated levels of cytokins IL-4 and IL-5. The latter cytokin is leading to cytokines mediated eosinophilia and elevated IgE levels.
The *HTLV* and *mycoplasma* infected T cells, predominantly of Th2 type will presumably secrete cytokines promoting autoimmune and allergic inflammations. Omenn syndrome is identified as leaky SCID syndrome caused by hypomorphic mutations in the recombinase genes RAG-1 and RAG-2, which impair but do not eliminate the recombination of the VDJ segments of TCR and Ig genes. The inability to productively rearrange the VDJ regions of the T and B cell receptors results in abnormal T cells and absent B cells. The mutations in the genes RAG-1 and RAG-2 in case of Omenn syndrome differ from the T-cell negative, B-cell negative and Natural Killer Cell positive SCID form also caused by RAG-1 or RAG-2 mutations.
Symptoms: are characterized by erythrodermia, desquamation, alopecia, chronic diarrhea, failure to thrive, lymphadenopathy and hepatosplenomegaly. Patients have several different life-threatening bacterial *(Staphylococcus aureus)*, viral and fungal infections. *Pneumocystis carinii* pneumonia and *poliomyelitis* due to attenuated oral poliovirus vaccines are characteristic infections occuring in Omenn syndrome and in SCID as well.
If untreated, Omenn syndrome is fatal. Patients often have *Staphylococcus aureus* sepsis, related to the generalized dermatitis experienced. Chronic diarrhea resulting inanition and live viral infections may be responsible for their dying.
Diagnosis: by examining hypomorphic mutations in RAG-1/RAG-2 genes and symptomatically.
Treatment: symptomatically by administering antibiotics.
Bone marrow transplantation is usually successful, though a life-threatening acute or chronic graft versus host disease might be its complication.
RFR method: detects and can eliminate all pathogen microorganisms present.

The most frequent resonances are: 348, 372, 376-387, 405-410, 416, 442-451 kHz
As to other often present frequencies, see Chapter of SCID.

23.3. HIV and AIDS

All viruses which are able to infect lymphocytes have an important role in the development of an AIDS syndrome. These viruses cause a false response of the host's immune system. A number of viruses are known to infect human lymphocytes, such as *EBV, Human Herpes Virus-8 (HHV-8), CMV* and retroviruses. Human pathogenic retroviruses are the *Human T-cell Lymphotropic Viruses* i.e. *HTLV-1, HTLV-2, HTLV-3, HTLV-4*, and the human immunodeficiency viruses such as *HIV-1 (*formerly known as HTLV-3*), and HIV-2,* among which HTLV-1, HTLV-2, HIV-1, and HIV-2 are causing epidemics as well. HTLVs can be distinguished from HIVs by their potency to cause lymphoproliferative disorders.

HIV is a Lentivirus, a subgroup of retroviruses. The infections of this family of viruses are characteristically responsible for illnesses of long-duration, with a long incubation period, by causing persistent viremia, can infect the nervous system and weaken the host' immune responses. HIV has high affinity to CD4+ T lymphocytes and monocytes. Binding itself to a CD4+ cell HIV gets internalized and replicates itself by generating a DNA copy by a virally encoded reverse transcriptase present in the virus particle. Thus the viral DNA becomes incorporated into the host's DNA enabling further replications. This incorporated virus can either become latent and the infected cell continues its functioning, or the virus becomes active and replicates, and its liberated particles will infect other cells. The HIV infected vital cells i.e. the CD4+ T cells, the macrophages-monocytes and the dendritic cells will die and get a low level due to the increased rate of apoptosis and to their being killed by the viruses and by the CD8+ cytotoxic lymphocytes, which recognize the infected cells. If the $CD4^+$, T cells in the blood are below a critical number the cell-mediated immunity does not work any more and the body becomes progressively more susceptible to opportunistic infections. If untreated, most HIV-infected individuals develop AIDS and die. Since the introduction of the highly active antiretroviral therapy and the prophylaxis against opportunistic pathogens, the death rates of AIDS patients decline significantly.

The pathogenicity of HIV-1 originating from wild chimpanzees overspread to human beings in southern Cameroon in the twentieth century. This more virulent and easier transmittable HIV-1 causes the majority of the HIV infections. The previous names of HIV-1 were HTLV-III, lymphadenopathy-associated virus (LAV) and AIDS-associated retrovirus (ARV). The less transmittable HIV-2 is said to originate from Sooty Mangabey, an Old World monkey of Guinea-Bissau, Gabon, and Cameroon is solely confined to West Africa.

HIV can be transmitted primarily by sexual intercourse via the contact of the mucous membranes (mouth, vagina, penis, rectum) with contaminated body fluids, its parenteral transmission occurs mostly among intravenous drug users via contaminated blood, or, rarely, by contaminated blood products. The virus can be transmitted from an infected mother to a child before or during its birth or by its mother's milk. These transmissions can occur through the placenta or via the birth canal. HIV infection is pandemic among people.

The HIV virus first attaches itself to the CD4 molecule and penetrates its target cell. The HIV-RNA i.e. the genetic code of the virus, is released into the cell. To be reproduced, the RNA must be converted into DNA. The enzyme performingthe conversion of the viral RNA into DNA is named reverse transcriptase. The viral DNA then enters the nucleus of the infected cell. With the help of an enzyme named integrase, the viral DNA becomes integrated with the DNA ofthe cell. The DNA now replicates and reproduces RNA and proteins. The proteins in form of a long chain must be cut into pieces after the virus leaves the cell. A new virus and short pieces of proteins will be produced by this process. The virus buds through the cell membrane of the cell, wrapping itself in a fragment of the cell

membrane. In order to become infectious for other cells, another viral enzyme, HIV protease, must cut structural proteins within the budded virus, causing them to be rearranged into the mature form of HIV.
According to definition, AIDS begins with a low CD4 positive lymphocyte count, less than 200 cells per microliter, or with the development of opportunistic infections and cancer, such as Kaposi's sarcoma and non-Hodgkin's lymphoma, cancer of the cervix and the rectum.
Symptoms: can be similar to those of an infectious mononucleosis, a few weeks after having first contracted HIV infection, f.i. fever, loss of weight, sweating in the night, dysphagia, maculopapular rashes, eosinophilic folliculitis, swollen lymph nodes and general discomfort can be experienced, which then disappear the enlarged lymph nodes excepted. Large amounts of viruses circulate in the blood and other body fluids, so that the person becomes contagious soon after becoming infected. Later on opportunistic infections, f.i. candidiasis, oral hairy leukoplakia, toxoplasmosis, mycoplasmosis, cryptosporidiosis, herpangina, infection with Pneumocystis carinii, CMV, EBV and mycobacterium avium can come to pass. The prognosis of these infections is bad. The HIV infection of the brain causes progressive multifocal leukoencephalopathy (PML), affecting the coordination and the balance. Sortness of breath, cough, chest pain, diarrhea, abdominal pain, vomiting, headache, dementia, depression, confusion and, dementia.
Characteristic and often occuring other symptoms of AIDS are: retina with cottage cheese and ketchup appearance, generalized adenopathy, meningismus hepatosplenomegaly, Kaposi's sarcoma, Molluscum contagiosum, secondary opportunistic infections such as Bacillary angiomatosis, Cryptosporidiosis, Pneumocystis carinii pneumonia, generalized herpes infection, generalised mycoplasmal infection (M. fermentans) and other intercurrent and opportunistic infections.
Diagnosis: symptomatically, by using laboratory tests HIV antibody detectation, (Western blot, ELISA), etc.
Treatment: by administering antiretroviral nucleoside reverse transcriptase inhibitors and protease inhibitors added to anti-HIV drugs known as fusion inhibitors in order to disrupt the structural rearrangement of the virus to fuse with healthy immune cells and to prevent HIV-replication. Drug combinations compared to single drugs can delay the onset of AIDS among HIV positive people and lengthten their life. The opportunistic infections need specific treatment! (f.i. P. carinii pneumonia nsuceeds Trimethoprim/sulfamethoxazole therapy, etc.)
RFR method: should only be used together with the conventional drug treatment! Detects and eliminates the HIV viruses, and the pathogens causing opportunistic infections!
The most frequent resonances of HIV-1 are: 312, 324, 340, 349-357, 362-367, 397-401, 416, 424-428, 454, 459-462, 487, 526 kHz
The most frequent resonances of HIV-2 are: 291-292, 296-298, 455, 462-465 kHz
The most frequent resonances of Mycoplasma fermentans are: 442-451, 491-495 kHz
The most frequent resonances of Human T-cell Lymphotropic Virus-1 are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz
The most frequent resonances of Human T-cell Lymphotropic Virus-2 are: 314, 320-324, 370-376, 493-501 kHz
The most frequent other resonances are: 296, 318-319, 349, 365-375, 383-384, 396, 400-403, 406, 424-428, 445, 458-478, 508-509, 534, 544, 556, 569 kHz
The most frequent resonances of AIDS Kaposi's sarcoma are: 428, 508 kHz, see the special cancer Chapter.
The most frequent resonances of Pneumocystis carinii are: 348, 379, 400-416 kHz
Infections caused by *Human T-cell Lymphotropic Viruses* (1-6), *Mycoplasma* and *Chlamydia species* all damage and decrease the effectivity of the person's immune system

The frequencies of other pathogens often attacking HIV patients see their special Chapters: f.i. *Candida* [redacted] *Toxoplasma* [redacted] *Mycoplasma* [redacted] *Cryptosporidium* [redacted] *Herpes Simplex Virus* [redacted] *Cytomegalovirus* [redacted] *Epstein-Barr Virus* [redacted] and *Mycobacterium* [redacted]
The optimal order to eliminate the found pathogens should be as follows: first mycoplasma, then herpes viruses, HTLV, HIV and others.
An additional advised therapy is the administering of vitamin C and IVIG.

23.4. Miscellaneous Immunodeficiency Syndromes

These immunodeficiency disorders form a group of diverse conditions in which the immune system doesn't function adequately, so that infections are more common, recur more frequently, are usually severe, and last longer than usual. Miscellaneous immunodeficiency syndromes include diseases with various degrees of immunodeficiency such as chronic hepatitis, SLE, diabetes mellitus, alcoholic cirrhosis, etc.
The **symptoms** of these syndromes include frequently occuring sinus infections, chronic ear infections, chronic bronchitis often progressing to pneumonia, etc. The skin and the mucous membranes lining the mouth, and the genitalia are susceptible to infections caused by fungi (f.i. thrush) or bacteria. Conjunctivitis, loss of hair, chronic eczema, spots of enlarged, broken capillaries under the skin are also signs of a possible immunodeficiency disorder. Infections of the gastrointestinal tract causing diarrhea, extreme gassiness and loss of weight are also characteristic. Opportunistic infections and more severe chronic infections can often come to pass among these patients. Autoimmune processes can easily develop and be associated with variable immunodeficiency forms.
Diagnosis: by immunological tests and laboratory examinations. By bacterial, viral and fungal culturing and analysis.(The disorders of the endocrine system can result in small testes, infertility and diabetes.)
Differential diagnosis: by distinguishing them from other immunodeficiency forms.
Treatment: symptomatically and according to the laboratory results.
RFR method: detects and eliminates the pathogen bacteria, viruses and fungi!
Mycoplasma species are the most frequent causative pathogens of these syndromes.
The most common frequency resonances of the members of this genus are:
The frequency resonances of Mycoplasma fermentans are: 312-315, 329-331, 352-355, 361, 371, 404, 442-451, 464, 491-495, 504, 520 kHz
The frequency resonances of Mycoplasma pneumoniae are: 321-324, 337-344, 346-350, 352, 363-364, 397, 499 kHz
The frequency resonances of other Mycoplasma species are: 293, 339, 367, 377, 398-400, 404, 410, 424, 442, 470, 496, 535, 546-551, 568 kHz and of the zoonotic Mycoplasma pulmonis are: 307-308 kHz

23.5. Chronic Fatigue Syndrome

Chronic fatigue syndrome (CFS) is an illness caused by chronic viral, bacterial, or fungal infections. In case of flu people can have a feeling of extreme exhaustion, tiredness and ache in the muscles, an inexplicable listlessness, that later proves to be the beinning of flu. In case of some chronic infections, such as *hepatitis, tuberculosis, brucellosis, infectious mononucleosis, candidiasis*, the presence of the infection may not be evident at once, but should always be suspected if the fatigue is out of proportion to other symptoms such as mood change, nervousness and anxiety.
The symptoms of this illness include debilitating fatigue, the loss of the ability to concentrate and, sometimes, a low-grade fever and the swelling of the lymph nodes. CFS often begins with an obvious infection persisting for several weeks after which it is

difficult to decide whether there does still exist a lingering infection or if the infection got complicated by a psychiatric illness. In case of many a disease f.i. *infectious hepatitis, brucellosis, infectious mononucleosis, herpes simplex viral, cytomegaloviral* and *HIV* infections there can co-existing long-standing neurotic symptoms be experienced. Abnormalites of the cell mediated immunity predispose to disseminated viral infections. Nevertheless it is difficult to dismiss an obscure secondary metabolic disorder consequent to the infection. Twice more women than men develop chronic fatigue syndrome. Anemia, moderate or severe, should be considered as a possible cause of an unexplained lassitude. Mild anemia usually goes without symptoms.
Every type of severe nutritional deficiency may cause lassitude and in its earlier stages this may be the chief complaint of the person. The loss of weight and the history of dietary inadequateness may provide the only other clues to the nature of the illness. Patients feel weak and tired after a myocardial infarct, and usually even suffer from depression. There are certain data concerning the role of the hypoactivity and the hyperserotonergic state of the hypothalamic-pituitary-adrenal axis and that of genetic predisposition in the pathogenesis of CFS.
Diagnosis: symptomatically and by examinations according to the viral, bacterial and fungal infection. Immunological tests can confirm the diagnosis of the Fatigue syndrome of infectious origin.
Differential diagnosis: by distinguishing it from connatal immunodeficiencies and immunodeficiencies caused by the side-effects of drugs.
Treatment: by specific antibacterial, antifungal and antiviral therapy [redacted] and, if necessary, by the compensation of biological trace elements.
RFR method: detects the pathogen microorganisms and eliminates them.
The most frequent resonances are: 290-293, 337-339, 342-347, 354-356, 365, 372-382, 384-388, 414-422, 430-438, 442-451, 474, 477, 487-490, 504, 516, 518, 544-555, 560, 576 kHz

23.6. Categories of Allergic Reactions

The immune system is the integral part of human protection against diseases. The mechanisms of the infected immune system can sometimes cause detrimental reactions of the host, i.e. hypersensitivity reactions, which can be characterized by their immunopathology. *Mycoplasma fermentans and/or Human T-cell Lymphotropic viral (HTLV-1) and Human B-cell Lymphotropic viral* infections influence and modify significantly the reactions of the immune system and provoke them to develop a hypersensitivity state.
The traditional classification for hypersensitivity reactions is that established by Gell and Coombs:
Reactions Type I (i.e. immediate hypersensitivity reactions) are characterized by immunoglobulin E (IgE)–mediated release of histamine and other mediators of mast cells and basophils.
Reactions Type II (i.e., cytotoxic hypersensitivity reactions) involve immunoglobulin G or immunoglobulin M antibodies bound to cell surface antigens, with subsequent complement fixation.
Reactions Type III (i.e. immune-complex reactions) involve circulating antigen-antibody immune complexes deposited in postcapillary venules, with subsequent complement fixation.
Reactions Type IV (i.e. delayed hypersensitivity reactions, cell-mediated immunity) are mediated by T cells rather than by antibodies.
Some authors believe this classification system to be too general and favor a more recently arranged classification proposed by Sell et al. This classification system sorts the immunopathologic responses into the following categories:

Inactivation/activation antibody reactions
Cytotoxic or cytolytic antibody reactions
Immune-complex reactions
Allergic reactions
T-cell cytotoxic reactions
Delayed hypersensitivity reactions
Granulomatous reactions
Multiple components of the immune system can be involved in various types of hypersensitivity reactions.
Certain bacterial or/and viral infections can modify the person's immune response significantly.
Infections caused by Mycoplasma fermentans and HTLV play a most important role concerning the influencing of the T lymphocyte functions (see the special Chapter) while they infect them. These infected T cells play an important role in the pathophysiology of allergic reactions.
Mycoplasma fermentans species are able to fuse with CD4+ T lymphocytes and change the characteristics of their cytokine production.
Mycoplasma infection can trigger the over-production of inflammatory cytokines (IL-1, IL-6 and TNFalfa) commonly experienced in case of CFS. The elevated level of these cytokines is held to be responsible for many a symptom of the chronic fatigue syndrome and fibromyalgia syndrome (CFS/FMS), including neurological involvements. They can have specific and nonspecific stimulatory or suppressive effects on lymphocytes, measured by B and T cell activation. In addition, the mycoplasma infection has immunomodulating effects, activating the hypothalmic-pituitary-adrenal axis. This can cause a cascade of limbic system symptoms characteristic of CFS/FMS.
Coxsackie viruses and parasitic antigens f.i. those of *ascaris* increase the effect of the immune response. These pathogens play an important role in the asthmatic processes.
Allergic reactions can clinically be manifested as anaphylaxis, allergic asthma, urticaria, angioedema, allergic rhinitis, some types of drug reactions and atopic dermatitis. These reactions are mediated by IgE, which differentiates them from anaphylactoid reactions that involve IgE-independent degranulations of mast cells and basophil cells. Allergens cause allergic reactions. Allergens are usually proteins, haptens or protein-combinations.
Haptens are inorganic antigens of low–molecular-weight not capable of eliciting an allergic response by themselves. They must be bound to serum or tissue proteins in order to elicit a response, which occurs typically in case of drug hypersensitivity reactions. Drug hypersensitivity reactions are seldom mediated by IgE. Drug reactions can be caused by cytotoxic, immune-complex formations and by other immunopathologic mechanisms.
Atopy is the infectious predisposition to produce IgE antibodies in response to allergen exposure. Atopy is defined as the genetic predisposition to form IgE antibodies in response to allergen exposure and thus a genetic predisposition exists for the development of atopic diseases. Mutations of specific alleles on the long arm of chromosome 5 is associated with high levels of IL-4 and IgE, known as IL-4 promoter polymorphism. Impaired function of Treg cells can also influence the development of atopic diseases.
Immediate hypersensitivity reactions in an infected immunsystem are mediated by IgE, and T and B cells play an important role in the production of these antibodies. TH1 cells produce interferon (IFN)gamma, IL-2, and tumor necrosis factor(TNF)-beta and promote a cell-mediated immune response (f.i. a delayed hypersensitivity reaction). TH2 cells produce IL-4 and IL-13, which then act on B cells and promote the production of antigen-specific IgE, playing thus an important role in the development of immediate hypersensitivity reactions.
The allergic reaction first requires sensitization to a specific allergen, develops in genetically predisposed individuals and occurs by cross binding IgE molecules on the

mastocytes delivering preformed mediators. The most important preformed mediators are: histamine, tryptase, proteoglycans chemotactic factors, leukotriens, prostaglandins thromboxane A2, PAF, adenosine, bradykinin and cytokines (IL-4, IL-5, IL-6, IL-13 and TNFalfa), which are all mediators of allergic reactions of this type.

Urticaria/angioedema: The release of the above mentioned mediators *in the skin* can cause pruritic wheals with surrounding erythema. If deeper layers of the dermis and the subcutaneous tissues are involved, painful angioedema can develop.

Allergic rhinitis: The release of the above mentioned mediators *in the upper respiratory tract* can cause sneezing, itching, nasal congestion, rhinorrhea and itchy or watery eyes.

Allergic asthma: The release of the above mentioned mediators *in the lower respiratory tract* can cause bronchoconstriction, mucus production and inflammation of the airways, causing wheezing and prolonged exspirations.

Anaphylaxis: *The systemic release* of the above mentioned mediators affecting more than one system causes the socalled anaphylaxis. In addition to all the above mentioned symptoms the gastrointestinal tract is also affected causing nausea, abdominal cramps, bloating and diarrhea. Anaphylactic shock is caused by the significant hypotension developing due to systemic vasodilation and pathologic vasopermeability. Anaphylactic shock, throat swelling and asphyxiation can even lead to death. Symptoms usually begin within minutes of allergen exposure (f.i. in case of drug administration, insect sting, food ingestion, allergen immunotherapy) and can recur hours after the initial exposure.

Patients might not be able to identify the allergen either because they are unaware of the allergy, or because they were unaware of being exposed to their allergen known.

Allergic reactions can be immediate reactions, **late-phase reactions** and can manifest a chronic allergic inflammation. Immediate or acute-phase reactions occur within seconds or minutes after allergen exposure.

Eosinophil and basophil cells, monocytes and T cells are believed to cause the late-phase reactions occuring hours after an antigen exposure and after the ceased symptoms of the acute-phase reaction. A late-phase reaction is characterized by redness and swelling of the skin, nasal discharge, airway narrowing, sneezing, coughing, and wheezing. These effects last a few hours and resolve within 24-48 hours.

Continuous or repeated exposure to an allergen can cause **chronic allergic inflammations** caused by eosinophils and T cells. Eosinophils release mediators causing tissue damages leading to structural and functional changes of the affected tissue.

Diagnosis: by special laboratory tests such as (RAST) and other in vitro IgE assays measuring antigen-specific IgE levels.

Treatment: is offered depending on the character and location of the allergic process by administering f.i. epinephrine, corticosteroids, antihistamines (anti H-1 and anti H-2), selective beta-2-adrenergic receptor agonists, leukotriene inhibitors given systemically, inhaled or topically applied.

RFR method: detects and may eliminate the pathogen microorganisms.

The most important pathogen microorganisms modifying asthmatic reactions are:

Mycoplasma fermentans: 442-451, 493-495 kHz

HTLV-1: 370-376 kHz

Coxsackie viruses A1, A5, A9, A16, B3, B5: 360-366 kHz

Bacteroides fragilis: 324-327 kHz

Herxheimer's reaction (known also as Jarisch-Herxheimer reaction or Herx) occurs if large quantities of toxins are released into the body from bacteria or fungi (typically from spirochetes such as *Treponema pallidum, Borrelia Burgdorferi sensu lato bacteria* and *Candida species*) dying due to antimicrobial or RFR method. This reaction is caused by an acute immune response to the toxins (endotoxins) released and not eliminated quickly enough and the released toxins either exacerbate the already ceased symptoms or create their own symptoms. This reaction is characterized by fever, chills, headache, myalgia,

confusion, psychosis and exacerbation of cutaneous lesions. The intensity and duration of the reaction reflects the intensity of the inflammation present.
Inflammatory cytokines, such as TNF alpha, Interleukin-6 and Interleukin-8 play a role in its development.

23.7. Autoimmunity

Autoimmune diseases involve immune reactions in which something triggers the immune system to react against the body's own tissues and to produce abnormal antibodies attacking these tissues. The appearance of antibodies directed against the body's own tissues represents an autoimmune response [redacted] A viral or bacterial antigen continuously adsorbed by the tissues can develop an autoimmune process. These autoantibodies may reflect a normal response to tissue antigens which, during the fetal development are anatomically separated from the immune system and appear later as a consequence of tissue breakdown. Autoantibodies can be produced also following the abrogation of the tolerance of the normal immune system caused by exogenous viral or bacterial antigens cross-reacting with own molecules or can be the product of an abnormal immune system, which lost its capacity to distinguish own molecules from foreign ones. Autoantibodies do not necessarily indicate an autoimmune disease. The latter term must be restricted to situations in which the autoimmune response (humoral or cellular) is responsible for tissue injuries.
Immune reactions are characterized by inflammation, normally provoked by a repair process and subsides when the repair is completed. In case of autoimmune diseases, the inflammation gets chronic, resulting in the damage of normal tissues. For example, in case of rheumatoid arthritis, the chronic inflammation damages the cartilage of the joints. The connective tissue in and around the joints and elsewhere in the body can become inflamed. The response does not kill the pathogen viruses or bacteria, as the response is not specific, not adequate and not sufficient. Concerning these autoimmune diseases Mycoplasma species play a significant role, [redacted]
Co-infections caused by Human T-cell Lymphotropic Virus-1 are frequently experienced among patients suffering from autoimmune diseases.
The resonant frequencies of HTLV-1 are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz

23.8. Allergic Reactions of Persons Infected by Human T-cell Lymphotropic Virus and/or Mycoplasma

The allergic condition is characterized by an altered immune response, an abnormal reaction to antigen substances. A person's immune system in order to prevent damaging of the invading allergens produces IgE antibodies against them. This, in turn, leads to the release of further mediators from other cells which cause irritation, inflammation and symptoms of allergic response. The symptoms of the allegic diseases may be caused by non-allergic factors as well. Allergic reactions can be conditioned and modified by *Human T-cell Lymphotropic Viruses* and/or *Mycoplasmas*. The term allergy is used to describe a response of the body to a substance, which is not necessarily harmful in itself, but provokes in case of predisposed persons an immune response and reaction that causes symptoms and diseases, which again can cause inconvenience, and misery. The different types of allergy and allergic reactions iniciated by T lymphocytes of mycoplasma-infected organs, can cause many a symptoms. The immediate hypersensitivity is easily recognizable as it involves quickly and dramatically occuring symptoms. The immune response to the

relevant allergen causes an IgE-dependent, mast cell-mediated reaction. An uncontrolled response could proceed from a physiologic local reaction to a self-perpetuating inflammatory state.
The body owns an antiallergic capacity and produces continuously mast cell-stabilizing factors. In case of *HTLV* or/and *mycoplasmal* infections its antiallergic capacity will be decreased. The body produces allergic reaction-blocking substances or neutralizing antibody factors which can prevent the allergic reaction. The antigen provokes an antibody and a neutralizing antibody production, which substances are either in a balanced or in an imbalanced state. The desensitization therapy is based on this mechanism. Different kinds of people have different kinds of antiallergic capacities, but in case of infections like those mentioned the antiallergic capacity becomes very low. Many antigen stimuli exhaust the allergic capacity of the body leading to the development of an allergic hypersensitivity. Endogen steroid-derivates inhibit the allergic response. This endogen steroid system again has also a capacity, which can also be exhausted causing a hyper allergic state. The diagnostic and therapeutic agents are generally of a low molecular weight and are considered to function as haptens, which form immunogenic conjugates with the host's proteins. The different types of allergic reactions are generally categorized according to their causes, the part of the body most affected, etc. In case of HTLV-infected T lymphocytes and/or mycoplasmal infected organs a chronic allergic syndrome will develop, which is difficult to treat leading thereby to a chronic hyper- or/and polyallergic syndrome. These chronic syndromes show a variety of complex signs and symptoms characterized by disabling fatigue, itching, swollen lymph nodes, myalgia, headache, urticaria, chronic dermatitis, atopic dermatitis, allergic purpura, photophobia, intermittent diarrhea, abdominal bloating, chronic bronchitis, chronic conjunctivitis, chronic rhinitis, food intolerance, chronic nephritis, chronic arthritis, irritability, depression and anemia.
Certain special forms of allergies not mentioned as yet are f.i.
the **physical allergies** developing in response f.i. to cold, sunlight, heat or a minor injury. *Exercise* can induce allergic reactions among asthma patients resulting sometimes even in an acute anaphylactic reaction.
Many substances existing in the nature have a photosensitizing potential and are recognized as potentially toxic. *Photo allergy* to drugs is an acquired, altered response of the skin to the energy of the light in the presence of a photosensitizer, and is presumably dependent on an antigen-antibody reaction or a delayed hypersensitivity reaction mediated by mononuclear cells. Drugs with photosensitivity effects are f.i. tetracycline, sulfanilamide, nalidixic acid, diethylstilbestrol, psoralen, chlorpromazine, tolbutamide derivates, etc. *Photo-dermatitis* is a mild-to-severe erythematous skin reaction causing often also vesicles and bullas. Skin allergy to sunlight frequently develops among people with *HTLV* or *mycoplasmal* infections. As to the various forms of **porphyria** endogenously synthesized photosensitizing molecules i.e. overproducted proto-, uro-, and coproporphyrins and their precursors cause photosensitivity reactions if exposed to light. Its symptoms can be a feeling of burning, itching, erythema, urticaria, edema, vesiculation, crusting, scarring and atrophy.
The resonant frequencies of the pathogens found among porphyria patients are: 355-358, 399 kHz
Among patients suffering from **atopic dermatitis or allergic purpura** HTLV and mycoplasmal infections can often be experienced (see their special Chapters).
Milk allergy and intolerancy often occur among infants who have no bifidobacteria infantis in their bowel. An allergy to milk can cause diarrhea, vomiting, rashes and loss of weight occuring most often among infants, though it can be acquired later on as well and even in adulthood. The aggravating factors of this allergic process are infections with *HTLVs* and *Mycoplasmas.*
The most frequent resonances found in case of milk allergy are: 370-374 kHz

Chinese Restaurant Syndrome is a hypersensivity reaction to monosodium glutamate (MSG), a flavor enhancer often used in Chinese cookings. This MSG can cause facial rashes, chest pain and burning sensations all over the body of susceptible persons. The amount of MSG causing these symptoms varies considerably concerning every person. This syndrome often develops among people infected with *Human T-cell Lymphotropic Viruses* or *Mycoplasma species.*

Allergic tubulointerstitial nephritis, an acute or chronic allergic tubulointerstitial kidney failure is caused by an allergic reaction triggered by drugs (such as penicillin, sulfonamides, diuretics and NSAIDs), or by certain fungal, bacterial and viral agents such as *Human T-cell Lymphotropic Viruses* and *Mycoplasma species.* These infections may result in an allergic-autoimmune process.

As regards **allergic arthritis** see in its special Chapter. Infectious agents, mostly viruses, may initiate the inflammatory, immunological and allergic process of rheumatoid arthritis.

The predisposing factors and etiology of allergy include physical stimuli (f.i. cold, solar rays, exercise, mechanical irritation), specific antigens (f.i. of parasites, viruses, bacteria, fungi and molds,) the hyperplasia of the mast cell membranes, hypereosinophilia, IgE-illnesses and platelet-activating factor anomalies and abnormalities, etc. *Human T-cell Lymphotropic viral* and *mycoplasmal* infections are frequently coexisting with allergic syndromes and can cause allergic, autoimmune and self-supporting processes.

Diagnosis: symptomatically, by identifying the specific allergen, etc. see above. Conventional serological detection of mycoplasmal or *Human T-cell Lymphotropic viral* infections are difficult, as Mycoplasma species are able to hide inside of the cells. This fact can result in normal antibody titers despite of an active mycoplasmal infection. The most reliable clinical testing for mycoplasmal infections uses blood, blood leucocytes or tissue biopsies and PCR. PCR and ELISA tests are specific to the examination of *Human T-cell Lymphotropic viral* infections.

Treatment: by eliminating the allergen sources, by treating the infections caused by parasites, bacteria, viruses, fungi and molds. If the patients suffer mycoplasmal or/and HTLV infections, the first to be done is to kill these pathogens. Frequently found pathogens which co-exist in allergic diseases are *Chlamydia, Borrelia Burgdorferi sensu lato, Histoplasma, Ureaplasma* and *viruses*, f.i. *EBV* etc. Symptomatically (see above)

RFR method: can eliminate even the antibiotic resistant bacteria, viruses and molds.

As regards the Mycoplasmal and Human T-cell Lymphotropic viral frequencies see Chapter 23.9. **As regards the frequencies of other pathogens: see their special Chapters.**

The treatment of allergic processes see Chapter 23.6., to treat these combined allergic syndromes is very difficult.

The most often found resonances in case of Photoallergy are: 315, 330-338, 372-378, 394-397, 402-404 kHz as well as the resonances of *Mycoplasma* or/and *Human T-cell Lymphotropic Viruses* (see their special Chapters).

23.9. Mycoplasmal and Human T-cell Lymphotropic Viral Infections in Chronic Syndromes

Certain chronic illnesses having diagnostic criteria are well defined, while others have mostly nonspecific multi-organ signs and symptoms that overlap and are indistinguishable from each other. The most autoimmune and degenerative diseases are multiorganic chronic diseases, their etiology is not exactly known. Certain bacteria and viruses can trigger autoimmune processes among genetically predisposed persons or among patients with

immune dysfunctions. *Mycoplasmal and/or Human T-cell Lymphotropic viral infections* are the triggers of these chronic syndromes.
Certain Mycoplasma species can either activate or suppress the host's immune system, and can thus evade the host's immune responses. Mycoplasma species produce immuno-modulating substances. They can secrete soluble molecules which stimulate the proliferation or inhibit the growth and differentiation of immune competent cells. Some Mycoplasmas can inhibit or stimulate the proliferation of certain normal lymphocyte subsets, induce B-cell differentiation and trigger the secretion of cytokines, f.i. IL-1, IL-2, IL-4, IL-6, TNFalpha, interferons and GM-CSF. Moreover, certain *M. fermentans*-derived lipids interfere with the interferon (IFN)-g-dependent expression of the MHC class II molecules of macrophages causing thus an impaired antigen presentation to helper T-cells. Mycoplasmas can evade the immune recognition by the potency of changing their surface antigens rapidly altering thus their cell surface structures. Their antigenic variability, the ability to suppress the host's immune responses, their slow growth rate and intracellular location explain the chronicity of mycoplasmal infections and the inability of the host to get rid off mycoplasmal infections. The human pathogenic species of the Mycoplasma family include *M. hominis, M. fermentans, M. penetrans, M. genitalium, M. salivarium, M. orale and M. pneumoniae.* It is a public evidence that certain mycoplasmas, i.e. *M. fermentans* (incognitus strain) species are unusually invasive and reside within the respiratory epithelial cells. The pulmonary macrophages are unable to kill pathogenic Mycoplasma species, nor certain Chlamydia species.
The pathogenic strains of the Mycoplasma family are obligate intracellular parasites being dependent on the intermediary metabolites and biosynthetic precursors of the host's cells. If these pathogens are released from the cells without cell lysis, they can carry with them the cell surface antigens of their host, and cause autoimmune reactions against the infected tissues. Mycoplasma effecting the host's cells can cause their apoptosis and thus the release of the host's antigens, some mycoplasmal antigens again mimic the host's own antigens, more over Mycoplasmas can capture some cell surface antigens of their host and incorporate them, these can all provoke autoimmune reactions.
Infections are often present in patients suffering from various forms of autoimmune diseases, such as RA, scleroderma, etc, and many of these patients respond to antibiotic therapy. Their recovery is slow; lasting for usually longer than a year. Other chronic infections with or without mycoplasmal or Human T-cell Lymphotropic viral infections can also be involved in various degrees in causing symptoms of chronic syndromes. Invasive bacterial and viral infections are associated with several acute and chronic illnesses, f.i. with Inflammatory Bowel Diseases; Rheumatoid Arthritis, HIV-AIDS; chronic genitourinary infections and chronic fatigue illnesses (CFS, FMS) etc.
Mycoplasmal infections seem to play an important but not yet well recognized role in these diseases, which are characterized by disabling fatigue, intermittent fever, swollen lymph nodes, sweating at night, arthralgia, myalgia, impairment in short-term memory, headache, skin rashes, intermittent diarrhea, loss of weight, abdominal bloating, chronic bronchitis, photophobia, anemia, autoimmune processes, confusion, transient visual scotomata, irritability, depression and degenerative symptoms. The symptoms affect more organs, and the signs, symptoms and laboratory test results are not consistent with any single, specific disease.
Mycoplasma can be related to the development of *RA* and may influence *HIV* pathogenesis. *M. hominis and U. urealyticum* play a significant role in a wide variety of *chronic urogenital diseases* (f.i. chronic pelvic inflammatory disease, infertility, chronic genital infections, pyelonephritis, Reiter's syndrome, etc). *Endocarditis and myocarditis* associated with M. pneumoniae infections are the cause of death in case of M. pneumoniae infections. The direct bacterial invasion of M. pneumoniae into the pericardial tissue appears to be more likely to cause pericarditis than autoimmune processes.

Human T-cell Lymphotropicviruses (1-6) decrease the immune responses. Human T-cell Lymphotropic Viruses reproduce themselves using usually CD4+ T lymphocytes. These immunodeficiency viruses are retroviruses [redacted] HTLV viruses released from the infected cells invade and destroy other lymphocyte cells as well. Their infection also disrupts the function of B lymphocytes, so that an immunodeficiency syndrome will develop. HTLV infections can initiate the development of chronic syndromes. These viruses are possible causative agents, cofactors or opportunistic pathogens in chronic illnesses, and have a significantly higher incidence rate of infection regarding these patients. Chronic fatigue is an often reported medical complaint of patients seeking medical care. Patients suffering from *fatigue syndromes* (f.i. chronic fatigue syndrome (CFS), fibromyalgia syndrome (FMS) etc.), feel muscle pain, weakness and most of all fatigue and can be distinguished from patients with many other multiorgan signs and symptoms, including immune system abnormalities.
Diagnosis: symptomatically, by serological and culturing procedures.
Differential diagnosis: by distingishing these chronic syndromes from infections caused f.i. by Chlamydia, Borrelia, Histoplasma, Ureaplasma, EBV, etc.
Treatment of Mycoplasma: by administering Doxycyclin, Ciprofloxacin, Azitromycin for a long time.
Treatment of Human T-cell Lymphotropic Viruses: by administering effective antiviral drugs.
RFR method: detects and may eliminate the microorganisms!
Mycoplasma fermentans can be a causative factor of chronic syndromes,
its most often found resonant frequencies are: 312, 329, 353, 361-365, 404, 442, 448-450, 505, 520 kHz
Those of Mycoplasma pneumoniae are: 307, 321-324, 337-344, 346-350, 352, 362, 397, 499 kHz
Non defined other Mycoplasma frequencies are: 312, 322-323, 329, 337-339, 342-349, 350-353, 361, 397-409, 424, 442, 448-450, 495-499, 534, 543-546 kHz
The frequencies of HIV-1 are: 365-367, 372, 382, 402, 450 kHz
The frequencies of HIV-2 are: 318, 372, 383, 396, 402, 450 kHz
AIDS associated frequencies are: 544, 569 kHz
Non defined HIV frequencies are: 349, 365, 424, 460 kHz
The frequencies of Human T-cell Lymphotropic Virus-1 are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz (found in cases of MS and autoimmune diseases).
The frequencies of Human T-cell Lymphotropic Virus-2 are: 314, 320-324, 370-376, 493-501 kHz (characteristic for Gulf War syndrome).
The frequencies of Human T-cell Lymphotropic Virus-3 are: 307, 312, 320-324, 338-340, 365-367, 397-400, 416, 428, 435, 453-455, 484, 526-530 kHz (often present in case of AIDS).
The frequencies of Human T-cell Lymphotropic Virus-4 are: 297, 454, 540-545 kHz
The frequencies of Human T-cell Lymphotropic Virus-5 are: 297-298, 315, 320-340, 354, 439, 480-482, 523, 544-545 kHz
The frequencies of Human T-cell Lymphotropic Virus-6 are: 359, 374-376, 382-383, 474-476, 570-578 kHz
The antibiotic therapy and the RFR method should be given contemporarily in case of mycoplasmal infections. Both Mycoplasma and HTLV infections lead to several different opportunistic secondary infections, so that the amount of the resonance frequencies of these secondary pathogens found is increased. The primer process is first to be treated.
As regards the secondary infections see their special Chapters.

Table of summary concerning the role of Mycoplasma in chronic syndromes, according to G. L. Nicolson.

Mycoplasma genitalium	Arthritis, chronic nongonococcal urethritis, chronic pelvic inflammatory disease, other urogenital infections and diseases, infertility, AIDS/HIV
Mycoplasma fermentans	Arthritis, Gulf War Syndrome, Fibromyalgia, Chronic Fatigue Syndrome, SLE, AIDS/HIV, other autoimmune diseases, ALS, psoriasis, scleroderma, Crohn's disease, IBS, cancer, endocrine disorders, Multiple Sclerosis, diabetes mellitus
Mycoplasma salivarium	Arthritis, Temporomandibular joint disorders, Eye and ear disorders and infections, gingivitis, periodontal diseases including even cavities.
Mycoplasma hominis and Ureaplasma urealyticum	Two Mycoplasmas commonly found in the urogenital tracts of healthy persons. However, over the years, the pathogenic roles of these Mycoplasmas have been proven in adult urogenital tract diseases, neonatal respiratory infections, and a range of other diseases usually in immunocompromised patients.
Mycoplasma pneumoniae	Pneumonia, asthma, upper and lower respiratory diseases, heart diseases, leukemia, CNS disorders and diseases, urinary tract infections, Crohn's dieseas and IBS, autoimmune diseases.
Mycoplasma incognitus (fermentans) and Mycoplasma penetrans	AIDS/HIV, urogenital infections and diseases, Autoimmune disorders and diseases
Mycoplasma pirum	Urogenital infections and diseases, AIDS/HIV

The treatment and cure of the chronic syndromes appear to be possible. Different severe pathogens together cause these diseases. Pathogenic Mycoplasmas, HTLV and other pathogen microorganisms can be found together in the blood or other specimens of patients suffering from a variety of chronic clinical symptoms caused f.i. by respiratory, oral cavity, genital and other infections, inflammatory, immunosuppressive diseases and fatigue syndromes. These, combined bacteria and viruses are the possible causative agents, cofactors or the cause of opportunistic infections in case of various other illnesses. Although they are not widely appreciated for their pathogenic properties, certain Mycoplasma, Chlamydia, Borrelia species, etc. and viruses appear to play a common role in the progression of chronic illnesses. The RFR method is very useful and effective in treating these chronic and multifactorial illnesses or syndromes. The conventional treatment and RFR method together give the optimal clinical result. The combined mycoplasmal and viral pathogenesis of these illnesses offer the possibility of a causal therapy.

23.10. Other Autoimmune Diseases

SLE
Antiphospholipid Syndrome
Rheumatoid arthritis
Sjögren's Syndrome
Polymyositis, Dermatomyositis
Progressive systemic sclerosis
Diabetes mellitus
Celiac Disease

PAN
Multiple Sclerosis
Etc.
Regarding these autoimmune diseases see their special Chapters.

23.11. Autoimmune Neuromuscular and Neural Diseases

Autoimmune neuromuscular diseases (ANDs) are progressive, usually fatal, neurodegenerative diseases, characterized by the degeneration of motor neurons, nerve cells in the central nervous system controlling the voluntary muscle movements. Muscle disorders caused by nerve dysfunctions include Amyotropic lateral sclerosis, Progressive muscular atrophy, Progressive bulbar palsy, Primary lateral sclerosis and Progressive pseudobulbar palsy. In case of these diseases, muscle weakness and muscle atrophy will develop throughout the body due to the degeneration of both the upper and the lower motor neurons that cease to send messages to the muscles. Unable to function, the muscles will gradually weaken, fasciculations (twitches) and eventually muscle atrophy will develop due to their denervation. The patient may ultimately lose the ability to initiate and control all his/her voluntary movements except those of the eyes. Cognitive functions are usually spared except in certain situations when the ALS is associated with frontotemporal dementia. (However, there are studies concerning MND patients that report on more subtle cognitive changes of frontotemporal type found when detailed neuropsychological testings were employed. Sensory nerves and the autonomic nervous system controlling functions like sweating, generally remain normal. Motor neuron diseases (MNDs) are progressive degenerative diseases in which the destruction of the cell bodies of the motor neurons is the primary process. These should be distinguished from diseases in case of which the axons of the motor neurons are primarily affected.

The classification and terminology of motor neuron diseases are, according to the affected cell types, as follows:

Primary Lateral Sclerosis (PLS): in which case solely the upper motor neurons are affected.

Progressive Muscular Atrophy (PMA) and **Spinal Muscle Atrophies (SMAs):** in which case solely the lower motor neurons are affected.

Amyotrophic lateral sclerosis (ALS): in which case the upper and lower motor neurons together are affected. ALS is the most common occuring illness of the MNDs. In British-English-speaking areas ALS is often called „motor neuron disease", but this Chapter will use the term MNDs (usually in plural form) as an umbrella term, as not every motor neuron disease is an amyotrophic lateral sclerosis.

In case of a genetic predisposition, AND can be caused by environmental risk factors, such as increasing age and a coinfection of the *HTLV* with *Mycoplasma fermentans*. These infections together provoke an autoimmune response in the brain, and cause a progressive degeneration of the motor neuron cells. (The antibodies might directly or indirectly impair the function of the motor neurons, interfering with the transmission of signals between the brain and the muscles. Cetain recent evidence indicates that microglia, the immune cells of the nervous system, are heavily involved as concerns the later stages of the disease. Though this hypothesis hase not been proved so far.)

Illnesses belonging to the group of autoimmune neuromuscular diseases are as follows:

23.11.1. Primary Lateral Sclerosis

Primary lateral sclerosis (PLS), a progressive, degenerative disease of the upper motor neurons is characterized by a progressive spasticity (stiffness). It affects the lower extremities, the trunk, the upper extremities and the bulbar muscles.
ALS can initially show only signs of the upper motor neuron involvement. Symptoms initially considered to belong to PLS have the potency to get reclassified as ALS if sufficient signs of both the upper and lower motor neuron involvement develop over time. In some cases, such reclassifications may occur only at the autopsy. There exist datas of patients referring to one of the genes associated with familial ALS showing only lower motor neuron involvements during their life, moreover, even at their autopsy. Most physicians would classify this disease as ALS, on the basis of the presence of the ALS gene. Patients with PLS occasionally have mild, nonspecific and nonprogressive findings of denervation experienced by electrodiagnostic testings. The severity of the denervation and re-innervation does not resemble that seen in case of ALS and does not justify these patients to be classified as suffering from ALS. These patients may be concerned that their PLS eventually could evolve into ALS.
Diagnosis: by laboratory studies (f.i. hemogram, erythrocyte sedimentation rate, lues serology, such as VDRL and RPR, serology tests concerning Borreliosis, Mycoplasmal, HIV and other HTLV infections. MRI, CT, SPECT, PET, diffusion tensor MR imaging and magnetization transfer MR imaging.
Treatment: there is no specific therapy, symptomatically, by administering baclofen, tizanidine, benzodiazepine and clonazepam, analgesics, antidepressants, etc.
RFR method: detects and may eliminate all the pathogen microorganisms.
The most frequently found resonancies are: 312-321, 329, 346-353, 364-365, 378-387, 397, 403-404, 416, 428, 440-451, 487-490, 493-497, 504, 520-523 kHz

23.11.2. Spinal Muscle Atrophies

Spinal muscle atrophies (Spinal muscular atrophies, SMAs) are hereditary, genetic predisposited diseases, caused by combined infections and characterized by progressive muscle hypotonia and muscular weakness. This disease is caused by a progressive degeneration of the alpha motor neurons of the anterior horn cells in the spinal cord. The weakness of the proximal musculature is more severe than that of the distal one. The motor neurons of the cranial nerves (mostly CNV-CNXII) can also be involved. Intelligence and sensation, which are related to the posterior horn cells of the spinal cord, are spared. The diaphragm, the involuntary muscles of the gastrointestinal system, the heart and the sphincters are neither affected.
Mutations in the SMN gene cause the loss of function of the SMN protein leading to a progressive motor unit degeneration of the lower motor neurons. Why these mutations results in the selective degeneration of the lower motor neurons is unclear, as the SMN protein is expressed in many types of neurons and organ systems, too.
Newborns and infants suffering from **SMA type I**, (named also Acute, severe, infantile SMA or **Werdnig-Hoffman disease**) can not hold their heads up when pulled to the sitting position, will slip through the examiner's hands when held vertically. They lay limp in the physician's hand when held under the abdomen and facing down. Their weakness affects mostly the proximal muscles and may mimic a muscle disease (myopathy). Deformities of the limbs and joints can be observed, and is caused by hypotonia in utero.
SMA type II is the chronic infantile form, where infants cannot get to a sitting position on their own, though they may stay upright if placed in that position. If the patient's hands are held out, a characteristic fine postural tremor may be observed.
In case of the **SMA type III**, the chronic juvenile SMA or **Kugelberg-Welander syndrome** children have proximal muscle weakness and various degrees of muscle hypotonia and wasting.

SMA type IV is the adult-onset form which findings are similar to those with SMA type III but the weakness is less severe.
The most frequent infections present in case of SMA patients include *Mycoplasma fermentans*, *HTLV* and *neurovirus* (i.e. vaccine virus modified by means of passage into and growth in nervous tissue) which are able to cause acute neurological infections.
Diagnosis: no specific laboratory test can identify SMA, though CT, MRI and electromyogram might be useful. Serum aldolase and serum CK findings are within the reference ranges in case of patients with SMA, opposed to findings of patients with Duchenne muscular dystrophy and Becker muscular dystrophy. In case of later-onset SMAs, these muscle enzymes may be slightly elevated.
Treatment: supportively, in order to improve the patients' quality of life and to minimize their disability, particularly concerning patients with slow progression.
RFR method: detects and may eliminate the found pathogen microorganisms.
The most frequent resonances are: 312-321, 346-353, 378-387, 403-404, 440-451, 493-497 kHz

23.11.3. Amyotrophic Lateral Sclerosis (ALS)

ALS (also named Lou Gehrig's Disease) is characterized by the slowly progressive degeneration of the upper motor neurons (UMNs) and the lower motor neurons (LMNs). Bulbar motor neurons are often involved at the onset, and result in bulbar weakness (progressive bulbar palsy). The upper motor neuron involvement of the spinal cord tracts (anterior horn cells) results in the spastic weakness of the limbs (primary lateral sclerosis). A gene that causes familial ALS resides on chromosome 21 and is the SOD (superoxyde dismutase) gene. [redacted] 10.25.)
The onset of ALS may be subtle, so that the symptoms are frequently overlooked. The earliest symptoms are obvious weakness and/or muscle atrophy. These are followed by twitching, cramping, stiffness of the affected muscles, as well as by muscle weakness affecting an arm or a leg and by a slurred and nasal speech. These symptoms are caused by the dying muscles, therefore these symptoms without the weakness or atrophy of the affected muscles are likely not to be the ALS disease. The early symptoms of the ALS depend on, which motor neurons innervating a certain part of the body are damaged first. If the symptoms first affect one leg, the patient will experience awkwardness when walking or running or notice that he/her is tripping or stumbling more often. In case of an onset affecting a hand or an arm the person will experience difficulty in simple tasks requiring manual dexterity such as buttoning a shirt, writing, or turning a key in the lock. Regardless on the part of the body first affected by the disease, the muscle weakness and atrophy will spread to other parts of the body as the disease progresses. Patients experience increasing difficulty in moving, swallowing (dysphagia), and speaking or forming words (dysarthria). Though the sequence of symptoms and the rate of progression vary from person to person, eventually the patients will not be able to stand or walk, get in or out of bed on their own and use their hands and arms. The disease usually does not affect the cognitive abilities. ALS is a fatal disease. Patients with bulbar onset, particularly the lower motor neuron type, have a poorer prognosis, the autoimmune process will quickly progress.
Differential diagnosis: there are no specific laboratory tests to identify ALS. Though MRI scans are often normal in case of patients with ALS, they can reveal evidence of other problems such as a spinal cord tumor, Multiple Sclerosis, a herniated disk in the neck, syringomyelia, or cervical spondylosis. ALS can be triggered by infectious diseases caused f.i. by *HIV*, other *HTLVs*, *Lyme disease*, *Mycoplasma fermentans syphilis* and *Tick-borne Encephalitis viruses* which can cause even ALS-like symptoms. Neurological disorders such as Multiple Sclerosis, post-polio syndrome, multifocal motor neuropathy and spinal muscular atrophy also can mimic certain facets of the disease. There are documented cases of patients presenting ALS-like symptoms, having positive test results of borrelia specific

antibodies and responding to antibiotics. A borreliosis occuring together with mycoplasma infection is particularly difficult to diagnose.
Prognosis carried by this diagnosis and the variety of diseases or disorders that can resemble ALS in the early stages of the disease, patients should always obtain a second neurological opinion.
Treatment: [redacted]**10.25.**
RFR method: detects and may eliminate the viral and mycoplasmal component of the disease.
The most frequent resonances of ALS are: 312-321, 329, 346-353, 364-365, 378-387, 397, 403-404, 416, 428, 440-451, 487-490, 493-497, 504, 520-523 kHz

23.11.4. Progressive Supranuclear Palsy

Progressive supranuclear palsy (PSP), also known as **Steele-Richardson-Olszewski syndrome**, is a neurodegenerative disease affecting mostly the eye movements, characteristically including supranuclear, vertical gaze dysfunction, accompanied by extrapyramidal symptoms and is causing cognitive dysfunctions. The disease usually develops after the sixth decade of life, its diagnosis is purely clinical.
Pathophysiology: The role of hereditary predisposition in case of PSP remains elusive. Although most cases of this illness appear to be sporadic, rare genetically determined forms may exist. A variant in the gene for tau protein called H1 haplotype, located on the chromosome 17, appears to be necessary, but not sufficient to cause PSP. Tau protein, a protein found in the axons of healthy neurons, binds to other proteins (called microtubules) in order to form the cytoskeleton of the neuron and to provide the tracks, over which, material can be transported from one part of the neuron to another. Under abnormal circumstances, this normally soluble tau protein may be collected into insoluble protease-resistant helical filaments. Abnormalities of tau-protein are present in several neurodegenerative diseases, f.i. in Alzheimer's disease and taupathies). The exact triggers for the conversion from normal tau to this aggregated form are not completely understood yet. Though the e4 allele of apoprotein E gene (ApoE) may be a risk factor for the development of Alzheimer's disease, it is not associated with the PSP, Parkinson's disease and alcoholic dementia.
Symptoms: The slowing of vertical saccades and square wave jerks are early signs in most cases. The classic gaze palsy characteristic for PSP, typically involving the downgaze before upgaze, is a supranuclear ophthalmoplegia (i.e. the lesion is above the ocular motor nuclei, thus sparing the ocular motor nuclei, the nerve fascicles, the neuromuscular junctions and the extraocular muscles as well.
Imbalance, caused by poor extrapyramidal postural reflexes, and a monotone, hypophonic dysarthria i.e. poor articulation can often accompany to the earliest symptoms. Later on, the ophthalmoparesis can affect the horizontal eye movements, so that a complete ophthalmoparesis can develop.
The trigger of PSP may be a combined infection caused by *HTLV*, *Mycoplasmas* and certain other neuropathogens.
Diagnosis: symptomatically and by examining features (f.i. damaged vestibular ocular reflex, absent Bell phenomenon) in order to establish that the infranuclear structures are intact and that the lesion lies within the supranuclear domain. No specific laboratory or imaging findings are associated with PSP.
Whipple PCR may be helpful in eliminating the possibility of Whipple disease.
Treatment: No therapy is proven to be effective yet.
RFR method: detects and may eliminate all the pathogen microorganisms.
The most frequently found resonances are: 288-290, 317-319, 321, 340-341, 353-359, 365, 372, 416, 440-452, 493-497, 504, 518-520, 578 kHz

Autoimmune neuromuscular diseases are progressive processes, nevertheless, the RFR method can inhibit and stop these progressive processes, if using it before the development of definite neuron damages.

23.11.5. Multiple System Atrophy

Multiple system atrophy (MSA) is a sporadic, autoimmune, progressive, neurodegenerative disease with infectious etiology affecting genetically predisposed persons. This illness is characterized by combinated extrapyramidal, pyramidal, cerebellar, and autonomic dysfunctions. Striatonigral degeneration (SND) is a neurodegenerative disease belonging to the group of multiple system atrophy (MSA). Other forms of this illness are Shy-Drager syndrome (autonomic failure predominates) and the sporadic olivopontocerebellar degeneration (cerebellum predominates).

The pathomechanism of this MSA is characterized by the progressive loss of neuronal and oligodendroglial cells in numerous loci of the CNS. Its possible etiology is an autoimmune process combined with a viral infection and toxic agents. Genetic predisposition plays also a role in its etiology. The clinical symptoms of MSA correlate with the loci of the lost cells in the CNS.

The chronic damages of the glial cells may impair the trophic function between the oligodendrocytes and axons and cause secondary neuronal damages. Whether the inclusions represent primary lesions or are nonspecific secondary markers of a cellular injury is unknown. In addition extensive myelin degenerations occur in the brain. Changes in myelin may play an important role in the pathogenesis of MSA. In case of MSA there are cnombined chronic infections caused by many a different pathogen, mostly viruses, such as *HTLV, HBLV, ECHO virus, Coxsackie virus, EBV, measles* and others, such as *mycoplasmas and Nocardia asteroides.*

The symptoms can be associated with the problems of the autonomic nervous system, causing dysphagia, incontinence, orthostatic hypotension, gastroparesis, erectile dysfunction, etc. The other group of its symptomes are aspecific Parkinsonian symptoms and nonspecific neurologic symptoms such as ataxia and pyramidal symptoms including light-headedness, dizziness, dimming of vision, pain of the head, neck, or shoulder altered mentation, weakness, especially of the legs, fatigue, etc.

Diagnosis: symptomatically. By Iodine-123 MIBG scintigraphy, in order to differentiate Parkinson disease and MSA. By MRI, PET, autonomic function testing and EMG.

Differential diagnosis: Parkinson disease, Steele-Richardson-Olszewski syndrome, corticobasal ganglionic degeneration and cerebrovascular syndromes.

Treatment: symptomatically. Levodopa administered in combination with a dopa decarboxylase inhibitor may be helpful.

RFR method: detects and may eliminate the pathogen microorganisms.

The most frequent resonances are: 289-303, 313-319, 326, 353-359, 370-374, 406-416, 442-452, 530-536, 578-580 KHz

23.11.6. Parkinson-like Multiple Sclerosis

Parkinson-like Multiple Sclerosis (PLMS) is an autoimmune, degenerative, progressive disorder of the central nervous system impairing often the sufferer's motor skills and speech, as well as other functions. PLMS is an autoimmune inflammatory demyelinating disease of the central nervous system. PLMS lesions, characterized by perivascular infiltrations of monocytes and lymphocytes, appear as indurated areas in pathologic specimens.

PLMS symptoms are muscle rigidity, tremor, slowing and, in extreme cases, loss of physical movements (akinesia). The primary symptoms are caused by the decreased stimulation of the motor cortex by the basal ganglia, usually caused by an insufficient

formation and action of dopamine, produced in the dopaminergic neurons of the brain. The secondary symptoms of this illness can include high level cognitive dysfunction and subtle language problems such as a very quick speech, difficult to understand. In case of PLMS the symptoms of the Parkinson disease and Multiple Sclerosis are mixed, the typical Parkinson syndrome symptoms such as those of multiple system atrophy (MSA), progressive supranuclear palsy (PSP) and corticobasal degeneration (CBD), and sometimes even dementia with Lewy bodies (DLB) can all be present. Lewy bodies experienced in the brain tissue of the patients with idiopathic Parkinson's disease are less dens and widespread than in this illness. PLMS diseases can progress quickly. This disease is caused by combined infections among genetically predisposed persons. These infections can be caused by mycoplasmas (mostly *M. fermentans*), *Neisseria species*, *Hemophilus influenzae*, *Cryptococcus*, *Nocardia asteroides*, *Borrelia Burgdorferi sensu lato*, *HTLV, Herpes Simplex Virus, CMV, ECHO virus, Coxsackie virus* and other *neurotrop viruses* such as *measles, chickenpox, rubeola*, one or more of them are coinfected with a *Chlamydia species* as well.

These viruses infecting the immune cells and the nerves can be periodically reactivated leading thus to acute exacerbations of PLMS. Periodical reactivations of *EBV, CMV* or/and *HTLV* are often found but their causative role in MS is difficult to prove. The mechanism of genetic predisposition concerning the losing of the brain cells in PLMS is associated with an abnormal accumulation of the protein alpha-synuclein bound to ubiquitin in the damaged cells. HLA-DRB1 1501 alleles show susceptibility to PLMS although complex genetic-environmental interactions are needed to the manifest this illness. The autoimmune inflammation and demyelination can occur in any part of the brain and the spinal cord, the symptoms depend on the area affected. Demyelination and damages in the nerve pathways bringing signals to muscles cause problems in moving, while demyelination in the nerve pathways carrying sensations to the brain causes disturbances of the sensorium.

The symptoms, similar to those of Parkinson disease are: tremor, rigidity, bradykinesia and akinesia, postural instability and shuffling.

The symptoms, similar to those of Multiple Sclerosis are: tingling, numbness, double vision, partial blindness, pain in one eye, dim or blurred vision, loss of central vision, mild emotional or intellectual changes.

Diagnosis: typically, the diagnosis is based on medical history and neurological examinations. By DaTSCAN, MRI. By blood tests tested for vitamin B12, folate levels, ANA titers, etc. By cerebrospinal fluid examinations, ELFO

Treatment: symptomatically. The treatment should usually be done in neurological centrums.

RFR method: detects and may eliminate all pathological viruses, bacteria and fungi.

PLMS has the same frequency resonances as PD and MS.

The most frequent resonances, which are the same as in case of Parkinson disease are: 288-303, 307, 317-319, 353-359, 363-374, 416, 442-451, 471-473, 576-578 kHz

The most frequent resonances, which are the same as in case of Multiple Sclerosis are: 288, 317-319, 321-324, 370-376, 378-387, 402-405, 440-451, 458, 480, 536, 560-564, 584 kHz

RFR method may temporarily increase the symptoms, so that patients should be kept in a neurological centrum. The development of PLMS can be inhibited by this method.

23.11.7. Guillain-Barré Syndrome

Guillain-Barré syndrome is an acute ascending polyneuritis, a form of acute polyneuropathy combined with rapidly worsening muscle weakness, sometimes leading to paralysis. Its presumed cause is a neurotrop viral infection followed by an autoimmune reaction of the body's immune system attacking the myelin sheath. This disease can develop after surgical intervention or immunization, too. Cranial nerves are often affected.

Retention of urine can also come about, requireing but seldom catheterization for more than a few days. Inflammatory cell infiltrates in the liver, spleen, lymph nodes, heart and other organs reflect the systemic nature of the disease. From a pathologic point of view, most evidences suggest that the clinical manifestations of this disease are the result of a cell-mediated immunologic reaction directed against the peripherial nerve. The causative factor of this neuro-immunological process is a neurotrop viral infection cooperating with a bacterial infection.
Diagnosis: symptomatically, by CT, by cerebrospinal fluid analysis, electromyography examinations, nerve conduction studies, etc.
Differential diagnosis: by distinguishing it from poliomyelitis, and other forms of polyneuritis.
Treatment: by administering corticosteroids, IVIG, physiotherapy and symptomatically.
RFR method: detects the neurotrop virus, bacteria or molds and eliminates them!
The most frequent resonances are: 310-318, 340-344, 359-370, 372-382, 389, 394, 397, 406-411, 447-452, 482, 496, 513-514, 541-543, 557, 564 kHz
As to the mold frequencies, see its special Chapter.
The resonant frequencies of molds are low, while those of the neutrop viruses are higher.

23.11.8. Myasthenia Gravis

Myasthenia gravis (MG) is the most common primary disorder of neuromuscular transmission. Its cause is usually an acquired immunological abnormality, but some cases result from genetic abnormalities of the neuromuscular junction. The acquired form of Myasthenia gravis is an autoimmune disorder of the peripheral nerves in which antibodies are formed against the acetylcholine (ACh) nicotinic postsynaptic receptors of the myoneural junction, so that their function will be abnormal, causing episodes of muscle weakness. The reduced number of ACh receptors results, if repeatedly used, in a characteristic progressive weakening of the affected muscle, and following a period of rest the strength of this muscle will be regained.
The cause of MG is a combined infection with *HTLV, HBLV, Mycoplasma fermentans* and other *neurotropic viruses* affecting genetically predisposed persons. In case of this autoimmune illness the specific antibody is completely known. A foreign antigen which cross-reacts with the nicotinic ACh receptor is the cause of MG, but the triggering antigen has not yet been identified. Anti-AChR antibodies are found in approximately 80-90% of patients with MG. The role of T cells in the pathogenesis of MG may also be important, as thymus hyperplasia and thymoma often come to pass in myasthenic patients. These are abnormalitis of the thymus, which is the central organ of the T cell–mediated immunity and the immunological self-tolerance. The thymus contains all necessary elements for the pathogenesis of myasthenia gravis: myoid cells expressing AChR antigens, antigen presenting cells and immunocompetent T-cells. Thymic abnormalities are clearly associated with this illness but the mode of the association is uncertain. *Mycoplasmas and HTLV or/and HBLV* infections can lead to the loss of immunologic tolerance to AChR.
Patients lacking anti-AChR antibodies can suffer from seronegative myasthenia gravis (SNMG). Many of these patients have antibodies against muscle-specific kinase (MuSK), an enzyme playing a role in the postsynaptic differentiation and clustering of acetyl choline receptors. The patients with anti-MuSK antibodies are mostly female, suffering from respiratory and bulbar muscle weakness.
MG is sometimes associated with other autoimmune disorders, f.i. with autoimmune thyroiditis, SLE, RA). Patients with MG can show a wide range of signs and symptoms, depending on the severity of the disease. The bulbar muscles are mostly affected and patients often develop some degree of intermittent generalized weakness as well.
Symptoms: Patients with myasthenia gravis complain of specific muscle weakness but do not suffer from generalized fatigue. Ocular motor disturbances, ptosis or diplopia, are

usually the initial symptoms of myasthenia gravis. The facial muscles can be slack, the face expressionless. The patients can not hold up their heads, their voice gets nasal. The severity of weakness fluctuates during the day, is usually least severe in the morning, gets worse as the day progresses, especially after a prolonged use of the affected muscles. Patients cannot breath adequately, nor clear-up their bronchial secretions, wheezing and pneumonia are often present. The course of the disease is usually progressive. Factors worsening the myasthenic symptoms are emotional upset, systemic illnesses (especially viral respiratory infections), hypothyroidism or hyperthyroidism, pregnancy, menstrual cycle, drugs affecting the neuromuscular transmission, as well as fever.
An insufficient medication leading to a myasthenic crisis, an excessive medication resulting in a cholinergic crisis can show similar symptoms. The latter results from an excessive administration of cholinesterase inhibitors (f.i. neostigmine, physostigmine, etc.) and causes a flaccid muscle paralysis clinically indistinguishable from weakness experienced in MG. Myasthenic crisis or cholinergic crisis may cause bronchospasm with wheezing, bronchorrhea, respiratory failure, diaphoresis, and cyanosis as well.
Miosis and Sludge syndrome (ie. salivation, lacrimation, urinary incontinence, diarrhea, GI upset, hypermotility and emesis) may also be markers of a cholinergic crisis. Deep tendon reflexes are usually normal in these cases.
Diagnosis: symptomatically, by EMG and by examination of anti-acetylcholine receptor antibodies. By CT scan or MRI of the chest in order to detect thymoma.
Treatment: by offering various immunomodulating therapies including plasmapheresis, corticosteroids, intravenous immunoglobulins (IVIg), immunosuppressants and by thymectomy. AChE inhibitors (f.i. Pyridostigmine) and immunomodulating therapies (f.i. Plasmapheresis and thymectomy are the fundaments of the treatment. By administering immunosuppressive drugs (f.i. high doses of corticosteroids, azathioprine, cyclosporine etc.) are important modalities for treating MG.
RFR method: detects and may eliminate all pathogenic microorganisms.
The most frequent resonances found in case of Myasthenia gravis are: 370-386, 416-420, 442-451, 484-490, 493-495 kHz

23.11.9. Paraneoplastic Lambert-Eaton Syndrome

The symptoms of this autoimmune disease causing muscle weakness are similar to those of Myasthenia gravis. Lambert-Eaton myasthenic syndrome (LEMS) is caused by the inadequate release of acetylcholine rather than by autoantibodies to acetylcholine receptors. This syndrome can appear also sporadically, though it usually occurs as a side phenomenon of certain cancers, especially small cell lung cancers. Some other cancers (including thymus cancer, bladder cancer, prostate cancer, lymphoma, non-Hodgkin's lymphoma, T cell leukemia, non-small cell lung cancer, thymoma, carcinoma of the breast, the stomach, the colon, the bladder, the kidney, the gallbladder and the transitional cell carcinoma of the bladder) can but very rarely be associated with Lambert-Eaton syndrome. This syndrome involves antibodies interfering with the substances (presynaptic calcium channels) that provide the communication between nerves and muscles with neurotransmitters. These antibodies are supposed to be produced by the immune system in order to attack cancer cells, they but accidentally bind and affect the substances in the neuromuscular junction. This syndrome is a paraneoplastic disorder associated with small cell lung cancer, a fatal, quickly progressing form of lung cancers. Its major characteristic symptom is muscle weakness, which may be the first and only sign of the associated cancer, can be experienced before, during or after the time of establishing the diagnosis of the related cancer. Lambert-Eaton syndrome is a remote effect of this small cell lung cancer affecting not only the muscles but also the autonomic nervous system, like f.i. the sweating, salivation, gut movement and sexual erection.

While in case of various similar diseases the disease is linked to a cross-reaction with an infective agent, there is no other causative pathogen known than the *Mycoplasma species* and the *Human B-cell Lymphotropic Virus*, that could account for LEMS.
Symptoms: Patients with Lambert-Eaton syndrome usually suffer from a progressive proximal muscle weakness of the lower extremities. Fatigue, pain, tingling in arms and legs, dry mouth, drooping eyelids and impotence can also come about. Knee jerk reflex can be diminished or even disappear. The weakness of the pelvic, thighs and shoulder-arm muscles are often accompanied by stiffness. The syndrome may precede the appearance of the malignant tumor even by 2 years, although not all patients get a tumor. In contrast to myasthenia gravis, the symptoms of LEMS tend to be worse in the morning and improve with exercise and nerve stimulation.
The microbiological background in this syndrome can vary. *Clostridium botulinum* bacteria produce toxic substances, paralyzing the muscles by inhibiting the release of acetylcholine from the neuromuscular junctions. *Borrelia* bacteria can iniciate an autoimmune process while *Human Papilloma Viruses* are responsible for the small cell lung carcinoma. LEMS develops effected by various different combined pathogen microorganisms, but *Mycoplasma species* are in each case present.
Diagnosis: by EMG, CT, PET, MRI, toxin identification, microbiological examinations. By the examinations of voltage-gated calcium channel antibodies, acetylcholine receptor antibodies, by clinical and laboratory findings, etc.
Treatment: symptomatically, by administering corticosteroids, azathioprine, cyclosporine etc. Treatment of lung carcinoma by surgery.
RFR method: detects and may eliminate the pathogen microorganisms!
The resonances of the most frequently found microorganisms in this syndrome are: 318, 331, 337-347, 370-382, 397-399, 403-410, 422, 442-451, 477, 486, 491-497, 513-518, 528 kHz
LEMS can be a sporadically occuring side phenomenon of certain cancers, especially small cell lung cancers. In these cases, the resonant frequencies are the same as those of the original cancer disease. The primarily LEMS is very seldom, with but few resonant frequencies.

23.11.10. Autoimmune Plexus Syndromes

In case of these diseases one or more nerve plexus are damaged by antibodies that attack the persons own tissues by autoimmune reaction. This autoimmune reaction is probably responsible for neuritis. This neuritis form can often be experienced in case of viral infections caused f.i. by *Herpes Simplex Virus-1 and 2* and more frequently by *HZV*. *Borrelia Burgdorferi sensu lato* infections often cause such plexus syndromes. Autoimmune diseases can develop many years after getting infected. Four nerve plexuses are located in the trunk of the body. Cervical plexus provides nerve connections to the head, neck and shoulder, brachial plexus to the chest, shoulder, arm, forearm and hand, lumbar plexus to the back, abdomen, groin, thighs, knees and legs, and sacral plexus, to the pelvis, buttocks, genitalia, thighs, legs and feet. The lumbar and sacral plexus are interconnected, they are sometimes referred to as lumbosacral plexus. Intercostal nerves are located between the ribs. Autoimmune plexus syndromes form a group of chronic autoimmune neuritis causing pain, paresthesia and sometimes even paralysis of the affected area of the nerve plexus. A nerve plexus is damaged mostly secondarily to physical injuries or cancer. An accident pulling or severely bending the arm in its socket may damage the brachial plexus. Falling down can similarly injure the lumbosacral plexus. A cancer growing in the upper region of the lung can invade and destroy the brachial plexus, a cancer of the intestine, the bladder, or the prostate can invade the lumbosacral plexus. Cancer viruses develop cancers in the damaged plexus regions.

The damage of the plexus caused by an autoimmune disease may be regenerated slowly over several months or years.
Diagnosis: by autoantibody examinations, by virus and Borrelia serology, by autoimmune process examinations, electromyogram, nerve conduction studies, etc. By CT and MRI.
Differential diagnosis: by distnguishing it from other neurological and autoimmune diseases.
Treatment: depends on the cause of the plexus disorder. Corticosteroids may be needed to treat the acute neuritis of an autoimmune plexus syndrome.
RFR method: detects and may eliminate the causative viral and bacterial infections!
The most frequent resonances are: 290-294, 344-345, 353-362, 376-386, 415-421 kHz
After eliminating the viral and bacterial components the autoimmune inflammation cascade should be stopped by administering corticosteroids.

23.11.11. Chronic Autoimmune Bell's Palsy

Concomitant infections caused by *Human T-cell Lymphotropic Viruses* and *Mycoplasma species* play an important role in the etiology of Chronic Autoimmune Bell's palsy (CABP) as these infections catalyse the occuring immunologic and autoimmunologic processes. Patients with other diseases or conditions, f.i. those suffering from Lyme borreliosis sometimes develop a peripheral facial nerve palsy, if they have also a mycoplasmal infection, but this condition can not be classified as Bell's palsy. Some frequent viral infections cause inflammation and/or a related autoimmune response resulting in local demyelination of the facial nerve. Pathogens leading the list are Human Herpes Virus type-1 and type-2 (HSV1, HSV2); Varicella-Zoster virus (VZV); Coxsackie viruses, and rarely Influenza B Virus; Adenovirus, EBV; Hepatitis A, B and C Viruses; West-Nile Virus, Measles virus, CMV; JC virus and Rubella virus.
Genetic predisposition was reported in approximately 5% of cases. The way of inheritance of these cases is supposed to be autosomal dominant. The causative factor is a deficient protective protein coded in the DNA; which protects the host against viral infections. It seems that the developing inflammatory immun-autoimmune processes lead to ischemia, compression and/or demyelination impairing the neural conduction at the affected locus of the nerve. Following the virus elimination the neural function will or will not get regenerated, so that the neural damage may either be reverzible or irreverzible. These autoimmune processes can affect other regions of the CNS as well.
Diagnosis: symptomatically, by electromyogaphy, by viral and/or bacterial antibody examinations.
Differential diagnosis: by distingiushing it from Heerfordt's syndrome, Melkerson's syndrome and other non-autoimmune Bell's palsy forms. Diseases or conditions associated with facial palsy (f.i. *Borrelia B. sensu lato* infections) are often misdiagnosed, meaning that the origin of the palsy is not cleared up.
Treatment: by administering ACTH or corticosteroids. This traditional treatment is not able to eliminate the causative viral/bacterial infections, it does only effect on autoimmune processes.
RFR method: can detect all the pathogen microorganisms, and may eliminate them.
The first step to take is to detect and eliminate the Mycoplasma species and HTLV.
The most frequent resonances are: 311, 330, 321-424, 370-374, 442-451, 496 kHz
The second step to take is to detect and eliminate the other infections present, **the most frequent resonances of these microorganisms are: 285-294, 297-302, 344-345, 352-363, 378-387, 402, 408-410, 416-420, 466-475 kHz**
As to the resonances of the viruses, see their special Chapters. The elimination of viruses is the first task of the RFR method. Immun-autoimmun processes can only be stopped following a complete virus elimination.

23.12. Autoimmune Panencephalitis

Measles viruses (paramyxoviruses) commonly cause a postinfectious autoimmune syndrome. This subacute or chronic sclerosing panencephalitis may be considered to be a "slow" form of measles encephalitis. Autoimmune panencephalitis develops after an infection with *HTLV* or/and *Mycoplasma species*. The interaction of the *measles virus* with *HTLV* and *mycoplasma* causes a chronic autoimmune process. Its most frequent symptoms are incoordination, ataxia and myoclonic jerks together with abnormalities of the pyramidal and exrapyramidal motor system. Cortical blindness, papilledema and optic atrophy may be present as well as focal chorioretinitis. This disease may develop after vaccination (Post-vaccination syndrome), but occurs extremely rarely and only if the patient has also *HTLV* and *mycoplasmal* infections.

Diagnosis: by antibody examinations and EEG.

Treatment: symptomatically.

RFR method: can detect and may eliminate the infectious viral and mycoplasmal microorganisms.

The most frequent resonances are: 350, 364, 368-374, 381, 387, 390, 402, 436, 442-454, 468, 478, 492, 522-528, 564 kHz

23.13. The Role of Coxsackie Viruses in Chronic Syndromes

Coxsackie virus B is an RNA enterovirus, usually causing asymptomatic or brief gastro-enteric and upper respiratory tract infections. Sometimes, there can develop severe sequelae of Coxsackie virus B infections, including chronic immune mediated meningitis, carditis and other chronic diseases and syndromes.

Coxsackie viruses may play an important role in chronic autoimmune processes f.i. in case of Alzheimer's disease, chronic myocarditis, osteomyelitis, dermato-polymyositis, idiopathic pulmonary syndrome, chronic fatigue syndrome, and possibly, juvenile-onset diabetes type I, which are all believed to be immune-mediated. The virus has an incubation time of a week in the gastrointestinal tract and then involves its target organs via hematogenous dissemination, mostly the skeletal muscles but also the CNS (causing there meningitis, encephalitis) and the myocardium (causing carditis with or without associated pericarditis).

The classic etiologic agent for these pathological changes are *Coxsackie virus B*, the most frequent serotypes being *B1, B2, B3, B4, and B5*. Other non-polio enteroviruses, f.i. *ECHO viruses type 6 and 19*, are also reported to cause syndromes, including chronic immune/autoimmune processes, very similar to those experienced in case of infections with Coxsackie virus B.

Human beings are the only known reservoirs of enteroviruses, their transmission occuring via the fecal-oral route. Their incubation time is usually 2-5 days. These viruses alone do not cause any autoimmune process, but can, as a participant in a combined infection together with other bacteria or viruses, cause chronic syndromes.

Diagnosis: by virus isolation on kidney cells of monkey or embryonic lungs. By fluorescent staining and neutralization assays, by RT-PCR and ELISA.

Treatment: Symptomatically.

RFR method: detects and may eliminate Coxsackie and other ECHO viruses. RFR method is the most effective way to eliminate Coxsackie viruses frequently present in certain chronic syndromes.

The mostly found resonances in case of Alzheimer's disease are: 288-291, 294-303, 313-321, 440-451 kHz

The mostly found resonances in case of Asthma bronchiale are: 361-366, 440-451 kHz

The mostly found resonances in case of Diabetes mellitus are: 307-308, 361-366, 424-426, 444-447 kHz
Common frequency resonances in case of Idiopathic pulmonary syndrome are: 288-291, 361-366 kHz
Common frequency resonances in case of Dermato polymyelitis are: 288-290, 441-451 kHz
Common frequency resonances in case of Osteomyelitis are: 318, 340, 348, 353, 372, 383-384, 396-397, 403, 409, 425, 442-451, 463-464, 513-514, 544, 555 kHz
Common frequency resonances in case of Idiopathic heart diseases are: 288-290, 407-413 kHz

23.14. Idiopathic Pulmonary Fibrosis

Pulmonary fibrosis can be caused by many a factor, f.i. by viral, rickettsial, mycoplasmal, tuberculous and fungal infections, bird droppings, other mineral or organic substances, such as asbestos, silica, carbon, metal dusts, and drugs: such as penicillamine, nitrofurantoin, paraquat and some autoimmune diseases. Yet, despite the many a possible cause, in half of the cases of pulmonary fibrosis, the cause can never be identified. This illness is a chronic, progressive, fibrosing interstitial lung disease of unknown etiology, poor prognosis and no proven effective treatment. The disruption of the homeostasis of alveolar epithelial cells caused by unknown endogenous or environmental stimuli results in a diffuse epithelial cell activation and an aberrant epithelial repair, in which process cytokines (TNFalpha, interleukins, TGFB-1) and apoptosis are involved. This pathological process leads to the overproduction of collagen and fibronectin by fibroblasts.
Genetic mutation, involvings surfactant protein C, and infections play together a significant role in its etiology. Some other secondary factors, f.i. wood and metal dust exposure, drugs, chronic aspiration secondary to Gastroesophageal Reflux Disease (GERD) and cigarette smoking can all be associated with increased severity and mortality of persons with IPF.
Its symptoms depend on the extent of the lung damage, the rate at which the disease is progressing, and on complications such as infections and heart failure. The most common symptoms are coughing, the loss of appetite and weight, tiredness, weakness and a vague chest pain.
Diagnosis: symptomatically, by HR-CT, x-ray, pulmonary function tests, biopsy, special immunological tests, etc.
Treatment: symptomatically, by administering corticosteroid, immunosuppressive drugs, etc.
RFR method: detects the primarily causative infective agent or/and the secondarily one causing co/reinfection. One should detect and eliminate them. RFR method can only be effective within the time of the autoimmune inflammation of infectious origin and before the development of fibrosis.
The most frequent resonances are: 321-324, 370-376, 442-445, 493-498, 510-513 kHz

23.15. Goodpasture's Syndrome

Goodpasture's syndrome or Goodpasture's disease is an anti-glomerular basement membrane disease characterized by the rapid destruction of the kidneys and the haemorrhaging of the lungs. It is an allergic/autoimmune disease (caused by a type II hypersensitivity reaction), where the patient's immune system attacks and damages the cells of the kidneys and lungs presenting Goodpasture's antigen. In most cases it affects genetically predisposed persons with HLA-DR2 haplotypes. Goodpasture's syndrome has a number of underlying etiologies and presents a small vessel vasculitis associated with antineutrophil cytoplasmic autoantibodies (ANCAs); similarly to Wegener granulomatosis

and microscopic polyangiitis. Upper respiratory tract infections or flulike illnesses often occur before the onset of this disease.
Symptoms: at the beginning of the illness patients suffer from a dry cough and a minor breathlessness which can last for many years. Later on they experience shortness of breath and cough up blood. Dyspnoe, hemoptoe-caused anemia and a rapidly progressing kidney failure are characteristic signs of this illness. Owing to the non-characteristic early symptoms and the rapid progression of the disease, the diagnosis can often be established but very late in the course of the disease.
Diagnosis: symptomatically, by urine testing, x-ray, kidney and lung biopsy, by tests for anti-GBM and ANCA antibodies.
Treatment: symptomatically. By administering corticosteroids and immunosuppressants. By apheresis, by oxigen therapy.
RFR method: detects usually many different pathogenic resonances and infections caused f.i. by *Mycoplasma, HTLV, Cytomegalovirus, Influenza* and other viruses, and can eliminate them.
The most frequent resonances are: 308-321, 367-387, 442-451, 493-497 kHz
The autoimmune cascade can be stopped by shortly administered corticosteroids.

23.16. Autoimmune Hepatitis

Autoimmune hepatitis is a disease in which the immune system of the body attacks the virus-infected liver cells. Autoimmune hepatitis is characterized by a chronic hepatocellular inflammation and necrosis, which tends to progress to cirrhosis. Immune serum markers are frequently present, the disease is often associated with other autoimmune diseases. The basis of autoimmune hepatitis can be an acute or chronic viral infection, alcoholism, exposure to hepatotoxic medications and hepatotoxic chemicals. Autoimmune hepatitis is a multisystem disorder affecting persons of all ages. This condition can coexist with chronic viral hepatitis, may be triggered by viral infections caused by f.i. *Human T-cell Lymphotropic Viruses, CMV, EBV* or/and *Mycoplasma fermentans* and by different chemical structures, f.i. minocycline, etc. This illness, in which the cell-mediated ,immunological attack is directed against the hepatocytes, affects genetically predisposed persons. This kind of destruction may be initiated or triggered by viral infections with *Hepatitis viruses*, or *EBV*, and by chemical agents including interferon, melatonin, alpha methyldopa, nitrofurantoin, etc. See list Chapter 23.1.2
Symptoms include abdominal discomfort, loss of weight, diarrhea, fatigue, myalgia, arthalgia, mild pruritus, anorexia, chest pain, cushingoid features, skin rashes, edema, itching, joint pain, nausea, vomiting, dark urine, hirsutism, anemia, eosinophilia, plasma cell invasion, hepatomegaly, jaundice, cirrhosis, erythema, ascites and amenorrhea.
Disease associations with other autoimmune diseases include rheumatoid arthritis, Felty syndrome, Sjögren's Syndrome, erythema nodosum, systemic sclerosis, mixed connective-tissue disease and immune complex vasculitis.
Diagnosis: by autoantibody examinations, serum protein electroforesis, aminotransferase examinations, by ultrasound and liver biopsy.
Treatment: by administering corticosteroids and immunosuppressants.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequently found pathogen microorganisms are as follows:
Hepatitis A Virus: 285-295, 320-330, 340-356, 361, 366, 403, 420-436, 449, 487-488, 498, 570-590 kHz
Hepatitis B Virus: 293, 340-341, 372, 384, 392-402, 414-420, 444-454, 488 kHz
Epstein-Barr Virus: 337-339, 342-347, 372-383, 518
Cytomegalovirus: 305, 349, 406-410, 534, 548-550 kHz
Coxsackie virus A9, B3-4: 334, 360-364, 430, 444, 552-554 kHz

ECHO virus 2, 3, 6-9, 11-14, 18-19, 22-24: 308-321, 369, 391, 403, 472, 526 kHz
Human T-cell Lymphotropic Virus 1, 2, 3, 6: 370-376, 382 kHz
Mycoplasma fermentans: 442-444, 447-451, 493-495 kHz
In case of autoimmune hepatitis the resonant frequencies found are various, other frequencies, differing from those mentioned above, might also be present. RFR method should be used together with the conventional treatment.

23.17. Addison's Disease

Addison's disease is a chronic adrenocortical glucocorticoid deficiency mostly associated with autoimmune processes. It can be caused by an infection of the adrenal gland, formerly mostly by tuberculosis. Viral infections, caused f.i. by *CMV, EBV* and *Coxsackie viruses*; fungal infections, such as *histoplasmosis*, *coccidioidomycosis*, and *cryptococcosis*; and, predominantly *Mycoplasma fermentans*, can all be causative agents of this Addison's disease. A primary adrenocortical insufficiency is relatively rare, while a secondary one occurs with increasing frequency. It may develop at any age and may affect both sexes with equal frequency. In developed countries Addison disease is caused mostly by the autoimmune nonspecific destruction of the adrenal gland. Other factors may sometimes also be the cause of this illness, such as bilateral tumor metastases, amyloidosis, or sarcoidosis.

Adrenal insufficiency can also be caused by a defect somewhere in the hypothalamic-pituitary-adrenal axis. Primary adrenal insufficiency is the result of the destruction of the adrenal cortex. Zona glomerulosa, i.e. the outer layer of the adrenal gland, produces aldosterone, while cortisol is produced in zona fasciculata and zona reticularis, the middle and the innermost layers of the adrenal gland. Dehydroepiandrosterone is produced in zona reticularis.

Clinical findings are noted only if 80-90% of the adrenal cortex is destroyed. Its etiology is multifactorial, including autoimmune processes, viral, mycobacterial and fungal infections, neoplastic, traumatic, iatrogenic factors, vascular hemorrhage, emboli, thrombus, and metabolic disorder such as amyloidosis events. In case of the destruction of the adrenal cortex, the feedback inhibition of the hypothalamus and anterior pituitary gland is interrupted, so that corticotropin hormones will be continuously secreted. Corticotropin and melanocyte-stimulating hormones (MSH) are components of the same progenitor hormone. Half of all patients with Addison's disease have complement-fixing and/or circulating adrenal antibodies.

The increased MSH level of patients results in a characteristic bronze pigmentation generally noted in case of primary adrenal insufficiency.

Its **Symptoms** include weakness, anorexia, asthenia, nausea, vomiting, diarrhea, loss of weight, fatigue, hypotension and occasionally hypoglycemia. Nausea, vomiting, diarrhea, and abdominal pain can also come to pass. Symptoms can vary, depending on the duration and degree of the adrenal hypofunction, there can even develop a fulminating shock associated with an acute massive destruction of the glands.

Polyglandular autoimmune disease (PGAD I) is characterized by the destruction of the adrenal and thyroid glands causing adrenal insufficiency, hypothyroidism and chronic candidiasis. PGAD I may also be associated with type 1 diabetes mellitus, hypogonadism, chronic hepatitis, immunoglobulin A (IgA) deficiency, chronic atopic dermatitis, keratoconjunctivitis, vitiligo and alopecia.

PGAD II, named also Schmidt syndrome, is an autoimmune-mediated adrenal insufficiency associated with autoimmune-mediated thyroiditis and type 1 diabetes mellitus.

The autoimmune destruction of the adrenal gland is characterized by lymphocytic infiltrates. If the adrenal destruction results from sarcoidosis or malignancy there can also noncaseating granulomas be experienced.

Mycoplasma fermentans and HTLV infections iniciate the antibody-mediated autoimmune destruction of the adrenal cortex.
Diagnosis: by examining morning cortisol levels, by corticotropin stimulation tests, by insulin tolerance tests, CT, MRI, etc. By blood tests showing the lack of corticosteroids, especially of cortisol, by low sodium and high potassium levels.
Treatment: by administering corticosteroids regardless of the cause of the illness.
RFR method: in case of an autoimmune adrenal process of infectious origin RFR method detects the pathogen microorganisms, eliminates them and cancel the autoimmune cascade.
The resonances of the most frequently found pathogen microorganisms are as follows:
Tuberculosis: 429-436 kHz
Cytomegalovirus: 408-410, 530-536 kHz
Epstein-Barr Virus: 372-383, 518-519 kHz
Coxsackie virus: 341-342, 370-376, 443-444, 533 kHz
Histoplasma: 296-306, 308, 315, 383, 434, 442 kHz
Mycoplasma fermentans: 442-444, 447-451, 493-495 kHz
HTLV: 370-376 kHz
Cryptococcus: 392-306, 313-319, 357, 363, 402-405, 457-459 kHz
Coccidioidomycosis: 321, 338, 347, 362-366, 382-389, 391-395, 440-449, 574 kHz
Candida albicans: 384-390, 443-453, 572-586 kHz
As to other frequencies, see their special Chapters.

23.18. Graves' Diseases

Hyperthyroidism can result from several causes, including immunological reactions, f.i. as in case of Graves' disease. This disease is supposed to be caused by antibodies stimulating the thyroid to produce too much thyroid hormones. People with Graves' disease have typical signs of hyperthyroidism and can additionally have the distinctive Merseburg triad (i.e. nodular or diffuse goiter, tachycardia and exophthalmus). Since the entire gland is stimulated, it can become greatly enlarged, causing a bulge in the neck. People with Graves' disease can also have bulging eyes and less commonly, raised edematous skin alteration over the shins (myxedem).
Thyroid-stimulating immunoglobulins (TSIs) bind to and activate the thyrotropin receptors, causing an enlarged thyroid gland and an increased synthesis of thyroid hormone by the thyroid follicles. Graves' disease, along with Hashimoto thyroiditis, is classified as an autoimmune thyroid disorder. In some patients, Graves' disease represents a part of a more extensive autoimmune process termed autoimmune polyglandular syndrome, which can be associated with pernicious anemia, vitiligo, diabetes mellitus type 1, autoimmune adrenal insufficiency and SLE. In case of Graves' disease, B and T lymphocyte–mediated autoimmun reactions are directed against certain well-known thyroid antigens, i.e. thyroglobulin, thyroid peroxidase, sodium-iodide symporter, and the thyrotropin receptor. The thyrotropin receptor is the primary autoantigen of Graves' disease and is responsible for the manifestation of hyperthyroidism. In case of this disease, the antibody and the cell-mediated thyroid antigen–specific immune responses are well defined. The thyroid gland is under continuous stimulation by circulating autoantibodies against the thyrotropin receptor, and the pituitary thyrotropin secretion is suppressed owing to the increased production of thyroid hormones. These stimulating thyrotropin receptor antibodies belong mostly to the immunoglobulin G1 subclass, cause the release of thyroid hormones and thyroglobulins and stimulate the iodine uptake, the protein synthesis, and the growth of the thyroid gland.
The anti–sodium-iodide symporter, antithyroglobulin, and antithyroid peroxidase antibodies appear to play an unimportant role in the etiology of hyperthyroidism in Graves' disease. They are however markers of autoimmune disease against the thyroid.

Intrathyroidal lymphocytic infiltration is the initial histologic abnormality of patients with autoimmune thyroid disease, which correlates with the titer of thyroid antibodies.
If left untreated, Graves' disease can cause severe thyrotoxicosis. A life-threatening thyrotoxic crisis can occur. A long-standing severe thyrotoxicosis leads to a severe loss of weight and to catabolism of bones and muscles. Cardiac and psychocognitive complications cause significant problems. Graves' disease is often associated with ophthalmopathy and acropachy.
The characteristic signs and symptoms of Graves' disease are as follows:
Heat intolerance, increased sweating, restlessness, anxiety, irritability, insomnia, loss of weight despite an increased appetite. Warm and moist skin; fine hair; onycholysis; vitiligo; alopecia; pretibial myxedem often come about. Tremor, proximal muscle weakness, hyperactive deep tendon reflexes, hypokalemic periodic paralysis occur mostly in case of susceptible ethnic groups. Osteoporosis, acropachy, elevated serum calcium and alkaline phosphatase levels can often be experienced. Tachycardia, palpitations, increased pulse, atrial fibrillation, cardiomyopathy, dyspnea, hyperdefecation with or without diarrhea, elevated transaminase levels, lid lag, lid retraction, proptosis, diplopia, normocytic anemia, slightly depressed total WBC count with a relatively hightend count of lymphocytes and monocytes, decreased total cholesterol and triglyceride levels are the most often present findings and signs of this illness.
Graves' disease is influenced by a combination of environmental and genetic factors.
CTLA4, HLA-DRB1, and HLA-DQB1 haplotypes seem to be associated with susceptibility to Graves' disease. Other influencing factors include infection, iodide intake, stress, female sex, steroids and toxins.
There are theories concerning the immune mechanisms involved, f.i. about a specific crossover between different cell antigens with an infectious agent or a superantigen, about the alteration of the T cell repertoire, idiotypic antibodies becoming pathogenic antibodies, about the association with a variety of infectious agents such as *Yersinia enterocolica*, *Borrelia Burgdorferi s. l. and Mycoplasma* species (*M. fermentans),* about stress as a factor relating to thyroid autoimmunity, about the role played by estrogens influencing the B-cell repertoire, etc.
Diagnosis: by specific laboratory immunological tests and hyperthyroidism tests.
Treatment: symptomatically, by corticosteroids, x-ray and surgery.
RFR method: detects and may eliminate the pathogen microorganisms!
The most frequent resonances are: 305, 327, 337-340, 342-347, 370-376, 380, 389-397, 402, 422, 428-440, 442-452, 500-510, 518, 528, 530-536 kHz

23.19. Hashimoto's Disease

This illness, also termed lymphadenoid goiter, is a chronic inflammatory disease of the thyroid, in which autoimmune factors play a significant role. This disorder affects mostly middle aged women. The histopathological evidence of the participation of autoimmune factors is the lymphocytic infiltration of the thyroid gland. In the serum increased concentrations of immunoglobulins and antibodies can be observed, which are directed against several components of the thyroid tissue. The clinically most important of them are the antimicrosomal and the antithyroglobulin antibodies. This disease has a viral etiology and affects genetically predisposed persons.
Symptoms: typically, the consistency of the thyroid gland get rubbery, its margins are scalloped and the general outline of the gland is preserved. After the beginning of the disease the patient may be metabolically normal, even if having an increased serum TSH. Owing to the progressive replacement of the thyroid parenchyma by lymphocytic infiltrations and fibrous tissues, there will develop later on hypothyroidism of increasing severity.

Diagnosis: by laboratory examinations and histological confirmations by needle biopsy.
Treatment: There is no specific treatment for Hashimoto's thyroiditis. The administration of anti-inflammatory drugs and steroids may be effective. The illness often requires a hormone replacement therapy.
RFR method: detects the virus and other pathogen microorganisms and eliminates them!
The most frequently found resonances are: 370-373, 402-410, 442-451 kHz
As to the frequencies of Herpes viruses, [redacted]
As to the frequencies of Borrelia B. sensu lato, [redacted]
Infections with Herpes virus and Borrelia Burgdorferi sensu lato are often present in case of patients with Hashimoto's disease, the early killing of these pathogens can be beneficial.

23.20. Autoimmune Alopecia Areata

Autoimmune alopecia (AA) is a cytotoxic, T cell mediated autoimmune disorder found mostly among genetically predisposed individuals. AA can primarily be caused by infections with *Mycoplasma fermentans, HTLV, CMV, Borrelia B.s.l., Syphilis,* triggered by emotional and psychosocial stress and by a decreased immune function in case of a familiar genetic predisposition [redacted]
The process is known to be T cell mediated, though antibodies directed to hair follicle structures can also be observed. The autoantibody response is heterogeneous and targets multiple structures of the anagen-phase hair follicle. Histologically, lesional biopsies of AA show a perifollicular lymphocytic infiltrate around the anagen phase hair follicles. The hair follicles are infiltrated by T-helper and T-suppressor cells. $CD4^+$ and $CD8^+$ lymphocytes play likely a prominent role, because the depletion of these T cell subtypes results in a complete or partial regrowth of the hair. AA is often associated with other autoimmune conditions, most often with thyroid diseases and vitiligo.
AA patients can have 5-6 kindes of circulating antibodies such as antithyroid, thyroglobulin antibodies, antinuclear antibodies, etc.
Diagnosis: symptomatically, by biopsy.
Differential diagnosis: by distinguishing it from androgenetic alopecia, pseudopelade, telogen effluvium, tinea capitis and trichotillomania.
Treatment: by corticosteroid injections, corticosteroid creams, topical minoxidil, anthralin, topical immunotherapy and phototherapy.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequently found pathogens are:
Mycoplasma fermentans: 442-444, 447-451, 493-495 kHz
Human T-cell Lymphotropic Viruses: see their special Chapter.
Cytomegalovirus: 408-410, 530-536 kHz
Epstein-Barr Virus: 372-383 kHz
Treponema p. pallidum: 346-348 kHz
Borrelia B. sensu lato species: 377-384 kHz

23.21. Histoplasmosis of Immunosuppressed Patients

The causative agent of Histoplasmosis is a dimorphic soil fungus *Histoplasma capsulatum.* This disease is endemic in Central and South America, the Caribbean, Africa and Asia; though it can occur anywhere, where the soil conditions (warm humid soil and large migratory bird populations) are appropriate for the growth of H capsulatum. It is the most common pulmonary and systemic mycosis of humans. Its clinical **symptoms** vary from a mild, flu-like, often unnoticed illness, to a rapidly progressing, often fatal, disseminated

disease. The clinical symptoms vary depending on the host's immunity and the size of the inoculum.
There exist five serotypes of H capsulatum, including some avirulent strains. Histoplasma species exist in mycelial form at ambient temperatures. The spores of H capsulatum (microconidia) become airborne when the soil is disturbed. The microconidia (1-5 mm in diameter) can be easily inhaled and deposited in the lungs. At body temperature, the proliferation of its infective yeast form occurs within 3-5 days. The initial immune response occuring with neutrophils is ineffective against it. Macrophages ingest the continuously proliferating yeasts. The specific immune response develops within 10-21 days after infection. Specific helper T cells will activate macrophages to form granulomas that characterize the disease. The extracellular killing is mediated by Natural Killer cells and enhanced by antibodies.
Symptoms: In most cases the symptoms are nonspecific such as fever, chills, myalgia, nonproductive cough and chest pain. This acute phase can last for 1-21 d, and be associated with fatigue, night sweats and loss of weight. Fatigue may persist for weeks after the resolution of acute symptoms. The histoplasmal pneumonitis, which is predominantly a mononuclear infiltrate, develops 2 weeks after getting infected. These granulomas are formed mostly in the pulmonary parenchyma and in the hilar and mediastinal lymph nodes, can be caseated and later on calcificated and fibrotic. These multiple granulomas with multinucleated giant cells are characteristic. The bone marrow, the liver, the adrenal glands, CNS, joints, heart valves and blood vessels can also be involved in case of persons with an impaired T cell–mediated immunity. Disseminated infections can localize in any tissue, leading to a variety of complications.
In case of immunocompetent adults histoplasmosis can progress into a **Severe Acute Respiratory Syndrome (SARS)** wich is a respiratory distress syndrome-like illness of adults, can cause **histoplasmoma, mediastinal obstructive syndromes** (obstructing f.i. the airways, the esophagus and the large blood vessels) **pericarditis, rheumatologic syndromes** (arthritis, arthalgia and erythema nodosum) persisting for months. **Chronic pulmonary histoplasmosis** is similar to pulmonary tuberculosis. CNS involvement causing seizures and neurological deficits can also come to pass.
A **progressive disseminated histoplasmosis (PDH)** can occur among infants and patients with damaged cell-mediated immunity, f.i. in case of AIDS, Hodgkin's disease, lymphoreticular malignancies, etc. Its **symptoms** are usually low-grade fever, loss of weight, malaise and oropharyngeal ulcerations. PDH may rapidly progress, causing hepatosplenomegaly, coagulopathy, adrenal insufficiency and pancytopenia.
Diagnosis: by the verification of H. capsulatum (by culturing sputum, BAL, blood, etc). By serology (CF, RIA, ID, ELISA tests), CT scanning, x-ray examinations, MRI.
Treatment: In case of immunocompetent patients the acute forms resolve without any specific treatment. In case of immunocompromised persons by administering systemic antifungal drugs and symptomatically.
RFR method: detects and may eliminate the H. capsulatum!
The most frequent resonant frequencies of H. capsulatum are: 298-308, 315-319, 374, 380-385, 424, 432-435 kHz. Use RFR method after the medical treatment.

23.22. Autoimmune Hemolytic Anemia

An immune response attacking the red blood cells will cause an autoimmune hemolytic anemia (i.e. an immune-mediated anemia). Autoimmune hemolytic anemia is a disorder characterized by autoantibodies reacting against the red blood cells, persons suffering from this type of anemia suffer usually hematologic malignancies (such as lymphoma, leukemia with *HTLV* infection), or connective tissue diseases (such as SLE or scleroderma with *Mycoplasma fermentans* infection).

Hemolysis is a form of the premature destruction of erythrocytes, and if the bone marrow cannot compensate for the erythrocyte loss with its activity, it will cause hemolytic anemia

Hemolysis can be the symptom of a large number of hereditary and acquired disorders as well. The etiology of a premature erythrocyte destruction is diverse and can be the result of conditions such as intrinsic membrane defects, abnormal hemoglobins, erythrocyte enzymatic defects, immune destruction of the erythrocytes, mechanical injury and hypersplenism. Hemolysis is associated with the release of hemoglobin and lactic acid dehydrogenase. The increase of indirect bilirubin and urobilinogen is derived from the released hemoglobin. A patient with mild hemolysis may have normal hemoglobin levels if the increased production matches the rate of erythrocyte destruction.
Marked anemia may occur in case of patients with mild hemolysis if the erythrocyte production in the bone marrow is transiently stopped by a viral infection (f.i. by *parvovirus B19*) or by other infections (f.i. by *Bartonella, Eperythrozoon i.e. Mycoplasma*). Sickle cell anemia and Thalassemia are diseases of infants and children wich illnesses are associated with skeletal and skull deformities and expansions of the bone occuring together with significantly increased hematopoiesis.
Several variants of glucose-6-phosphate-dehydrogenase deficiency exist. The A variant affects generally West African and African American persons. The Mediterranean B variant occurs among individuals of Mediterranean descent and among people in Asia
Penicillin, quinine, quinidine, L-dopa and other agents may cause immune-hemolysis.
Oxidant drugs and infections can trigger the hemolysis of patients with G-6-phosphate-dehydrogenase deficiency.
Splenomegaly occurs in case of hereditary spherocytosis and other hemolytic anemias but is not present in case of other hemolytic disorders, such as G-6-PD deficiency and can suggest underlying disorders such as chronic lymphocytic leukemia, some lymphomas and SLE.
Acquired hemolytic conditions can develop due to immune disorders, toxic chemicals and drugs, antiviral agents such as ribavirin, physical damage and various infections.
Diagnosis: by blood examinations, autoantibody examinations.
Treatment: symptomatically. Acute forms associated with infections get better on their own. In case of severe symptoms by administering corticosteroids.
RFR method: detects infections and eliminates the pathological microorganisms.
The most frequently found pathogen agents are:
Bartonella: 324, 330-335, 366, 373-375, 402-404, 430-438, 495 kHz
Eperythrozoon: 368-381, 404-408, 480-486 kHz and others.
Parvovirus B19: 314-318, 326-330, 386, 499, 515, 526, 574 kHz
Mycoplasma fermentans: 442-444, 447-451, 493-495 kHz
HTLV and HBLV: their most frequently found resonances are 370-376 kHz (see also their special Chapter).

23.23. Cold Antibody Hemolytic Anemia

Cold antibody hemolytic anemia is a condition in which the body develops autoantibodies reacting against red blood cells at room temperature or colder temperatures. This type of anemia can be acute or chronic. The acute form often develops among patients having acute infections, especially certain types of pneumonia or infectious mononucleosis. Its acute form usually doesn't last long, is relatively mild and disappears without any treatment. Its chronic form is most common among women having rheumatism or arthritis.
Diagnosis: by tests detecting antibodies on the red blood cell surfaces which are more active at temperatures below body temperature.
Treatment: symptomatically.

RFR method: detects and may eliminate the causative pathogen!
The most frequent resonancies: 442-451, 466-475 kHz

23.24. Systemic Immune Complex Diseases in General

Immune complex diseases are characterized by the deposition of antigen-antibody complexes in the vascular and glomerular basement membranes and by the presence of these complexes in the blood circulation and in other body fluid compartments. The deposition of these immune complexes in the tissues initiates immune mediated inflammations with resultant tissue damages. Immune complex diseases have a common pathogenic mechanism, but their etiology varies, their causative antigens are different, they are f.i. bacteria in case of Lyme disease and viruses in case of a Coxsackie viral infection. Immune complex diseases constitute clinical syndromes which include glomerulonephritis, arthritis, pericarditis, pleuritis and vasculitis and vasculitis caused skin eruptions. After the first exposion of a person to a foreign substance, the antibody response to it develops a week later. The immune system with its synthesized antibodies attacks the antigens forming antigen-antibody complexes, which facilitate the removal of the antigens. Normally, the majority of the immune complexes will be removed by the cells of the reticuloendothelial system. If these immune complexes are deposited along the vascular and glomerular basement membranes, inflammations can develop there, leading to the symptoms of an immune complex disease. In case of any given immune complex disease the etiology depends on the nature and source of the antigens that form immune complexes as well as on the immunogens that incite the immune response. In case of most immune complex diseases the antigen and immunogen are of the same substances, but in certain autoimmune diseases they may differ. The etiologic factors of the immune complex diseases may be microorganisms, drugs, or tumors. The etiology of an immune complex disease is defined if the specific antigens and antibodies causing the present tissue inflammation are identified. Immune complex vascular diseases can develop in case of viral or bacterial infections spontaneously, providing a continuous source of antigen for immune complex formation. Since the immune complex diseases have a common pathogenic mechanism, their clinical manifestations are often similar even though the responsible antigens may be quite different.

Several types of bacterial and viral infections are accompanied by an immune complex disease, and cause glomerulonephritis as a common feature. In case of Goodpasture's syndrome f.i., antigens derived from the responsible microorganisms are presumed to be released at the loci of the viral or bacterial growth and are then deposited as immune complexes in glomeruli. The identification of the bacterial or viral antigens in glomeruli is very difficult. Examples for these immune complex syndromes include f.i. poststreptococcal glomerulonephritis, glomerulonephritis associated with bacterial endocarditis, infected osteomyelitis, quartana malaria, and Hepatitis B infection.

Chronic viral infections can cause immune complex diseases. The three deposits, i.e. the virus, the host's antiviral antibody and C3 deposits were observed in a granular pattern along the glomerular capillary wall as well as in the mesangium. Granular deposits in the glomeruli experienced in case of infections caused by *Coxsackie B viruses, polyoma viruses, and other leukemic viruses* suggest that an immune complex disease can also be present in case of these infections. In case of a transplacental infection of the infant the maternal antiviral antibodies transferred in utero or by milk may very early induce a severe immune complex disease of the fetus or of the newborn infant.

Fungal diseases such as *coccidioidomycosis* can be accompanied by erythema nodosum and arthritis, most likely due to immune complex deposition.

Patients with malignancies may develop arthritis, arthralgia and skin eruptions, moreover a nephritic syndrome may associate f.i. with adenocarcinoma.
Drugs can cause immune complex diseases either by acting as immunogens or by inducing the synthesis of autoantibodies. Numerous drugs behaving as haptens will be bound to proteins forming them into antigens. Drugs, such as penicillins, sulfonamides, and different animal serums are potential immunogens and can cause immune complex diseases. Gingivitis of periodontal diseases are thought to be generated by complexes composed of bacterial antigens from the plaques and of specific antibodies.
Glomerulonephritis, arthritis and skin lesions can be frequently observed, sometimes individually or in various combinations. Renal involvement may not be clinically apparent or experienced by urinanalysis; but biopsy may show immune deposits in the mesangium or in the glomerular capillary loop. Pleuritis, pericarditis and small-vessel vasculitis can also be caused by immune complexes.
The clinical signs and symptoms of an acute form of immune complex disease include fever, myalgia, skin lesions, arthralgia, arthritis, gastrointestinal symptoms, lymphadenopathy, uveitis, nausea, vomiting and abdominal pain. The skin lesions are usually urticarial, petechial, erythematous and maculo-papular. An immune-complex arthritis begins usually in one or two joints and then rapidly affect even more joints. Wrists, ankles, knees and the small joints of the hands are mostly involved. An acute glomerulonephritis with red blood cell casts, proteinuria and a decreasing renal function may develop. Vasculitis of the vasa nervorum can cause peripherial neuropathy. Meningoencephalitis can but seldom develop.
Diagnosis: symptomatically, by immune complex examinations, by serology (ELISA, Western blot), by other identification of the pathogens, by biopsy etc.
Differential diagnosis: by distinguishing it from autoimmune diseases.
Treatment: by removing the offending antigen and reducing the inflammation threatening the functioning of the organ. If a drug is suspected of causing immune complex disease, its administration should be immediately stopped. In case of patients with an immune complex disease associated with an infection, an adequate dose of appropriate antimicrobial drugs should be given. High doses of corticosteroids are usually given intravenously for about a week and then administered in form of tablets. By administering cyclophosphamide, azathioprine, etc. By plasmapheresis.
RFR method: detects and eliminates the pathogen microorganisms!
It can eliminate laboratory-identified and other pathogen microorganisms! If the nephritic syndrome is associated with *HIV* the first step to be done is to eliminate the infection. As to the frequencies of HIV, see its special Chapter.

23.24.1. Wegener's Granulomatosis

Wegener's granulomatosis (WG) is caused by a chronic, allergic-autoimmune response resulting in an inflammation of the lining of the airways (the nose sinuses, throat and lungs) and also in an inflammation of the blood vessels (vasculitis) and in an often fatal kidney disease. This illness can occur at any age and is twice as common in men as in women.
WG is characterized by vasculitis and granulomatosis, which ultimately destroy the affected tissues. The upper respiratory tract, i.e. the nose, sinuses, trachea is first affected causing severe nose bleeding, sinusitis, otitis media, laryngitis, fever, malaise, loss of weight, myalgia, myocardial damages and arthralgia. The trachea, bronchi and the lung parenchyma develop granulomatous masses, and those in the lungs may get cavitated [redacted] WG may get generalized, disseminated, resulting sores of the skin spreading extensively, and healing with scars. Kidney damages range from mild impairments to life-threatening kidney failures. Focal necrotizing glomerulitis and necrotizing vasculitis of the small arteries can be observed in the kidneys, causing

hematuria and proteinuria. Necrotizing vasculitis with granulomas can occur in every organ.
Diagnosis: symptomatically, by clinical laboratory examinations, biopsy, x-ray, CT, etc.
Treatment: symptomatically, by administering corticosteroids or/and immunosuppressants.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances are: 311, 339-344, 370-383, 409-411, 422, 442-444, 447-451, 446-450, 518 kHz

23.24.2. Churg-Strauss Syndrome

Churg-Strauss syndrome is an immune-mediated vasculitis affecting the medium and small vessels and causing their necrosis. It attacks mainly the blood vessels of the lungs, the gastrointestinal system, peripheral nerves, but can affect also the heart, skin and kidneys and is associated with antibodies to neutrophil cytoplasmic antigens (ANCAs [redacted] Chapter 12.1.2).
The criterion of the establishing the diagnosis of Churg-Strauss syndrome are the symptoms of asthma (wheezing, prolonged expiration), the presence of eosinophilia of more than 10% of the peripheral blood, paranasal sinusitis, pulmonary infiltrates (may be transient), the histological proof of vasculitis with extravascular eosinophils, and mononeuritis multiplex or polyneuropathy.
Hypergammaglobulinemia, increased levels of immunoglobulin E (IgE), rheumatoid factor positive and ANCA positive blood tests all point to the autoimmune character of this illness.
Symptoms of this disease include malaise, fatigue, flu-like symptoms, loss of weight, fever, myalgia, asthma symptoms with pulmonary vasculitis, paranasal sinusitis, allergic or immune rhinitis, arthralgia, skin eruptions such as purpura, nodules, urticarial rash, and necrotic bullae. Its cardiac involvements are heart failure, myocarditis, pericarditis, constrictive pericarditis and myocardial infarction. Peripheral neuropathy, stroke, ophthalmologic involvement but rarely occur.
Churg-Strauss syndrome is an allergic/autoimmune reaction to an environmental agent or drug, causing the absorption of antigen-antibody complexes on vessel walls. *Mycoplasmas* play an important role in these processes. The effect of Nanobacteria on this process is not cleared yet. Churg-Strauss syndrome indicates an immunoregulatory defect associated with vasculitis and eosinophilia, which is caused by a combined chronic infection. *HTLV, HBLV, Mycoplasma fermentans or Mycoplasma pneumoniae, RSV* and other bacteria and viruses together can develop this immunological process.
Diagnosis: by immunological examinations, CT, MRI, x-ray, ECG, EMG and biopsy. The characteristic pathologic changes, found especially in the lungs, include small necrotizing granulomas, as well as necrotizing vasculitis involving small arteries and venules. The granulomas are composed of a central eosinophilic core surrounded radially by macrophages and epithelioid giant cells. Glomerulonephritis is caused by the tissue deposition of immune complexes.
Treatment: by administering glucocorticoids. IVIG, interferon alpha, rituximab, cyclophosphamide and plasma exchange can be beneficial.
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequent resonances in case of Churg-Strauss syndrome are: 299, 315, 321-325, 328-330, 339, 370-376, 378-387, 402, 407-412, 432-433, 442-451, 470, 493-497, 518-519, 523-525 kHz

23.24.3. Behçet's Syndrome

Behçet's Syndrome (BS) is a chronically relapsing inflammatory disease, which can recurringly cause painful mouth sores, skin blisters, genital sores and swollen joints. Its other manifestations include arthritis, thrombophlebitis, neurologic abnormalities, inflammation of the eyes, blood vessel damages and gastrointestinal inflammations with severe cramping and diarrhea. Some of the above mentioned clinical features are at any given time experienced. This disease affects mostly people thirty years old, but can occur at any age. Men are affected more often than women. People living in Mediterranean countries, Middle East, Korea, Japan and the area along the silk route through China are at highest risk. BS is caused by combined infections, primarily bacterial, secondarily viral and autoimmune components also play a role.
Its predominant histopathological lesion is vasculitis, affecting mainly the small to medium-size arteries and veins. The walls of the blood vessel and the perivascular tissues are infiltrated predominantly by lymphocytes and plasma cells. The lumen of the vessels may be narrowed or obliterated. Fibrinoid necrosis and the disruption of the vessel walls can also be experienced.
Symptoms: are recurrent, painful mouth sores, similar to canker sores, developing first. Sores on the penis, scrotum and vulva are painful; those in the vagina may be painless. Its other symptoms, f.i. recurring painful inflammation of the eyes, sensitivity to light, and hazy vision can appear weeks or years later. Several other eye problems can also occur, causing blindness if untreated.
The symptoms or/and symptom-free periods may last for weeks, years or even decades depending on the patient's immune reactivity. Paralysis is a potential complication. Certain damages of the nervous system, gastrointestinal tract and blood vessels can be fatal.
Diagnosis: by physical examinations, BS can not be detected by laboratory tests.
Differential diagnosis: by distinguishing it from Reiter's syndrome, Stevens-Johnson syndrome, SLE, Crohn's disease and ulcerative colitis.
Treatment: there is no specific therapy known. Symptomatically, by administering corticosteroids and immunosuppressive drugs.
RFR method: detects and may eliminate the pathogen microorganisms.
There are many different pathogenic frequencies to be found in case of BS.
The most frequent resonances are: 291-293, 342-350, 363, 370-385, 426, 442-444, 447-451, 486, 493-495 kHz

23.25. Lymphadenopathy

Lymphadenopathy is a term used for the disease of lymph nodes. If the infection affects the lymph nodes themselves, it is named lymphadenitis, but if it attacks the lymph channels, it's name is lymphangitis.
The infected lymph nodes become usually enlarged, warm and tender. A swelling of lymph nodes due to the growth of the lymph cells is named lymphadenopathy.
Regional lymphadenopathy involves the enlargement of one single node or that of multiple contiguous nodi of a region. Lymph nodes are clustered in groups throughout the body and are concentrated in the head and neck, axillae, mediastinum, abdomen, and along the vascular trunks of the extremities. Generalized lymphadenopathy is defined as the enlargement of more than 2 noncontiguous lymph node groups. An exact history of patient and a thorough physical examination are necessary to establish a diagnosis. The causes of generalized lymphadenopathy include infections, autoimmune diseases, malignancies, histiocytoses, storage diseases, benign hyperplasia and drug reactions.
Generalized lymphadenopathy is most often associated with systemic viral infections. An *infectious mononucleosis* results in a widespread adenopathy. Roseola infantum (caused by *Human Herpes Virus-6), CMV, varicella, rubella, herpes, measles, Coxsackie viruses and adenoviruses* cause all generalized lymphadenopathy. *HIV* is often associated with generalized adenopathy, which can be its first characteristic sign. Bacterial infections are

usually associated with localized lymph node enlargements. Some bacterial infections, f.i. with *Salmonella typhi, Treponema pallidum, Yersinia pestis, Mycobacterium tuberculosis, Bartonella henselae, Staphylococcus aureus* and *Streptococcus pyogenes* can cause generalized adenopathies, too. Less common bacteremia, including those caused by endocarditis, result in generalized lymphadenopathies f.i. in case of *anthrax, human African trypanosomiasis, toxoplasmosis. Coccidioidomycosis* and *histoplasmosis* may cause mediastinal or generalized lymphadenopathy.
Lymphomas are mostly associated with regional lymphadenopathy, though there often occur a generalized lymphadenopathy in case of hematologic malignancies. Acute leukemia types and lymphomas show nonspecific signs like fever, anorexia, pain, sweating at night, etc.
Firm, non-mobil, matted lymph nodes can be found mostly in association with malignancies.
Generalized lymphadenopathy is an important manifestation of lipid storage diseases. Sphingomyelin and other lipids accumulate in the spleen, liver, lymph nodes, and the central nervous system in case of Niemann-Pick disease. Gaucher disease is characterized by the accumulation of glucosylceramides leading to the engorgement of the spleen, lymph nodes and the bone marrow.
Mycoplasmal, HTLV and HBLV infections can be the cause of generalized lymphadenopathies experienced in case of autoimmune diseases, f.i. SLE and RA.
Adverse drug reactions, f.i. owing to administered phenytoin, phenylbutazone, allopurinol, isoniazid, etc. can also be the cause of generalized lymphadenopathy.
Diagnosis: by laboratory examinations, CT, ultrasound, biopsy and histological examinations. By serology of the suspected infective agents (HIV, EBV, CMV, Toxoplasma, etc)
Treatment: depending on the specific etiology of the lymphadenopathy.
RFR method: detects and may eliminate the pathogen microorganisms.
As to the frequencies of these pathogens see their special Chapters.
RFR method is a most useful method for treating inflammations caused by infections, moreover, it is a useful method in the treating of malignant processes. In case of generalized, lymphadenopathy caused autoimmune processes look for mycoplasmas. In case of bacterial infections use RFR method combined with antibiotics.
The treatment of malignant processes usually lasts for a long time.

23.26. Amyloidosis

Amyloidosis is a disorder caused by various chronic infections or persistent inflammations; in case of which, amyloid, a protein normally not present in the body, accumulates in various tissues. Transthyretin (TTR) is one of the 20 proteins that form human amyloid fibrils. Systemic amyloidoses are designated by a capital A (for amyloid) followed by the abbreviation for the chemical identity of the fibril protein. Thus, for example, TTR amyloidosis is abbreviated ATTR, and amyloidosis of the immunoglobulin light chain type is abbreviated AL. Normal-sequence TTR and variant-sequence TTR form amyloidosis. Normal-sequence TTR forms cardiac amyloidosis among elderly people and is termed senile cardiac amyloidosis (SCA).
If the peripheral nerves are affected predominantly, the disease is termed familial amyloidotic polyneuropathy (FAP), if the heart is involved predominantly, the disease is named familial amyloid cardiomyopathy (FAC). Regardless of the organ involved, the general term is amyloidosis-transthyretin type (ATTR).
The morbidity and mortality of amyloidosis depends on the type of a present TTR variant. Some variants cause clinical diseases in all gene-carrier patients aged about 40 and will prove to be fatal within a few years after the onset of the symptoms. Other variants cause

mild diseases of later onset, while some carriers of the variant genes remain asymptomatic until late in life.
The morbidity of amyloidosis depends on the organs involved. In case of *chronic Coxsackie viral infections* neuropathy or/and cardiomyopathy do often develop. The most common immediate cause of death is caused by cardiac failure or fatal arrhythmia.
Among people carrying the same TTR variant, the clinical picture varies widely. The development of amyloidosis is caused by immune responses to different infections and the genetic factors significantly influence the course of the disease.
Amyloidosis can be systemic (affecting different organ systems) or organ-specific. Primery amyloidosis forms are inherited due to the mutations of the precursor protein. The secondary forms occur due to different diseases causing an overabundant or abnormal protein production, f.i. the over production of immunoglobulin light chains in case of multiple myeloma (termed AL amyloid), or the continuous overproduction of acute phase proteins in case of chronic inflammations (which can produce AA amyloid).
The precursor protein of AA amyloidosis is the serum amyloid A protein (SAA), an acute-phase protein associated with chronic inflammations. AA amyloidosis can occur in diseases associated with chronic inflammation, f.i. Rheumatoid arthritis, Familial Mediterranean fever, etc.
Neurological amyloid develops in case of
Alzheimer's disease
Parkinson's disease
Huntington's disease
Transmissible spongiform encephalopathies caused by prion protein
Creutzfeldt-Jakob disease.
Cardiac amyloidosis is present in case of
Senile cardiac amyloidosis-
Other:
Amylin deposition can occur in the pancreas in some cases of type 2 diabetes mellitus.
Diagnosis: by biopsy and examinations with Congo red staining with polarized light microscope, by immunostaining methods and radionuclide SAP scan.
Treatment: if amyloidosis is caused by another disease, the treatment of this disease usually slows or reverses the development of amyloidosis, multiple myeloma excepted. Symptomatically by administering corticosteroids, melphalan and colchicine.
RFR method: detects the primary infections and eliminates the causative pathogens.

23.27. Sarcoidosis

Sarcoidosis (also named Besnier-Boeck disease) is a multisystemic inflammatory disease with autoimmune-like characteristics and multifactorial etiology. It usually affects young adults of both sexes all over the world, its incidence being highest for persons in the age-group from 20-29 years, and has a second peak affecting women over 50. This illness *affects mostly the lungs and the intrathoracic lymph nodes*, the skin, the eyes and the brain, but can be systemic and present anywhere in the organs as well. Sarcoidosis is characterized by the presence of noncaseating granulomas (NCGs) in the affected tissues.
The cause of sarcoidosis is a genetic predisposition and a combined infection caused by *HTLV, HBLV, Mycoplasma fermentans*, and several different other viruses and bacteria, such as Corynebacterium, Mycobacteria, Chlamydia species, and frequently EBV or CMV. The coinfective microorganism may influence the localization and manifestation of the illness.
Genetic predisposition: gene BTNL2 is supposed to play a role in the development of this disease, although several HLA-DR risk alleles are also suspected. In case of a persistent sarcoidosis HLA-B7 and HLA-DR15 haplotypes or another gene between these two loci is associated with the disease. In case of a non-persistent sarcoidosis there is a strong genetic

association with HLA DR3-DQ2. Siblings have only a modestly increased risk to develop the disease, indicating that genetic susceptibility plays only a small role in its development. The alternate hypothesis that family members are similarly exposed to environmental pathogens is quite plausible for explaining its being present in the family.
Symptoms: The illness is sometimes *asymptomatic*, but, depending on the affected organs, it can have symptoms.
The most common symptoms of this disease are fatigue, weight loss, arthralgia, dry eyes, blurry vision, shortness of breath, a dry hacking cough and skin lesions. Sarcoidosis can manifest itselt *in different cutan forms*, f.i. annular, papular, erythrodermic, ichthyosiform, ulcerative, hypopigmented, and can be scar sarcoid, or morpheaform skin lesions, in a form named lupus pernio, microscopically resembling lupus vulgaris. Renal, heart and/or brain involvement and liver portal hypertension may cause further symptoms and altered functioning.
Sarcoidosis affecting the brain and the peripheral nerves is named *neurosarcoidosis* which mostly affects the cranial nerves. Sarcoids can also occur in the thyroid gland. The *ocular sarcoidosis* affects about 25-60% of patients with systemic sarcoidosis and can cause severe visual impairment and even blindness. The most common ocular manifestations are uveitis and conjunctival nodules.
Heerfordt-Waldenström Syndrom is a peculiar form of sarcoidosis characterized by the enlargement of the parotid glands, mild fever, anterior uveitis and facial nerve palsy.
Löfgren's syndrome is characterized by the acute sarcoidosis of the lungs, erythema nodosum, migratory polyarthritis, fever (and iritis).
Mikulicz syndrome consists of bilateral sarcoidosis of the parotid, and the submandibular, sublingual and lacrimal glands.
Darier-Roussy type sarcoidosis (also named subcutaneous sarcoidosis) is characterized by multiple subcutaneous sarcoid nodules on the trunk and extremities.
Lymphocytic meningitis is one of the most common neurologic manifestations and there may occur cranial nerve palsies and hypothalamic/pituitary dysfunctions.
Human T lymphocyte virus-infected T cells play a central role in the development of sarcoidosis, as they likely propagate an excessive cellular immune reaction with elevated IFN, TNF and TNF receptors. The presence of other viral and mycoplasmal antigens influencing T and B cells does also play a role. The hyperreactivity of B cells producing immunoglobulins is evident. Antigen-presenting cells also accumulate at the involved loci of sarcoidosis. Finally, the level of fibrinogenic cytokines gets increased.
Prognosis: patients suffering from sarcoidosis can be healed without being treated, or can be cured within 12-36 months, most of them within 5 years. Nevertheless, some cases persist for several decades.
Diagnosis: by x-ray examinations, electrocardiogram, ocular examination, liver function tests, serum calcium and 24-hour urine calcium examinations. In case of female patients sarcoidosis is significantly associated with thyroid diseases. The level of the Serum ACE (angiotensin converting enzyme) can help to diagnose and to monitor the disease as it tends to rise and fall according to the activity of the disease.
Treatment: if necessary, by administering corticosteroids, though some patients do not respond to steroid therapy. As the granulomas are caused by collections of immune cells, particularly T cells, immunosuppressants can help successfully.
RFR method: can detect the pathogen frequencies and eliminate the causative agents.
The most frequent resonances are: 370-376, 442-451 kHz
Other resonant frequencies are: 297-299, 311, 315-317, 321-324, 339-344, 446-451, 372-381, 383-390, 406-410, 430-434, 447-451, 453-455, 467-490, 393-497, 519-519, 526-530 kHz

23.28. Vogt-Koyanagi-Harada Syndrome

Vogt-Koyanagi-Harada syndrome (VKH) is a rare systemic disease involving various melanocyte-containing organs. Bilateral uveitis associated with cutaneous, neurologic, and auditory abnormalities does characterize this syndrome. The clinical course of VKH syndrome occuring with influenza-like symptoms suggests a viral or postinfectious origin. Other clinical and experimental data support an immunologic-autoimmunologic etiology.
Pathogenesis: VKH syndrome has an infectious-immune-autoimmune pathogenesis.
Genetical predisposition: Although almost all the cases of VKH syndrome are sporadic and familial cases are rare, some authors suggest that the illness may be inherited, probably in an autosomal recessive manner. Numerous data demonstrate the association of HLA-DR4 antigen with the VKH syndrome in different racial groups and in various countries.
An autoimmune reaction seems to be directed against an antigenic component shared by uveal, dermal and meningeal melanocytes, which antigen is possibly a tyrosinase or a tyrosinase-related protein. Circulating antibodies against a retinal photoreceptor region was detected in patients suffering from this disorder. An autoimmune reaction to melanocytes with the involvement of T-cell–mediated cytotoxicity and apoptosis can possibly occur.
VKH syndrome has an *infectious* origin. The most frequent infectious agents include:
Viral components, such as *Coxsackie viruses, Influenza viruses, CMV, EBV, HTLV, HPV, HSV1* or/and *2, VZV, Measles* and *Adenoviruses.*
Mycoplasmal components can be: *M. fermentans, M. penetrans* or *other M. species.*
Symptoms: VKH syndrome is usually preceded by a prodromal phase of nonspecific symptoms including headache, vertigo, nausea, nuchal rigidity, vomiting and low-grade fever that may last for a few days. Viral antigens will be bound to the cell membranes and provoke an immune-autoimmune pathological response.
VKH syndrome consists of 3 phases:
The first one is the infectious phase with meningoencephalitis: the degree of neurologic symptoms can vary and be f.i. generalized muscle weakness, hemiparesis, hemiplegia, dysarthria, inflammatory arachnoiditis resulting in subarachnoidal adhesions. Mental changes ranging from mild confusion to psychosis may occur.
The second, the ophthalmic-auditory phase is characterized by common features such as a decreased visual acuity, eye pain, eye irritation, loss of vision, dysacusis and tinnitus.
The third, the convalescent phase is characterized by cutaneous signs developing after the uveitis begins to subside, usually within 3 months from the onset of the disease. Pigmentary changes tend to be permanent.
VKH syndrome has 4 diagnostic criteria:
Cerebrospinal fluid pleocytosis or evidence of tinnitus and dysacusis,
Headache, meningismus, or cranial nerve involvement;
Bilateral iridocyclitis, posterior uveitis, which may include exudative retinal detachments, optic nerve swelling or atrophy of the retinal pigment epithelium;
Cutaneous signs of vitiligo, alopecia and poliosis.
Diagnosis: symptomatically, based on the 4 diagnostic criteria.
Treatment: symptomatically.
RFR method: detects and can eliminate all pathogen infectious components.
The most frequent resonances are: 288-302, 307-321, 332-334, 370-383, 402, 408-411, 416-420, 442-451, 454, 518-519, 534-544, 576-581 kHz

24. ENDOCRINE DISEASES CAUSED BY VIRAL AND BACTERIAL INFECTION

Viruses, which can live in the epithelial cells, or in the central nervous system can cause various endocrine diseases. Characteristically, endocrine abnormalities arise as a consequence of an increased or decreased hormone secretion. The clinical manifestations of the affected patients derive mostly from the excessively or deficiently secreted hormones. Serious endocrin disorders will result only in cases, in which the servoregulating mechanism, or the feedback response of the person fails to stimulate appropriately the reactions of the trophic gland. Endocrine abnormalities may also develop if the peripheral target tissues are unable to respond to the normal hormone level. Heightened tissue susceptibility to hormone action, caused by an inherited viral infection is the determining factor of some endocrinopathies.

Homeostasis and adaptation of the organism to its environment require a continual monitoring of the internal and external milieu. The neurosecretory neurons of the hypothalamus are regulated only by neural inputs. The hypothalamus and possibly other regions of the brain as well are responsible for the feedback effects of the circulating hormones, such as thyroxine, cortisol, testosterone, estrogen and progesterone. These effects, which include negative and positive regulatory influences, act to determine the degree of the hypothalamic activity. The circulating hormones act also upon the pituitary gland in order to determine the pituitary sensitivity to the hypothalamic hormones.

24.1. Diabetes Mellitus

Diabetes mellitus (DM) is a chronic metabolic disorder caused by an absolute or relative insulin deficiency. Insulin, an anabolic hormone, is produced by the beta cells of the islets of Langerhans located in the pancreas. The absence, destruction, or some other loss of these cells result in type 1 diabetes (i.e. in insulin-dependent diabetes mellitus, IDDM). Most children suffering diabetes have IDDM and a for a lifetime lasting dependence on exogenous insulin.

Type 2 diabetes (i.e. non–insulin-dependent diabetes mellitus, NIDDM) is a heterogeneous disorder. Most patients with NIDDM are resistant to insulin, their beta cells lack the ability to overcome this resistance. Although this form of diabetes was previously uncommon among children, in some countries 20% or more of new patients with diabetes in childhood and adolescence have NIDDM, which change in its prevalency is associated with the increased prevalence of obesity. Moreover, some patients may have inherited disorders of insulin release leading to a maturity onset diabetes of the young (MODY).

The etiologic classification of the types of diabetes is as follows:

1. Genetic (hereditary, idiopathic, primary, essential) diabetes, subdivided according to the age of the patient at the time of the onset and/or the severity of the illness (juvenile and adult diabetes types).
2. Pancreatic diabetes, in case of which the carbohydrate intolerance can be attributed directly to the destruction of the pancreatic islets caused by a chronic viral inflammation, carcinoma, hemochromatosis, or surgical removal.
3. Endocrin diabetes, where the disease is associated with endocrinopathies, such as hyperpituitarism, hyperthyroidism, hyperadrenalism, and pancreatic islet-cell tumor of A-cell type. Gestational diabetes and stress diabetes can also be included in this group.
4. Iatrogenic diabetes, caused by administered corticosteroids, certain benzothiadiazine type diuretics, estrogen-progresterone combinations, etc.

The classification of diabetes according to an other standpoint is:

1. Type I diabetes mellitus (IDDM)
2. Type II diabetes mellitus (NIDDM), which is a receptor diabetes. Type II diabetes also tends to run in families, due to an inherited viral infection.

Insulin is essential to process carbohydrates, fat and protein. It allows glucose to enter the cells and stimulates the conversion of glucose to glycogen (glycogenesis) reducing thus the level of blood glucose. Insulin inhibits the glycogenolysis in the liver, slows down the breakdown of fat into triglycerides, free fatty acids and ketones. It stimulates the storage of fat and inhibits the breakdown of protein and fat into glucose in the liver and kidneys.
In case of insulin deficiency hyperglycemia (i.e. the random blood glucose concentration is more than 200 mg/dl/or 11 mmol/l) will be present. The kidneys will be unable to reabsorb the excess glucose load, causing glycosuria, osmotic diuresis, thirst and dehydration. The increased fat and protein breakdown enhances the ketone production and leads to loss of weight. Without insulin one can die due to diabetic ketoacidosis.
The most important cause of diabetes is a combined viral *(Coxsackie B4, Human T-cell Lymphotropic Virus),* and *Mycoplasma fermentans* infection, which destroys the beta cells of the pancreatic islets of Langerhans. Their interaction leads to the development of an autoimmune disease directed against the insulin-producing cells of the pancreatic islets of Langerhans. These cells will be progressively destroyed. Insulin deficiency can develop in case of a destruction of more than 90% of these cells.
Genetic factors play an evident role in the etiology of IDDM.
The frequency of diabetes developing among children of diabetic mothers is 2-3% while 5-6% if it is the father who suffers from IDDM. The risk of getting diabetes as a child rises to about 30% if both parents are diabetic.
HLA-DR3 and HLA-DR4 haplotypes are strongly associated with IDDM. More than 90% of white peoples with IDDM express 1 or both of these molecules, compared to 50-60% of the general population.
Patients owing HLA-DR3 haplotype are at a greater risk to develop other autoimmune endocrinopathies and celiac disease as well. They are likely to develop diabetes at a later age, have islet cell antibodies and have a longer period of residual islet cell function.
Patients with HLA-DR4 haplotype are usually younger at the time of being diagnosed, are more likely to have insulin antibodies, and are not likely to have other autoimmune endocrinopathies.
Environmental factors can play an important role in developing diabetes, infections and diet are considered to be the most likely environmental factors.
Viral infections are the most important environmental factors of the development of IDDM, as they initiate an autoimmune process. Instances have been reported of the direct toxic effect of *connatal rubella* infection. A recent survey suggests that an *enteroviral infection* during pregnancy carries an increased risk of IDDM concerning the offspring. Paradoxically, the incidence of IDDM is higher in areas where the overall burden of infectious disease is lower.
Dietary factors are also relevant. Breast-fed infants have a lower risk to suffer IDDM, while there exists a direct relation between cow milk consumption and the incidence of diabetes. Some cow-milk proteins (f.i. bovine serum albumin) are antigenically similar to certain islet cell antigens.
Diabetes mellitus can develop as a consequence of an imbalance between production and release of insulin, as well as caused by hormonal and tissue factors modifying the insulin requirement.
Insulin can be absolutely lacking in cases of **secondary diabetes** caused by the destruction or removal of the pancreas.
An **overt juvenile-onset diabetes** similarly shows insulin deficiency, characterized by unextractable pancreatic insulin, no response to oral hypoglycemic agents of sulfonylurea

type, a marked tendency to ketoacidosis and thus by the dependence on exogenous insulin for survival. It is assumed that a child's diabetes begins when the pancreatic production of insulin is declining. This disease is not in every case irreversible, as at least one third of all juvenile diabetics will get a transient remission, usually within 3 months after getting an *acute Coxsackie B 4 viral infection* after having had a *HTLV 1-6*, and/or a *Mycoplasma fermentans* infection already at the onset of the disease. This remission may last several days to several months, rarely exceedig one year. There is no insulin treatment necessary at the time of such a remission, even the result of a glucose tolerance test may be normal. Patients should at this time be treated with the RFR method! Nevertheless, after this remission the juvenile diabetic child reaches rapidly a state of total insulin deficiency and an autoimmune process will be iniciated. The inhibition of this autoimmune process is very difficult, being caused by *Mycoplasma fermentans*, and *HTLV 1-6* (see their special Chapters).

A patient with **maturity-onset diabetes of the young** gets diabetic considerably more slowly. At the early stage there may be no symptoms present, the diagnosis is suspected by the discovery of elevated blood glucose levels 1 or 2 hs postprandially. Measurements of serum insulin may indicate close to normal fasting blood sugar levels;, the insulin response to administered glucose is abnormal by being delayed. As the insulin release increases simultaneously with the rising of blood glucose, the blood glucose will significantly decline due to the delayed but excessive amount of insulin, provoking the symptoms of reactive hypoglycemia between the third and fifth hours postprandially. The disease progressing further, the insulin release becomes less pronounced, the episodes of reactive hypoglycemia will disappear; while finally the amount of circulating insulin will be insufficient to return the blood glucose to normal levels between meals. In case of MODY the reserve of pancreatic insulin is decreased but rarely totally absent, so that the occurrence of diabetic ketosis is uncommon. Similarly to juvenile onset diabetes, MODY may be caused by a *combined viral infection.*

Symptoms of type II diabetes include blurred vision, drowsiness, nausea and a decreased endurance when exercising. Poorly controlled diabetic patients are more susceptible to infections.

The symptoms of type I diabetes begin abruptly, may progress rapidly to diabetic acidosis caused by the break down of fat. The initial symptoms of diabetic ketoacidosis include excessive thirst and urination, loss of weight, nausea, vomiting, fatigue and abdominal pain.

Complications of diabetes are coma, and diabetic micro and macro angiopathy. The most serious of them are retinopathy, nephropathy, neuropathy and gangrene.

Diabetic macro and micro-angiopathy: Diabetic microangiopathy includes capillary degeneration leading to the leakage of fluid from the vessels causing edema. In case of retinopathy the earliest lesions recognizable by fundoscopy are the dilatation of veins and microaneurysms consisting of small punctuate hemorrhages. Patients with long-term juvenile diabetes may develop proliferative retinopathy with neovascularization.

Diabetic nephropathy can cause a certain type of hypertonia. The specific diabetic nodular glomerulosclerosis consisting of discrete ball-like PAS-positive masses in the mesangial regions of the capillary tufts as well as a diffuse glomerulosclerosis was first described by Kimmelstiel and Wilson. These diffuse lesions can usually cause hypertension, proteinuria or a nephrotic syndrome.

The diabetic neuropathy is a frequent, distressing complication of diabetes, involving mostly the peripheral nerves, though it may involve any part of the nervous system owning an almost unlimited range of manifestations. Peripheral diabetic neuropathy is characterized by a nonsegmental distribution. If an intracranial aneurism can be ruled out by angiogram, diabetic neuropathy may be the cause of the symptoms.

If this neuropathy is in relation to sexual functions it is a disturbing complication for diabetic men. Diabetic neuropathy is most difficult to treat.
An important characteristic sign of diabetes is the **susceptibility to different bacterial, viral and fungal infections**, causing several symptoms. This susceptibility of diabetic patients is caused mostly by the immunomodulating effect of a concommittant *mycoplasmal or/and HTLV infection* (the latter being usually caused by *HTLV-I*)[redacted]
Haemophilus influenzae B (HIB) infection and *vaccination* may be a great risk factor for juvenile onset diabetes mellitus (i.e. IDDM or type 1).
Diagnosis: by laboratory tests.
Differential diagnosis: by determining the type of diabetes.
Treatment: by insulin replacement therapy, by administering antidiabetic drugs, (Symptoms of low blood sugar levels are: sudden severe hunger, headache, anxiety, tremor, sweating, confusion and coma). By symptomatic treatment of ketoacidosis, diabetic angiopathy, retinopathy and neuropathy.
RFR method: detects and eliminates the virus, as well as other pathogens!
The most frequent other resonances of Diabetes mellitus type-I are: 306-308, 361-365, 370-374, 442-451, 493-495 kHz
The most frequent other resonances of Diabetes mellitus type-II are: 370-374, 420-426, 442-451, 493-495, 534-544 kHz
The most frequent other resonances of Diabetes mellitus mixed type are: 306-308, 361-365, 370-374, 420-426, 442-451, 493-495, 534-544 kHz
The most frequent resonances of the Coxsackie virus B4 are: 307-308, 360-366, 419-426, 430, 534-544, 552-554 kHz
The most frequent resonances of HTLV-1 are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz
The most frequent resonances of Mycoplasma fermentans are: 312, 329, 353, 361, 404, 442-451, 493-495, 505 kHz
The most frequent resonances of HIB diabetes are: 306-308, 335-338, 361-365, 370-374, 424-426, 442-451, 534-536 kHz
The most frequent secondary infections are usually caused by:
Staphylococcus: 327-331, 345, 372, 377-386, 397, 402-403, 425, 434, 445, 462, 482, 491, 537, 557, 562-567 kHz
Streptococcus: 310-315, 337-340, 364, 372-389, 397-403, 432-434, 442, 450, 454, 476, 478, 486, 516-520, 542, 576 kHz
Candida: 338, 379, 384-390, 422, 443-453, 460-466, 520, 570-580 kHz
Pseudomonas: 324, 331-335, 351, 374, 380, 396-397, 401, 438, 447, 579 kHz
Epstein-Barr Virus: 337-342, 372-382, 518 kHz
Cytomegalovirus: 305, 349, 407-412, 534 kHz
As to other infections, see their special Chapters.
Due to the effect of RFR method the necessary amount of the applied insulin will decrease, so that the applying of insulin will eventionally become unnecessary. After the elimination of the pathogens the total regeneration process of beta cell function may take about a year. Beta cells develop from stem cells.

24.1.1. Diabetes Mellitus and Zygomycosis

Zygomycosis is an infection caused by fungi of the class of Zygomycetes, which consists of orders of Mucorales and Entomophthorales. Mucormycosis does also belong to the zygomycotic group of diseases.
These fungi are ubiquitous and generally saprophytic, which but rarely infect immunocompetent hosts, but often cause invasive fungal infections to immunocompromised or diabetic patients (their immune damages are often caused by

Mycoplasma fermentans). Healthy people own a strong natural immunity to infections of zygomycetes, the risk factors for developing zygomycosis were nevertheless recognized several decades ago. Diabetes mellitus was held to be the major risk factor for developing rhinocerebral zygomycosis already many years ago. Gregory et al. were the first who described and published the first cases of fulminant rhinocerebral zygomycosis of diabetic patients.

The most common fungus causing zygomycosis is *Rhizopus arrhizus (Rhizopus oryzae)*, which is a Rhizopus species belonging to the Mucoraceae family.

Zygomycetes are relatively rarely isolated in clinical laboratories being either environmental contaminants or, less commonly, the causative fungi of a zygomycosis. While this disease is mostly linked to Rhizopus spp, some other species can also cause infections to people, f.i. *Mucor, Rhizomucor, Absidia, Apophysomyces, Saksenaea, Cunninghamella, Cokeromyces* and *Syncephalastrum spp.* Although *Mortierella spp.* do cause diseases of animals, they may also be human pathogens. The spores of these molds are transmitted by inhalation, via a variety of percutaneous routes, or by the ingestion of spores. Human zygomycosis caused by Mucorales generally occurs as an opportunistic infection of immunocompromised hosts. The risk factors of persons may be diabetes mellitus, neutropenia, sustained immunosuppressive therapy, for a long time administered prednisone, or broad-spectrum antibiotics, iron chelation therapy, severe malnutrition and the primary breakdown in the integrity of the cutaneous barrier caused f.i. by trauma, chirurgical intervention, needle sticks, or burns. The loci of the manifestations show the mode of transmission, rhinocerebral and pulmonary diseases are their most common manifestations. Cutaneous, gastrointestinal and allergic diseases can also be experienced.

Mucorales are associated with angioinvasive diseases, leading often to thrombosis, infarction of the involved tissues, and tissue destructions mediated by a number of fungal proteases, lipases and mycotoxins. If not early enough diagnosed, dissemination will most often occur. An effective therapy has to be started early, requires combinations of antifungal drugs, surgical intervention and the reversal of the risk factors present.

Entomophthorales are causing human diseases predominantly in tropical regions, their transmission occurring by implantation of spores via minor trauma, f.i. an insect bite or by inhalation of spores into the sinuses. *Conidiobolus* causes typically mucocutaneous infections, mostly sinusitis, while *Basidiobolus* infections are subcutaneous mycotic diseases of the trunk and extremities.

Entomophthorales are true pathogens, infecting primarily immunocompetent hosts. They generally do not invade blood vessels and rarely disseminate. Occasional cases of disseminated and angioinvasive diseases occuring among immunocompromised patients were recently described, suggesting the possibly emerging role of this fungus as an opportunist. The infection caused by this species is acute and rapidly fatal despite early diagnosis and treatment. These organisms have a particular predilection to invade major blood vessels, with ensuing ischemia, necrosis and infarction of adjacent tissues, resulting in the production of black pus. This infection plays an important role in the diabetic gangrenisation. Granulocytopenic and acidotic patients are particularly at risk. Zygomycetes have a propensity to affect acidotic patients, particularly those with diabetes. They infect also patients with acidosis secondary to renal insufficiency, diarrhea and aspirin intake. Patients on glucocorticoid or deferoxamine therapy and those with a previous splenectomy are also at risk.

The hallmark of this disease is angioinvasion, thrombosis, infarction and necrosis of the tissues involved. Among patients who are not neutropenic an acute inflammatory response is generally experienced. Thick, necrotic fluids may be aspirated from the affected areas. *Rhizopus spp.* are by far the most common organisms isolated from patients with zygomycosis, representing about 90% of all infections with zygomycetes.

Rhinocerebral zygomycosis is the most frequently encountered form of the disease, observed primarily, among patients with diabetic acidosis. The illness characteristically involves the nose, then the eyes, brain, and, occasionally, the meninges.
Patients suffer typically from fever, unilateral facial pain, headache, nasal congestion, epistaxis, visual disturbances and lethargy.
Physical examinations may reveal periorbital cellulitis, proptosis and the loss of the extraocular muscle movements. These lesions are frequently accompanied by cranial nerve palsy of the II., III., IV. and VI. nerves.
Black necrotic lesions are generally observed on the hard palate or nasal mucosa of such extremely ill patients.
Patients with **pulmonary zygomycosis** suffer typically from fever, cough, hemoptysis, chest pain and increasing shortness of breath. Physical examinations may reveal pleuritic rub and rhonchi over the affected area. Primary pulmonary zygomycosis tends to occur affecting patients with hematological malignancy or profound neutropenia and those with corticosteroid therapy.
Patients suffering from **gastrointestinal zygomycosis** have typically abdominal pain or distention, dyspepsia, nausea and vomiting, diarrhea and hematochezia. Physical examinations may reveal decreased bowel sounds, guarding or rebound tenderness, and localized or diffuse abdominal tenderness.
Gastrointestinal zygomycosis usually results if a patient who is malnourished or experiencing renal problems ingests the fungi. This infection results in necrotic ulcerations, with ischemia and gangrene of the stomach and colon.
Cutaneous zygomycosis is a primary disease due to local trauma or inoculation, and if occuring secondarily, the disease is caused by a hematogenous dissemination of fungi into the skin. Patients mention a previously got local trauma and pain around the locus of the trauma. The affected skin lesion will get indurated, erythematous and gradually develop into a necrotic ulcer (diabetic ulcer) with a characteristic, dark central area. The margins of the ulcer are sharply demarcated. Cutaneous zygomycosis can occur associated with trauma caused by Elastoplast bandages applicated on biopsy sites of diabeticers and burned patients previously colonizated by these fungi. Secondary cutaneous infections can occur by hematogenous seeding in case of disseminated zygomycosis.
A disseminated form arises usually from the lungs and can spread hematogenously to the **central nervous system**, in which case the zygomycotic patients can have headache, fever, visual disturbances and changes in their mental state. Lethargy, obtundation, coma, sudden onset of focal neurologic deficits and necrotic ulcerations of the mucosa of the respiratory tract or the skin can also come about.
Disseminated zygomycosis occurs among patients with hematological malignancies. It begins in the lungs and spreads to the CNS, producing there infarctions and abscesses. These fungi may spread also to the liver, spleen, kidney, heart and skin.
The majority of the affected patients are immunocompromised, although sporadic cases of noncompromised persons are also known.
Diagnosis: by identifying the yeast directly from the patient's cytologic specimens. By phase-contrast microscopy, by serology, PCR.
Differential diagnosis: by distinguishing it from Actinomycosis, Aspergillosis, Cryptococcosis, Nocardiosis and Toxoplasmosis.
Treatment: by starting an antifungal therapy as early as possible. Amphotericin B is the mainstay of the therapy. (Owing to its drug toxicity the monitoring if its adverse effects and toxicity is necessary.)
RFR method: detects and can eliminate the fungi.
The most frequent resonant frequencies are: 290, 295, 313-320, 334-337, 350-354, 364-368, 373-376, 384-389, 392-398, 446-455, 480-482, 484-488, 511, 574 kHz

24.2. Obesity

The accumulation of an excessive amount of body fat is named obesity, which is a public-health disorder in developed countries.

The body mass index (BMI), (known also as Quetelet index) is used commonly to define obesity. It is closely correlated with the degree of body fat.

BMI = weight/height^2, (weight is given in kilograms and height in meters).

The body fat percentage can be estimated by using the Deurenberg equation, i.e.

body fat percentage = 1.2(BMI) + 0.23(age)-10.8(sex)-5.4 (age is given in years and sex is 1 for male and 0 for female.

Although the BMI is correlated with the body fat percentage, in case of muscular persons their BMIs, indicating overweight or mild obesity, may be spurious.

The degree of obesity according to the criteria of WHO based on BMI are as follows:

Grade 1 overweight (i.e. overweight) is a BMI of 25-29.9 kg/m^2.

Grade 2 overweight (i.e. obesity) is a BMI of 30-39.9 kg/m^2.

Grade 3 overweight (i.e. severe or morbid obesity) is a BMI higher than or equal to 40 kg/m^2.

The regional fat distribution is in relation to certain comorbidities associated with obesity, f.i. the high abdominal fat content (including the visceral and subcutaneous abdominal fat) is more predictive of adipose-related comorbidities than the gynecoid obesity, which has a relatively peripheral, i.e. gluteal distribution.

The metabolically active adipocyte is the cellular basis for obesity. This adipocyte can be perceived as an endocrine cell with several peptides and metabolites that may control the body weight. Among its products are cytokines, such as TNF-alpha, IL-6, lipotransin, adipocyte lipid-binding proteins, acyl-stimulation proteins, prostaglandins, adipsin, perilipins, lactates, adiponectins, monobutyrins, and phospholipid transfer proteins.

Other important enzymes involved in the metabolism of the adipocytes are hormone-sensitive lipases, acylcoenzyme A, the endothelial derived lipoprotein lipases, synthetases and a cascade of enzymes.

The differentiation of preadipocytes to adipocytes occurs in white and brown adipose tissues as well, even in case of adults, and has a potential role in the development of obesity and the relapse to obesity after loss of weight. The pathogenesis of obesity is a complex process, where also a substantial genetic component takes part. Obesity is far more than the mere result of too much eating and/or too little exercised. Leptin, which is a 16-kD protein produced predominantly in the white adipose tissue plays a great role in the regulation of the body-weight by signaling satiety to the hypothalamus, reducing thus the dietary intake and fat storage, modulating the energy expenditure and carbohydrate metabolism to prevent gaining further weight.

Proopiomelanocortin (POMC) and alpha–melanocyte-stimulating hormone (alpha-MSH) act centrally on the melanocortin receptor 4 (MC 4) to reduce the dietary intake. Genetic defects in POMC production and mutations in the MC4 gene are monogenic causes of obesity of human beings. Recent data suggest that 5% of obese children have MC4 or POMC mutations.

Obesity related comorbidities

can be *cardiovascular*, f.i. essential hypertension, coronary artery disease, left ventricular hypertrophy, cor pulmonale, obesity-associated cardiomyopathy, accelerated atherosclerosis, pulmonary hypertension of obesity, venous varicosities, venous and/or lymphatic edema of the lower extremities.

can affect the *CNS*, f.i. stroke, idiopathic intracranial hypertension, meralgia paresthetica

can affect the *GI system*, f.i. gall bladder diseases (cholecystitis, cholelithiasis), nonalcoholic steatohepatitis (NASH), fatty liver infiltration, etc.

can affect the *respiratory system*, f.i. obstructive sleep apnea, obesity hypoventilation syndrome (Pickwickian syndrome), increased predisposition to respiratory infections,

malignancy, f.i. association with endometrial, prostate, gall bladder, breast and colon cancers,
psychologic disorders, f.i. social stigmatization, depression,
orthopedic diseases, f.i. osteoarthritis, coxa vera, slipped capital femoral epiphyses, Blount disease, Legg-Calvé-Perthes disease, chronic lumbago,
metabolic disoders, f.i. insulin resistance, hyperinsulinemia, diabetes mellitus type 2, dyslipidemia (characterized by high total cholesterol, high triglycerides, normal or elevated low-density lipoprotein, and low high-density lipoprotein)
reproductive disorders, f.i. anovulation, early puberty, infertility, hyperandrogenism, polycystic ovaries of women and hypogonadotrophic hypogonadism of men.
obstetric and perinatal disorders, f.i. pregnancy-related hypertension, fetal macrosomia, pelvic dystocia
immune deficiency and autoimmune predisposition.
The etiology of obesity is multifactorial, f.i. metabolic, genetic, endocrine, race, sexual and age-caused, ethnic and cultural, socioeconomic, psychologic factors, behavior, dietary habits, smoking cessation, etc can all play a role in its etiology.
Certain illnesses are related to secondary type obesity, f.i. hypothyroidism, Cushing's syndrome, insulinoma, PCOS etc.
Genetic syndromes associated with obesity are f.i. Prader-Willi syndrome, Alström syndrome, Bardet-Biedl syndrome, Cohen syndrome, Börjeson-Forssman-Lehmann syndrome and Fröhlich syndrome.
Obesity can develop also in a medication-related way, f.i. phenothiazines, sodium valproate, carbamazepine, tricyclic antidepressants, lithium, glucocorticoids, megestrol acetate, thiazolidine diones, sulphonylureas, insulin, adrenergic antagonists, serotonin antagonists oral contraceptives can all be related to it.
Researcher in the United States, Nikhil Dhurandhar univ. prof. published in the International Journal of Obesity his opinion about the adipogenic effect of *Human Adenovirus-36* on contributing to the obesity of patients in certain cases.
This Human Adenovirus-36 (HAdV-36) or **AD-36** is one of the 51 types of adenoviruses known to infect people. AD-36 is the only human adenovirus linked with human obesity, and is present in 30-40% of obese persons. An AD-36 infection can induce the cellular differentiation of the 3T3-L1 preadipocytes and stem cells derived from the human adipose tissue.
The pathogenesis of obesity is far more complex than the simple paradigm of an imbalance between energy (calorie) intake and energy (calorie) output.
Diagnosis: symptomatically, by body mass index, by examining high-density lipoprotein cholesterol (HDL-C) levels, by thyroid function tests, glucose and insulin tests etc.
Treatment: by using dietary programs with a very low-calorie diet, sporting, by administering medical treatment (f.i. anorexiants, sibutramine, orlistat, ephedrine, phentermine, etc), by surgery.
RFR method: detects the pathogen microorganisms.
The resonant frequencies of Human Adenovirus-36 are: 333-336, 340, 370-387, 390-392, 393, 394-400, 560-570 kHz

24.3. Hyperthyroidism

In case of hyperthyroidism the thyroid gland is hyperactive producing too much hormones. The most important forms of hyperthyroidism are the immune mediated hyperthyreosis (i.e. Graves' disease), the TSH-receptor hyperthyreosis (i.e. the autonomous hyperthyreosis), hyperthyreosis caused by thyreoditis and the central hyperthyreosis (i.e. if the hyperthyreosis is caused by a hypophysis adenoma overproducing THS).

Graves'disease, (named also Parry's or Basedow's disease), is a disorder of hardly know etiology causing hyperthyroidism with diffuse goiter, ophthalmopathy and dermopathy. These three major manifestations do not need to be present at the same time. Hyperthyroidism, which merely reflects an excessive supply of thyroid hormones to the tissues, can arise also in case of various other circumstances distinctly different from Graves' disease. It is very important to know, that a patient with **thyroiditis,** which is the inflammation of the thyroid gland, gets typically through a phase of hyperthyroidism. The inflammation may damage the thyroid gland, so that its initial overactivity is but a prelude to a transient or a permanent hyperactivity causing hyperthyroidism. **Toxic thyroid nodules,** i.e. abnormal tissue growths within the thyroid gland, sometimes escape the mechanisms normally controlling the thyroid gland and produce thus thyroid hormones in large quantities. In case of hyperthyroidism, certain functions of the body speed up, f.i. the heart pounds, beats more quickly and sometimes arrhythmically. The blood pressure is likewise increased. The patients's skin can become moist by sweating profusely, their hands may tremble. Most patients feel nervous, tired and weak, and have yet an increased level of activity. Despite an increased appetite, they lose weight. Older people with hyperthyroidism may not develop these characteristic symptoms but have apathetic or masked hyperthyroidism. Hyperthyroidism can cause puffiness around the eyes, an increased tear formation and an unusual sensitivity to light. These eye symptoms disappear soon after the thyroid hormone secretion gets normal, except in case of Graves' disease, which causes special eye problems.

Graves' disease is a relatively common disorder, may occur at any age, but mostly occurs among women of 30-40 years old. Patients with Graves' disease.have a distinct familial predisposition to this illness. I suppose, its cause is a microorganism present in the family and when the immune response of the person decreases the disease gets manifested [redacted] 23:18.)

The histopathology of Graves' disease is characterized by a parenchymal hyperplasia usually accompanied by lymphatic infiltrations that may reflect the cell-mediated immune origin of the disease or may merely reflect the chronic thyroiditis. The substances of the microorganisms are absorbed to the cell membranes, so that they change the antigenity of the cell membranes. This alteration provoke an immune response. Graves' disease is associated with generalized lymphoid hyperplasia and infiltration, and occasionally also with the enlargement of the spleen and thymus, which pathologies can all be caused by a chronic viral infection. The RFR examination can detect pathological frequencies.

Thyrotoxicosis occuring in case of Graves' disease may lead to the degeneration of the skeletal muscle fibers, to the enlargement of the heart, to fatty infiltrations or diffuse fibrosis of the liver and to the decalcification of the skeleton.

The ophthalmopathy of Graves' disease is characterized by inflammatory infiltrates (caused predominantly by lymphocytes, mast cells and plasma cells) of the orbital contents, exclusively of the globe. Dermopathy is characterized by the thickening of the dermis, which is infiltrated with lymphocytes and mucopolysaccharides.

Diagnosis: symptomatically, by measuring THS levels, by basic metabolism examinations, antibody examinations, etc.

Differential diagnosis: by distinguishing it from a toxic adenoma, a toxic multinodular goiter or tumors.

Treatment: By administering propylthiouracil, methimazole or carbimazole, by the surgical removal of the thyroid gland or by treating with radioactive iodine.

RFR method: detects and may eliminate the microorganisms.

RFR method should be started as soon as possible. If this method is ineffective for a month, it can be finished as proved ineffective.

The most frequent resonances found in case of Struma parenchymatosa (hyperplasia) are: 293-300, 408-420, 503, 540-549 kHz

The most frequent resonances found in case of Struma cystica are: 340-346, 380-390, 543-545 kHz
The most frequent resonances found in case of Struma nodosa are: 320-324, 332-335, 360-365, 408-416, 480, 520, 524-530 kHz
Its rare resonant frequencies are: 337-339, 305, 347-349, 352, 377, 422, 491, 516, 518, 543, 548, 560 kHz
According to my opinion this list is not complete yet. One must go on to measure and treat with a special local electrode.

24.4. Hyperparathyroidism

Primary hyperparathyroidism is a disorder of the parathyroid glands. Most people with this disorder have one or more enlarged, overactive parathyroid glands secreting too much parathyroid hormones.
In case of a secondary hyperparathyroidism, a problem, f.i. a kidney failure can result in the resistance of the body to the action of parathyroid hormones.
Parathyroid glands secrete parathyroid hormones (PTHs), which help to maintain the correct balance of calcium and phosphorus in the body. PTH regulates the release of the calcium from the bones, the absorption of calcium in the intestines, and the excretion of calcium into the urine.
When the amount of calcium in the blood falls too low, the parathyroid glands secrete just enough PTH to restore the balance with a feed back mechanism.
If the glands secrete too much hormone, (such as in case of hyperparathyroidism), the balance is disrupted, so that the calcium amount in the blood will be increased, i.e. hypercalcemia will be developed. 80 percent of people with this disorder has a benign tumor (adenoma) of the parathyroid glands, causing overactivity. Hyperparathyroidism can but rarely be caused by a cancer of the parathyroid gland. These adenoma and/or neoplasia are caused by *different special viruses*. *Mycoplasma* can play a role in the development of this disease. In case of hyperparathyroidism the excess amount of PTH triggers the release of too much calcium into the bloodstream. The bones lose calcium, more over, too much calcium can be absorbed from food. The amount of calcium will increase in the urine, causing kidney stones. PTH increases the excretion of phosphorus into the urine reducing thus the phosphorus level in the blood.
The vast majority of the cases affects patients with no family history of the disorder. Familial endocrine neoplasia type I is a rare, inherited syndrome that affects the parathyroids, the pancreas and the pituitary gland as well. Familial hypocalciuric hypercalcemia, which is sometimes confused with the typical hyperparathyroidism, is also a rare genetic disorder.
A person with hyperparathyroidism may have severe symptoms, subtle ones, or none at all.
Symptoms are often mild and nonspecific, f.i. weakness, fatigue, depression and pain. In more severe cases loss of appetite, nausea, vomiting, constipation, confusion or impaired memory, increased thirst and urination often come about. Patients may have thinning of the bones without any symptom, but with a risk of fracture. The increased calcium and phosphorus excretion in the urine may cause kidney stones. Patients with hyperparathyroidism develop more likely peptic ulcers, high blood pressure and pancreatitis.
Diagnosis: by calcium, PTH, phosphorus testing of the blood and urine.
Treatment: by surgery.
RFR method: detects and may eliminate the pathogen microorganisms!
As to the frequencies of neoplasia [redacted] **the most frequent resonances found are 402-410 kHz**
As to the resonant frequencies of Mycoplasma [redacted]

24.5. Addison's Disease

Addison's disease (AD) is an adrenocortical insufficiency. Addison's classic description of this disorder sounds as follows: „general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of the color of the skin." The disease, if unrecognized and untreated, carries an almost uniformly poor and frequently fatal prognosis [redacted] Chapter 23.17.).

The adrenal glands are sometimes the loci of chronic, granulomatous, infectious diseases predominately of *tuberculosis* but also of fungal infections, f.i. *histoplasmosis*, *coccidioidomycosis*, and *cryptococcosis* or/and of viral infections such as *HTLV*, and *Mycoplasma fermentans*. An idiopathic atrophy of the adrenal glands can be frequently observed, suggesting that an autoimmune mechanism may be responsible for its pathogenesis.

Addison's disease occurs when the adrenal glands do not produce enough hormone cortisol, nor hormone aldosterone in some cases. Its problem may be due to a disorder of the adrenal glands themselves (primary adrenal insufficiency) or to an inadequate secretion of ACTH by the pituitary gland (secondary adrenal insufficiency). Cortisol belonging to the glucocorticoids is normally produced by the adrenal glands and it affects almost every organ and tissue of the body. Its most important function is to help the body to respond adequately to stress. The most important effects of cortizol are as follows:

– Helping to maintain blood pressure and cardiovascular function;
– Helping to slow down the inflammatory response of the immune system;
– Balancing the effects of insulin in breaking down sugar for energy; and
– Helping to regulate the metabolism of proteins, carbohydrates and fats.

Cortisol is of vital importance to health, its amount produced by the adrenals is precisely balanced. Like many other hormones, cortisol is regulated by the hypothalamus and the pituitary gland. First, the hypothalamus sends corticotropin-releasing hormones (CRHs) to the pituitary gland, which responds by secreting hormones that regulate growth, thyroid and adrenal function, and sex hormones such as estrogen and testosterone. One of the pituitary's main functions is to secrete ACTH (adrenocorticotropin), that stimulates the adrenal glands. When the adrenals receive the pituitary's signal in form of ACTH, they respond by producing cortisol. Completing the feed back cycle, cortisol then signals the pituitary to lower the amount of the secretion of ACTH.

Aldosterone belongs to a class of hormones called mineralocorticoids, produced likewise by the adrenal glands. It helps to maintain the blood pressure, water and salt balance in the body by helping the kidney to retain sodium and to excrete potassium. If the aldosterone production gets to be too low, the kidneys will be unable to regulate the salt and water balance, so that the blood volume and pressure will decrease.

Almost every case of Addison's disease is caused by the gradual destruction of the adrenal cortex by the immune system of the body. About 70 percent of the reported cases of Addison's disease **are caused by autoimmune disorders**, in which the immune system produces antibodies that attack the body's own tissues or organs and slowly destroys them. Adrenal insufficiency occurs if at least 90 percent of the adrenal cortex is destroyed, as a result of which, glucocorticoid and mineralocorticoid hormones are often both lacking. Sometimes only the adrenal gland is affected, fi. in idiopathic adrenal insufficiency; though sometimes also other glands are affected, as f.i. in case of polyendocrine deficiency syndrome.

Polyendocrine deficiency syndrome Type I occurs among children, the adrenal insufficiency may be accompanied by the hypofunction of the parathyroid glands, slow sexual development, pernicious anemia, chronic candida infections and chronic active hepatitis.

Polyendocrine deficiency syndrome Type II, (Schmidt syndrome), afflicts usually young adults. It is associated with the hypofunction of the thyroid gland, a slow sexual development, diabetes mellitus and sometimes vitiligo. Scientists are of the opinion that polyendocrine deficiency syndrome is inheritable, as often more than one member of the family suffer from one or more endocrine deficiencies.

Tuberculosis, *chlamydial* and *mycoplasmal* (M. fermentans) infections can cause **primary adrenal insufficiency**. Less common causes of the primary adrenal insufficiency are chronic infections, mainly fungal infections such as *candida,* etc., cancer cells spreading from other parts of the body to the adrenal glands; amyloidosis and the surgical removal of the adrenal glands.

A temporary form of secondary adrenal insufficiency may occur if a person who have been receiving glucocorticoid hormones for a long time abruptly stops or interrupts this medication. Glucocorticoid hormones, which are often used to treat inflammatory illnesses (like rheumatoid arthritis, asthma bronchiale and ulcerative colitis), block the release of the corticotropin-releasing hormone and ACTH. Normally, CRH instructs the pituitary gland to release ACTH. If the CRH level drops, the pituitary gland does not release ACTH, the adrenals then fail to secrete sufficient levels of cortisol.

An other cause of secondary adrenal insufficiency is the surgical removal of noncancerous, ACTH-producing tumors of the pituitary gland (causing thus Cushing's syndrome). In this case, the source of ACTH will suddenly be removed, a replacement hormone therapy must be applied until a normal ACTH and cortisol production will come to pass. In case of the less commonly occuring adrenal insufficiency the pituitary gland will either decreases or stops to production of ACTH. This can.results from tumors or infections of the pituitary gland, from the loss of blood flow to the pituitary gland, from the radiation treatment of pituitary tumors, or from the surgical removal of parts of the hypothalamus or the pituitary gland.

The **Symptoms** of adrenal insufficiency are characterized by loss of weight, muscle weakness, fatigue, low blood pressure, and sometimes by hyperpigmentation, a striking sign of the disease, which commonly appears as a diffuse brown, tan, or bronze darkening of the skin at the elbows or creases of the hand. There appear bluish black patches on the mucous membranes of some patients.

Addison's disease can cause irritability and depression. Craving for salty food is common, hypoglycemia is more significant in case of children. Menstrual periods may become irregular or stop.

As the symptoms progress slowly, they are usually ignored until a stressful event like an illness or an accident causes their worsening This attack is termed addisonian crisis, or acute adrenal insufficiency.

Symptoms of an addisonian crisis include sudden penetrating pain in the lower back, abdomen and legs; severe vomiting and diarrhea, followed by dehydration; low blood pressure and loss of consciousness. If left untreated, an addisonian crisis can be fatal.

Diagnosis: symptomatically, based on the patient's medical history, by laboratory tests, x-ray, etc.

Treatment: by substitution of the failing hormones. During an addisonian crisis, low blood pressure, low blood sugar, and high levels of potassium can become life threatening. Its standard therapy involves intravenous injections of hydrocortisone, saline and dextrose.

RFR method: detects and may eliminate the pathogen microorganisms!

The most frequent resonances found in case of Addison's disease are: 312, 329, 339, 344-347, 350-353, 360-362, 384-405, 408-410, 424, 442-445, 449-450, 494-505, 520, 534-546, 569 kHz

Medical treament is most necessary while using RFR method.

24.6. Adrenal Hemorrhage (Waterhouse-Friderichsen Syndrome)

Adrenal hemorrhage is a rare, but potentially catastrophic event observed in patients of all ages, occurring usually in form of a complication of physiologic stress, trauma, or coagulopathy.

Infectious adrenal hemorrhage is a relatively uncommon condition causing an acute adrenal crisis, shock, and can, unless recognized promptly and treated appropriately, lead to death. Bilateral adrenal hemorrhages are associated with acute stressful illnesses, acute and chronic infections, congestive heart failure, myocardial infarction, complications of pregnancy or event surgery on invasive procedures. Other frequent associations are hemorrhagic diatheses, thromboembolic diseases including the antiphospholipid antibody syndrome, blunt trauma, and septic states caused f.i. by pseudomembranous colitis, wound infections and certain other severe infectious diseases. The acute adrenal insufficiency is due to massive haemorrhages in the adrenal glands most often a bilateral damage.

The **Waterhouse-Friderichsen syndrome (purpura fulminans)** is characterized by hemorrhagic necrosis of several organs, including an adrenal hemorrhage, caused by an overwhelming sepsis. Patients frequently suffer from a distinctly hemorrhagic skin rash. This acute syndrome affects mostly infants and children and does only occasionally develop in case of adults. Although the syndrome was originally described in association with *meningococcal* diseases, the syndrome can also be caused by other bacterial pathogens, including *Streptococcus pneumoniae*, antibiotics resistant *Streptococcus pyogenes* species, *beta-hemolytic streptococci*, Neisseria gonorrhoeae, Escherichia coli, Klebsiella pneumoniae, Haemophilus influenzae *(group B), Pseudomonas,* Salmonella choleraesuis, Pasteurella multocida, Acinetobacter calcoaceticus, Plesiomonas shigelloides, *rare tropic acute viral infectious diseases, f.i.* ebola, etc.

Antiphospholipid antibody syndrome, a primary or secondary Systemic Lupus Erythematosus can also be complicated with bilateral adrenal hemorrhages.

Mycoplasmal and *HTLV infections* are often present in infectious adrenal hemorrhagic syndromes.

Cytotoxic hypoxia causes tissue damages of the endothelial cells (due to an intensive release of endothelin 1-3), leading thus to their hemorrhage. Due to the complexity of its vasculature the adrenal tissue is disproportionately susceptible to massive intraglandular hemorrhages. Many patients with adrenal hemorrhage suffer from a coexisting renal vein thrombosis as well.

Symptoms: The onset is often explosive, shivering with cold, violent headache, vertigo, vomiting and prostration. The patient's skin gets cyanotic, there develop petechial or purpuric rashes and sometimes disseminated purpuric cutaneous haemorrhages, followed by sloughing and necrosis. In the preshock phase, patients are alert though pale, their extremities become cold and cyanotic due to a generalized vasoconstriction. The inicially moderate fever gets then high. An acute adrenal haemorrhage can cause circulatory collapse, characterized by clammy skin, high fever, rapid thready pulse, labored respiration, an alarming drop of blood pressure and coma.

Complications of adrenal hemorrhages include volume loss and shock in case of infants, as well as adrenal pseudocysts and adrenal calcifications. Death due to adrenal hemorrhages can frequently be caused by massive blood losses of neonates and by adrenal insufficiency of adults. Confusion, disorientation and collapse of circulation rapidly ensues and death may occur within 5 to 48 hours.

Diagnosis: symptomatically, by ultrasound, CT scanning, MRI (all of them demonstrating a nonspecific enlargement and hemorrhage into one or both adrenal glands). By biopsy.

Differential diagnosis: by distinguishing it from an adrenal neuroblastoma occuring in the neonatal period, and from xanthomatosis (Wolman disease).)

Treatment: by shock therapy and by controlling infections by effective intravenous antibiotics. Steroid treatment is indicated in case of all patients with septicemia associated with shock.
RFR method: could preliminary detect the infective microorganisms and eliminate them.
The most frequent resonances are: 308-313, 317-319, 321-323, 330-346, 359-360, 364-370, 372, 376-381, 452, 490-494, 564 kHz
Rarely *HTLV* or *Mycoplasma fermentans* can be present in case of this syndrome; as to their frequencies, see their special Chapters.

24.7. Cushing's Syndrome

Cushing's syndrome is a hormonal disorder caused by a prolonged exposure of the body's tissues to high levels of cortisol (named thus „hypercortisolism"). Symptoms vary, but most people have obesity of the upper parts of their body, rounded face, increased fat around the neck, but the thinning of arms and legs. Children tend to be fat with a slowed growth rate. Their skin becomes fragile and thin, it easily get bruised and heals but slowly. Purplish pink stretch marks can appear on the abdomen, thighs, buttocks, arms and breasts. The bones get weakened, routine activities such as bending, lifting or rising from a chair may lead to backache, rib and spinal column fractures.
Most people feel severe fatigue, have weak muscles, high blood pressure and high blood sugar. Irritability, anxiety and depression are common.
Women usually have excess hair growth on their faces, necks, chest, abdomen and thighs. Their menstrual periods may become irregular or stop. Men have decreased fertility with diminished or absent desire for sex.
Cushing's syndrome occurs if the body's tissues are exposed to excessive levels of cortisol for a long period of time. Many people suffer from the symptoms of Cushing's syndrome because they take glucocorticoid hormones such as prednisone for asthma, rheumatoid arthritis, SLE or other chronic inflammatory diseases.
Others develop Cushing's syndrome because of the overproduction of cortisol. Normally, the production of cortisol follows a precise chain of events. It is at first the hypothalamus that sends corticotropin releasing hormones (CRH) to the pituitary gland. CRH stimulates the secretion of ACTH (adrenocorticotropin) in the pituitary gland whereafter the adrenal glands receiving ACTH will release cortisol into the bloodstream.
Cortisol performs vital tasks in the body. (See Chapter 24.5)
Pituitary adenoma may cause Cushing's syndrome. These benign, non-cancerous tumors of the pituitary gland, secrete an increased amount of ACTH. Most patients have but one single adenoma. This form of the syndrome is the socalled „Cushing's disease", affecting women five times more often than men. Pituitary adenoma is caused by *Human Papilloma Viruses*.
Ectopic ACTH Syndrome: certain benign or malignant (cancerous) tumors arising outside of the pituitary gland can produce ACTH. The most common forms of ACTH-producing tumors are the oat cell or small cell lung cancers, accounting for about 25 percent of all lung cancer cases, and the carcinoid tumors. Other less common types of tumors that can produce ACTH are the thymomas, the pancreatic islet cell tumors, and the medullary carcinomas of the thyroid.
The abnormalities of the adrenal glands, i.e. mostly adrenal tumors, can cause Cushing's syndrome. People get ill mostly at the age of 40. Most of these cases involve non-cancerous tumors of the adrenal tissue, named **adrenal adenomas**, releasing excess amounts of cortisol into the blood.
Adrenocortical carcinomas, (i.e. adrenal cancers) are but seldom the cause of Cushing's syndrome. Cancer cells secrete excess levels of several adrenal cortical hormones, including cortisol and adrenal androgens. Adrenocortical carcinomas cause usually very high hormone levels and the rapid development of the symptoms.

Though Cushing's syndrome can but seldom be inherited, due to inherited factors tumors of one or more endocrine glands will develop in certain rare cases.
In case of the **Primary Pigmented Micronodular Adrenal Disease**, children or young adults develop small cortisol-producing tumors of the adrenal glands. In case of the **Multiple Endocrine Neoplasia Type I (MEN I)** there can develop hormone secreting tumors of the parathyroid glands, pancreas and pituitary gland. Cushing's syndrome of MEN Type I may be caused by pituitary, ectopic or adrenal tumors. Patients suffering from Cushing's syndrome frequently have *Chlamydial* or/and *Mycoplasmal* infections.
Diagnosis: symptomatically, based on the patient's medical history, by physical examinations and laboratory tests. By x-ray examinations.
Treatment: depends on the causes of the illness and may include surgery (transsphenoidal adenomectomy), radiation, chemotherapy or the use of cortisol-inhibiting drugs. The choice of cancer treatment are surgery, radiotherapy, chemotherapy, immunotherapy, or a combination of these treatments,
RFR method: can succesfully treat the original disease, i.e. the tumor. Detects the tumorous frequencies (see their special Chapters) and eliminates the pathogen microorganisms!
The most frequent resonances found in case of adenoma are: 337-340, 345, 369-373, 393, 397, 402-410, 447-451, 555 kHz
The most frequent resonances found in case of carcinoma are: 426-438 kHz
The resonances of Mycoplasma are: 307-308, 321-324, 337-350, 442-451, 493-495 kHz

24.8. Prolactinoma

Prolactinoma is a benign tumor of the pituitary gland producing prolactin hormone. It is the most common type of the pituitary tumors. Symptoms of prolactinoma are caused by hyperprolactinemia or by the pressure of the tumor on the surrounding tissues.
The pituitary gland (named sometimes master gland) plays an important role in regulating growth and development, metabolism and reproduction. It produces besides prolactin a variety of other key hormones, including the growth hormone, which regulates growth; ACTH (corticotropin), which stimulates the adrenal glands to produce cortisol; thyrotropin, which signals the thyroid gland to produce thyroid hormones; and luteinizing and follicle-stimulating hormones, which regulate ovulation and the estrogen and progesterone production in women and the sperm formation and testosterone production in men.
Affecting women, prolactinemia often causes infertility and changes in their menstruation, f.i. their menstruation periods may become irregular, their menstrual flow may change and, in some cases, their menstruation periods may cease altogether. Breast milk can be produced even by non-pregnant and non-nursing women. Some women experience the loss of libido, their sexual intercourse can become painful due to vaginal dryness.
The most common symptom of prolactinoma of men is impotence. As men have no reliable indicator such as menstruation to signal the problem, men often delay going to the doctor until they suffer from headache or eye problems caused by the enlarged pituitary pressing the nearby eye nerves.
Prolactin secretion in the pituitary gland is normally suppressed by dopamine, the brain neurotransmitter. Drugs that block the effects of dopamine at the pituitary gland or deplete dopamine stores in the brain may cause the hypersecretion of prolactin. Such drugs are f.i. the major tranquilizers (trifluoperazine, haloperidol, metoclopramide, etc., used to treat gastroesophageal reflux and nausea caused by certain cancer drugs); as well as alpha methyldopa and reserpine, used to control hypertension.
Diagnosis: symptomatically. By examinations of prolactin, CT and MRI.
Treatment: by administering dopamine agonists, surgery and x-ray.
RFR method: detects and may eliminate the pathogen of this benign tumor!

The most frequent resonances found in case of prolactinoma are: 337-339, 348, 370-373, 397, 402-410, 442-450, 510-516, 540-548 kHz

24.9. Endometriosis

Endometriosis is a disease in which patches of endometrial tissues, normally found only in the uterine endometrium, grow outside of the uterus. This tissue, possessing the same steroid receptors as a normal endometrium, is capable of responding to the normal cyclic hormonal milieu. *ECHO, HPV, HTLV and other combined viral infections* can cause the metaplastic conversion of the coelomic epithelium (peritoneal anlage) and the hematogenous or lymphatic dispersion of the endometrial cells and lead to their microscopic internal bleeding, subsequent inflammatory responses, neovascularization and fibrosis. All these alterations can be manifested as severe dysmenorrhea, chronic pelvic pain and infertility. Ectopic endometrial tissues are present mostly in the dependent parts of the female pelvis, f.i. on the posterior and anterior cul-de-sac, the uterosacral ligaments, tubes and ovaries, though any organ is at risk. These ectopic foci respond to cyclic hormonal fluctuations in the same way as the intrauterine endometrium does, with proliferation, secretory activity and cyclic sloughing of menstrual material. Endometriosis is confined to women of reproductive age with an active hypothalamic-pituitary-ovarian axis.

The **Symptoms** of endometriosis reflect the locus of the involvement. Patients usually suffer from a progressively increasing pelvic pain and/or from a secondary dysmenorrhea, rarely diarrhea, or even cyclic hematochezia if the endometriosis involves the rectosigmoid colon. Dysuria, flank pain and hematuria may likewise be present if the bladder or ureters are involved. More uncommon cyclic symptoms include hemophtysis (pulmonary involvement), catamenial seizures (endometriotic lesions in the brain), and umbilical bleeding (implants in the umbilicus). A familial/genetic predisposition may play a role.

Diagnosis: symptomatically, by ultrasound scans, x-ray, CT and MRI.

Cancer antigen 125 (CA-125) levels may be elevated in certain advanced cases but are rarely elevated in case of a mild-to-moderate disease. By laparotomy and histologic demonstrations.

Treatment: by administering gonadotropin-releasing hormone agonists, progestins, oral contraceptive pills and androgens in order to decrease the symptoms. By surgery.

RFR method: detects the combined viral infections, the chlamydial infections, and eliminates these microorganisms.

The most frequently found resonances are: 307-321, 330, 354-359, 369-376, 391-392, 396-397, 402-410, 427-431, 470-472, 496, 500, 526-530 kHz

24.10. Polycystic Ovarian Syndrome (Stein-Leventhal Syndrome)

Polycystic ovarian syndrome (PCOS), also known as Stein-Leventhal syndrome, is an endocrine disorder affecting approximately 10% of all women and is characterized by enlarged ovaries containing many fluid-filled cysts. It can affect all races and nationalities, is the most common hormonal disorder of women of reproductive age and a leading cause of infertility. Its most often signs are weight problems, lack of a regular ovulation and/or menstruation, and excessive amounts and effects of androgenic (masculinizing) hormones. The symptoms and severity of this syndrome can greatly vary. Insulin resistance, diabetes and obesity usually correlate with the PCOS.

The pathophysiology of this PCOS is very complex. In normal cases, the hypothalamus secretes gonadotropin-releasing hormones (GnRH) in a pulsatile manner. The pituitary gland responds to this by releasing luteinizing hormones (LH) and follicle-stimulating

hormones (FSH) in a similar cyclic manner. In case of a complex interaction, the LHs surge, the elevated levels of estradiol and an increase in the circulating progesterone level will trigger the midcycle surge of FSH. The cycle described above is disturbed in case of a polycystic ovarian syndrome, so that elevated LH levels, normal or low FSH levels, increased levels of free estrogen, and increased androgen precursors in the serum are characteristic. The same androgens inhibit the production of sex hormone-binding globulins in the liver, indirectly increasing the levels of free estrogen in the bloodstream. The elevated local androgen levels in the ovary exert an inhibitory effect on the follicular maturation. Owing to the permanent presence of FSH, these follicles continue to develop without maturing. Numerous follicles are thus present in the polycystic ovary and show various phases of development and atresia. Several studies have revealed an inherited form of the disease that appears to exhibit autosomal dominant transmission with incomplete penetrance. The pathophysiology of this disease is very complex, may include E vitamine deficiencies, *Coxsackie and other* viral infections, f.i. *HPV,* and inherited predisposition. Endometrial adenocarcinoma can also be associated with polycystic ovarian syndrome. A *Coxsackie viral* infection can cause the estrogenic stimulation of the endometrium and an other viral i.e. *HPV* infection increases the risk of endometrial hyperplasia and its subsequent transformation into an endometrial carcinoma. The present hormone balance may increase also the risk of breast cancer.

There are multiple possible causes of infertility experienced in case of PCOS. The main causes are thought to be:

Genetic: About the half of all early miscarriages occurs owing to chromosomal abnormalities. The most chromosomal abnormalities result from a defective egg or sperm, which result in wrong number of chromosomes or a chromosomal defect of the embryo. These embryos usually fail to thrive and the pregnancy will get miscarried.

Immunological: *German measles* or some other infections, caused f.i. by *HTLV, Mycoplasma* are accompanied by fever and may lead to miscarriage. Women with a *bacterial vaginal* infection have a significantly greater risk of miscarriage in their second trimester of pregnancy. *Mycoplasma* and *HTLV* are pathogens, which may iniciate autoimmune processes causing endocrine disorders.

Women with inherited tendency to produce antibodies causing an excessive tendency to form blood clots, blocking thus the circulation to their developing fetus have more miscarriages and problems in their pregnancy.

Anatomical: A weak or open cervix, an irregularly-shaped uterus as well as large fibroids or scars of the uterus can lead to a later miscarriage.

Lifestyle: alcohol abuse and smoking are risks for miscarriage, premature birth, and low birth weight.

Diet: Extremely low serum levels of zinc, folic acid and vitamin E can be associated with miscarriages.

Symptoms: Untreated patients with polycystic ovarian syndrome suffer from irregular menses or amenorrhea and infertility from their menarche to menopause. Primary amenorrhea is an uncommon symptom. Obesity and insulin resistance are also related abnormalities. Half of all patients have systemic signs of androgen excess, i.e. hirsutism, acne, or male-pattern baldness may also be signs of this disorder. In case of approximately one half of all patients, sonograms show polycystic ovaries. One quarter of the patients may not have any clinically evident abnormality.

Diagnosis: symptomatically. By pelvic ultrasound (Although not all women with PCOS have polycystic ovaries nor do all women with ovarian cysts have PCOS; so that its diagnosing might prove to be difficult.)

Differential diagnosis: by distinguishing it from other causes of irregular or absent menstruation and hirsutism, f.i. from connatal adrenal hyperplasia, tumor, Cushing's

syndrome, hyperprolactinemia, androgen secreting neoplasms and other pituitary or adrenal disorders.
Treatment: symptomatically, by the restoration of fertility, by treating hirsutism and acne, by the restoration of regular menstruation, by preventing endometrial hyperplasia and cancer. By administering insulin-lowering medications and extra hight dose vitamin E. in order to restorate fertility, by administering contraceptive pills containing cyproterone acetate and spironolactone treating acne and hirsutism respectively.
RFR method: detects and may eliminate the combined viral and other pathogens!
The most frequent resonances of PCOS are: 303, 361-366, 370-374, 402-410, 442-452, 500-506, 576 kHz
As to the frequencies of *Mycoplasma* and *HTLV*, see their special Chapters.
RFR method should be applied for a long time!
HPV, ECHO virus, Ureaplasma and other infections can also be causal coinfections in case of PCOS.

24.11. Pineal Calcification

Pineal calcification is a lesion, becoming first to be visible often by an x-ray examination of the skull at the time of puberty, and it is suggested that the gland degenerates at this time of life and then becomes calcified.
This pineal calcification is caused by *nanobacterium sanguineum*. If pineals are examined by appropriate microscopic techniques, evidence of calcification often can be observed in patients who died long before their puberty. A ground substance believed to serve as the matrix for calcification was seen in 8 of 28 pineal glands taken from children under one year of age. Pineal function (this brain center that controls our sleep-wake cycle) have failed to show any differences between heavily calcified glands taken from aged subjects and pineals from young subjects with no gross evidence of calcification. The functional significance of pineal calcification remains entirely unexplained. The chemical identified in the calcified material appears to be hydroxyapatite; the crystals of this compound taken from human pineals are similar to those prepared from the bones or the teeth. *Nanobacterium* is *sanguineum* initiates this calcification. Nanobacteria build shelters and precipitated minerals. With aging there is usually no calcium deposition within the pineal gland, but if there is, it is not normal. The Ultrafast CT Scanner can localize and quantitatively measure the calcium within the pineal gland. Calcification is a destructive process occuring in the pineal glands.
Its most frequent **symptom** includes disturbed sleep, somnipathy, dysssomnia. Senile insomnia can be caused by nanobacteria and by the calcification of the pineal glands.
Diagnosis: by CT and by measuring the calcium level in the pineal gland, by the identification of nanobacteria.
Differential diagnosis: by distinguishing it from pineal tumors.
Treatment: the administration of Doxycyclin is not effective enough.
RFR method: detects and may eliminate the nanobacterium and the associated other pathogen microorganisms!
The most frequently found resonances are: 294-298, 305-310, 317-318, 324-325, 332-336, 345, 372-387, 395-398, 430-435, 440-442, 466-476, 486, 528, 556 kHz

24.12. Diabetes Insipidus

Diabetes insipidus (DI) is a disease in which insufficient levels of antidiuretic hormones cause excessive thirst, polydipsia and excessive production of very dilute urine (i.e. polyuria).

DI is caused by the decreased production of vasopressin, which hormone normally restrains the body from the production of too much urine.
The renal collecting ducts are unable to concentrate urine in case of vasopressin deficiency or resistance. The collecting ducts concentrate urine by reabsorbing water, which function is controlled by the posterior pituitary gland via secretion of vasopressin (named also antidiuretic hormone, ADH). The reabsorption of sugars, amino acids and virtually all electrolytes is completed by the time the urine has reached this segment of the nephron. The inability to conserve water by reabsorption in the collecting duct leaves thus sodium unaffected. DI causes an extremely diluted, increased urine and hypernatremia, followed by polydipsia, as the thirst mechanism urges the replenishment of the body water.
The secretion of vasopressin occurs in the posterior pituitary gland regulated by the paraventricular and supraoptic nuclei, which are sensing the changes in osmolality. The destruction of the paraventricular and supraoptic nuclei and the posterior pituitary gland caused by tumor, pressure, surgical ablation, genetic predisposition, or by a viral infection can cause a decreased vasopressin secretion and a central diabetes insipidus (CDI). DI may be an inherited autosomal dominant disease or an autosomal recessive disorder.
Nephrogenic Diabetes Insipidus (NDI) arises from defective or absent receptor sites at the cortical collecting duct segment of the nephron (caused by an X-linked, vasopressin V2 receptor deficiency, its locus being in Xq28) or defective or absent aquaporin, the protein transporting water through the collecting duct. In case of these defects, the ducts do not respond appropriately to the normal amounts of vasopressin.
In both forms of DI dehydration results from the inability to reabsorb water at a site distal to electrolyte reabsorption, leading to hypernatremia, hyperchloremia and prerenal azotemia. The diminished blood volume associated with increased blood viscosity increases the risk of slugging and thrombosis. Dehydration is threatening the patients especially in warm weather. Their symptoms are excessive thirst and urine production, urinating large quantities frequently even during the night.
Postencephalitis DI is usually caused by combined viral infections such as by *Coxsackie virus, ECHO virus, CMV, EBV or/and HTLV and HPV, etc.*
Diagnosis: symptomatically, by monitoring urine, by cranial MRI, by antidiuretic hormone examinations.
Treatment: by administering vasopressin analogs and their derivates, diuretics, NSAIDs and sulfonylurea compounds.
RFR method: in case of infectious DI it detects and eliminates the combination of the pathogen viruses.
The most frequent resonances of Coxsackie viruses are: 287-290, 294-303, 307-309, 315-327, 361-365 kHz
The most frequent resonances of CMV are: 408-410, 530-536 kHz
The most frequent resonances of EBV are: 372-383 kHz
The most frequent resonances of HTLV see its special Chapter.
The most frequent resonances of HPV are: 346, 429, 442 kHz, as to its other frequencies see its special Chapter.
The resonant frequencies of Mycoplasma fermentans are: 442-451, 493-495 kHz
The most frequent resonances of ECHO virus are: 317-319, 369, 397-405, 471-473, 526 kHz
The most frequent resonances of Adenovirus are: 371-383, 393 kHz

24.13. Polyglandular Deficiency Syndromes

In case of polyglandular deficiency syndromes several endocrine glands produce less than normal amounts of hormones. Viral infections caused f.i. by *CMV* or *other viruses* and *mycoplasmal* or certain other infections can also suppress the activity of endocrine glands. It frequently does occur that after one gland gets damaged, the functioning of the others

will slow down or stop causing multiple endocrin hormone failures. The symptoms of polyglandular deficiency depend on the fact, which of the endocrine glands are malfunctioning. Polyglandular deficiency syndromes are categorized into four types, i.e. according to whether symptoms develop in childhood, or adulthood, which of the endocrin glands are involved and which microorganisms play a role in ther development of the polyglandular syndromes. People developing polyglandular deficiency syndromes probably have a genetic predisposition or a familiarly inherited pathogen agent. Autoimmune and degenerative polyglandular deficiency syndromes are complex multiorgan diseases. These chronic syndromes might be caused by *mycoplasmal or/and viral* infections. The symptoms of these chronic syndromes are characterized by disabling fatigue, intermittent fever, swollen lymph nodes, arthralgia, myalgia, headache, skin rashes, intermittent diarrhea, loss of weight, anemia, immunodepression or autoimmune processes, hypothyroidism, hypoparathyroidism, Addison's disease, chronic yeast infections, or chronic mucocutaneous candidiasis, etc. People suffering these syndromes have an inadequate immune response to common yeasts and if infected with yeasts, are unable to overcome them. The pankreas of patients with polyglandular deficiency syndromes can produce decreased amounts of insulin causing diabetes. Patients often suffer from hepatitis, gallstones, difficulty in absorbing food and get prematurely bald.
Diagnosis: by blood tests measuring the hormone production in the affected glands.
Treatment: by hormone replacement therapy.
RFR method: detects the viral or mycoplasmal pathogens and eliminates them! [redacted]

The most frequent resonances found in case of polyglandular deficiency syndromes are: 291-293, 312, 322-323, 329, 337-339, 342-349, 353, 361, 372-383, 397-404, 408-410, 424, 442-451, 485, 505, 543-546, 569 kHz
The treatment of these chronic syndromes may prove to be effective and the cure possible. The combination of various different pathogens difficult to treat results in diseases like these. Their mycoplasmal and viral combinated pathogenesis offers the possibility of a causal therapy.

24.14. Multiple Endocrine Neoplasia Syndromes

Multiple endocrine neoplasia syndromes are rare disorders, in case of which, several endocrine glands develop to be noncancerous (benign) or cancerous (malignant) tumors or grow excessively without forming any tumors.
These syndromes can affect infants or persons older than 80 years. Almost all of the multiple endocrine neoplasia syndromes are inherited.
Multiple endocrine neoplasia syndromes can be manifested in three patterns, called types 1, 2A, and 2B, though these types may occasionally overlap. The tumors and the abnormally larged glands often produce excess amounts of hormones. These abnormally enlarged glands or tumors may affect more glands at the same time, but can also develop gradually over time.
Multiple endocrine neoplasia syndrome Type-1 is characterized by multiple tumors or by enlarged glands with excessive growth and activity, affecting: the parathyroid glands, the pancreas, the pituitary gland and, less often, the thyroid gland and the adrenal glands.
Almost all patients with type 1 disease have noncancerous tumors of the parathyroid glands; producing too much parathyroid hormone (causing hyperparathyroidism). This excess amount of parathyroid hormone raises the level of calcium in the blood, leading sometimes to the development of kidney stones. Most people with type 1 disease develop also tumors of the hormone-producing cells of the pancreas. Some of these tumors produce high levels of insulin, causing hypoglycemia. More than half of these islet cell tumors produces excessive amount of gastrin stimulating the stomach to overproduce gastric acid. People with gastrin producing tumors generally develop bleeding peptic ulcers that often

perforate and leak stomach contents into the abdomen, or obstruct the stomach. High amounts of gastric acid can interfere with the activity of the enzymes of the pancreas, resulting in diarrhea and fatty, smelly stools (steatorrhea). The remaining islet cell tumors may produce other hormones, f.i. vasoactive intestinal polypeptides, causing severe diarrhea leading to dehydration.
Some of the islet cell tumors are cancerous and able to spread (metastasize) to other areas of the body. Cancerous islet cell tumors tend to grow more slowly than other types of pancreas cancers. People with type 1 disease can develop pituitary gland tumors as well. Some of these tumors produce hormone prolactin, leading to menstrual abnormalities and to erectile dysfunction (impotence). Some other tumors produce growth hormones, causing acromegaly. A small percentage of pituitary tumors produces corticotropin hormones, overstimulating the adrenal glands, causing high levels of corticosteroid hormones and Cushing's syndrome. Some pituitary tumors produce no hormones at all, others cause headache, impaired vision, and a decreased pituitary gland function by pressing against nearby parts of the brain.
People with type 1 disease may even develop different types of carcinoid tumors. Some people develop soft, noncancerous fatty growths just below the skin (lipomas).
Multiple endocrine neoplasia syndrome Type-2A can be characterized by developing tumors or the excessive growth and activity of two or three of the following glands: the thyroid gland, the adrenal glands and the parathyroid glands.
People with overactive parathyroid glands have increased levels of calcium in their blood, causing kidney stones. In other cases, the parathyroid glands can increase in size without producing large amounts of parathyroid hormone. This neoplasia type can consist of medullary thyroid cancer, pheochromocytoma, and neuromas (i.e. growths around the nerves). In case of type-2A the medullary thyroid cancer usually affects infants of 3 months.
Some people with type 2B disease have no family history of Multiple endocrine neoplasia syndrome. Medullary thyroid tumors of this type grow faster and spread more rapidly than those of type 2A. Most people with type 2B disease develop neuromas of their mucous membranes appearing as glistening bumps around the lips, tongue and the lining of the mouth. Neuromas may develop also on the eyelids and on the glistening surfaces of the eyes, including the conjunctiva and cornea. Digestive tract abnormalities of this illness cause constipation and diarrhea. The colon can get enlarged and dilated (megacolon). These abnormalities are probably caused by neuromas growing on the intestinal nerves. People with type 2B disease often develop spinal abnormalities, especially the curvature of the spine, as well as abnormalities of the bones of the feet and thighs. Many people suffering from this disease have long limbs and loose joints.
Diagnosis: tests for the identification of each type of these tumors are available. A test for abnormalities of the genes responsible for all the three types are available permiting thus an earlier and more effective diagnosis and treatment of persons having a family history of multiple endocrine neoplasia syndromes.
Treatment: Symptomatically, by surgery and by correcting the hormone imbalance with drugs.
RFR method: detects and may eliminate the associated pathogen microorganisms!
Surprisingly there are a lot of resonances to be found.
As to the frequencies of tumors see Chapter 26
As to the frequencies of Mycoplasmas and HTLVs see Chapter 23.4
The secondary frequencies of patients can differ.
In case of multiple endocrine neoplasia syndromes RFR method is the main therapy.

24.15. Autoimmune Polyendocrine Syndrome

Autoimmune polyendocrine syndromes are a heterogeneous group of rare diseases characterized by autoimmune activity against more than one or more endocrine organs, though non-endocrine organs can also be affected.
The presence of chronic inflammatory infiltrates mainly composed by lymphocytes in the affected organs and the presence of autoantibodies reacting with target tissue-specific antigens support the autoimmune etiology of these syndromes. The antibodies may be caused by the breakdown of the normal immunologic tolerancy or by immunization with an environmental agent having a similar antigenic molecular structure to a self-antigen.
Certain different genetic predispositions are described concerning these complex diseases.
The Autoimmune polyendocrine syndrome is associated with mucocutaneous *candidiasis, Mycoplasma fermentans, HTLV, CMV* and *adenoviral infections. Mycoplasmal* antigens adsorbed on the gland tissue, change some antigen structures of the tissue, *HTLV* infections can damage the functioning of the T cells. Combined *Candidal, Mycoplasmal and HTLV infections* cause damages of the T-cell functions, provoke a non adequate immune response, developing autoimmune polyendocrine syndromes and an increased susceptibility to opportunistic infections. This autoimmune process can only be triggered by these above mentioned combined infections if the support of an inherited chromosomal damage is also present.
Diagnosis: by endocrine autoantibody examinations of the serum, by function tests of the organs
Treatment: symptomatically. Mucocutaneous candidiasis can be treated with oral antifungal drugs.
RFR method: detects and may eliminate all pathogens
The most frequent resonances are: 288-293, 307, 338, 371-380, 382-390, 392-394, 370-376, 390, 406-410, 442-453, 475-476, 494-495, 572-580 kHz

24.16. Geriatric Syndromes

Geriatric syndromes are a group of diverse conditions, in which the functions of the affected organism are inadequate, f.i. the blood flow to the kidney, the liver and the brain, as well as the ability of the kidneys and the liver to clear toxins is decreased. Due to a decreased glucose tolerance diabetes type 2 can develop. The brain disorders lead to the progressive loss of memory and other intellectual functions, causing Alzheimer's disease, Parkinson's disease and other dementias of viral origin. In case of elderly persons, depression sometimes causes confusion, loss of memory and apathy, all these can be mistaken for dementia. Degeneration of the cartilages and bones (i.e. osteoarthritis or/and osteoporosis) may develop and cause pain. Benign prostate hyperplasia or a prostate cancer, urinary incontinence, an inability to control urination all can develop. Cataracts and glaucoma develop in case of elderly people. Rarely there develop monoclonal gammopathies, in case of which the abnormal proliferation of a single type of cells produces high levels of immunoglobulins. Patients suffer from various different chronic viral infections, caused f.i. by *herpes viruses, adenoviruses, Coxsackie viruses, etc.* exhausting thus the immune capacity of the organism. The immune response is decreased, infections will occur more often, recur more frequently, get unusually severe and last for a longer time than usually. Acute illnesses, such as heart attacks, hip fractures and pneumonia are more likely to cause the death of old people.
A young person has usually 1-2 pathogen resonance frequencies, while an elderly person has frequently 20-25. These microorganisms are pathogens of chronic viral, bacterial or fungal infections.
Genes determine how long the cells are alive. If the cells die, the organs begin to malfunction and eventually cannot maintain the biologic functions necessary to sustain life. With age, more and more damage is done until many cells cannot normally function or die.

Free radicals damage ultimately the DNA of cells causing a person's aging. The DNA repair system of a young person owns a high capacity, elderly people has a low one. The stimulation of the DNA repair system may stimulate the biological functions of the organism.

RFR method can eliminate the pathogen microorganisms, whereafter the immune response can get increased, but these frequencies of pathogens do not stimulate the DNA repair mechanism so that the genetic information of the cells remains defective.

The most frequent resonances are: 289-293, 297-308, 440-452, 534-544 kHz

25. PARANEOPLASTIC SYNDROMES IN GENERAL

Paraneoplastic syndromes (PS) are diseases or symptoms that are consequences of the presence of cancer in the body, though they are not direct signs of locally present cancer cells. These phenomena may be mediated by humoral factors, f.i. by hormones or by cytokines produced by the tumor cells or caused by immune responses against the tumor of patients suffering mostly from cancer of the lung, breast, ovaries or lymphomas. PSs are remote effects of a present cancer, affecting many different functions of the body, often those of the nervous system, too.

PSs can be triggered by an altered immune system responding to a neoplasm. They are defined as clinical syndromes involving nonmetastatic systemic effects that accompany a malignant disease. In a broad sense, these syndromes are collections of symptoms that result usually from substances produced by the tumor, and manifest themselves far from the tumor itself. The symptoms can be endocrine, neuromuscular, musculoskeletal, cardiovascular, cutaneous, hematologic, gastrointestinal, renal or miscellaneous-natured. Though fever is their most common manifestation, several clinical signs may also be observed, each simulating common benign conditions. These syndromes vary from those of dermatomyositis-polymyositis, Cushing's syndrome and malignant carcinoid. The mechanisms how cancers affect these distant loci are not precisely understood yet. When a tumor arises, the body produces antibodies to bind and destroy tumor cells. Unfortunately, in some cases, these antibodies cross-react with the normal tissues destroying them, and may thus stimulate the onset of paraneoplastic disorders, though not all paraneoplastic syndromes are associated with antibodies. Physicians dealing with cancer-associated syndromes try to differentiate the paraneoplastic syndromes and the benign disorders that but mimic paraneoplastic syndromes. The tumor-related antibodies determine an immune reaction or response in certain body parts, and lead to tissue damages and, hence, to a clinical manifestation. These cancer-fighting antibodies or T cells in the blood can attack the normal nervous cells by chance in the nervous system, thus causing there paraneoplastic symptoms. Certain cancer types produce fetal proteins physiologically expressed only in embryonic cells during the fetal life but not in normal adult cells. These substances may help laboratories to detect malignancies and are usually used as tumor markers (f.i. carcinoembryonic antigen (CEA), alpha-fetoprotein (AFP), cancer antigens (CA 19.9), etc). Paraneoplastic arthropathies arise as rheumatic polyarthritis or polymyalgia, particularly among patients with myeloma; lymphoma, acute malignant histiocytosis, leukemia and tumors of the pancreas, colon, prostate and the CNS. In some cases, the tumor can be preceded by scleroderma, with its peculiar clinical manifestations. Malignancy arising from epithelial cells of the thymus (i.e. thymoma), accounts for most cases of myasthenia gravis and, rarely, of hypogammaglobulinemia and pure red cell aplasia. Patients with lymphoma or cancer of the lungs, breast, or gonads may have SLE. Patients with myeloma, renal carcinoma and lymphoma may suffer also from secondary amyloidosis of the connective tissues.

Patients with tumors that secrete ACTH or ACTH-like substances may have nephropathy, characterized by urinary potassium leakage of more than 20 mEq per 24 hours, occuring f.i. in 50% of individuals with ACTH-secreting tumors of the small cell lung cancer.

Other types of tumors again may produce ACTH, antidiuretic hormones (ADH), gut hormones, or may cause hypokalemia, hyponatremia, or hypernatremia, hyperphosphoremia, alkalosis or acidosis. Nephrotic syndromes can sometimes be observed among patients with Hodgkin's lymphoma (HL), non-Hodgkin's lymphoma (NHL), leukemia, melanoma, or in case of malignancies of the lung, thyroid, colon, breast and ovary. Herpes Zoster, ichthyosis, flushes, alopecia, and hypertrichosis may also be

observed. Acanthosis nigricans and dermic melanosis are characterized by a blackish pigmentation of the skin usually affecting patients with metastatic melanoma or pancreatic tumors.

PSs can be sorted into 5 groups, such as

1. Mucocutaneous paraneoplastic syndromes

Dermatomyositis

Leser-Trélat sign, a sudden onset of multiple seborrheic keratoses

Acanthosis nigricans

Necrolytic migratory erythema

Sweet's syndrome

Pyoderma gangrenosum

2. Neurological paraneoplastic syndromes, such as

(Paraneoplastic) cerebellar degeneration, often associated with small cell lung cancer, ovarian, breast and other cancers and with Hodgkin's lymphoma. It can cause gait difficulties, dizziness, nausea and diplopia, followed by ataxia, dysarthria and dysphagia.

(Paraneoplastic) encephalomyelitis, characterized by symptoms caused by brainstem encephalitis, limbic encephalitis, cerebellar degeneration, myelitis and autonomic dysfunctions.

(Paraneoplastic) limbic encephalitis, characterized by depression, seizures, irritability and a rapidly developing short-term memory loss. This syndrome is mostly associated with small cell lung cancer.

(Paraneoplastic) sensory neuropathy, affecting the lower and upper extremities is characterized by progressive sensory loss, either symmetric or not.

Brainstem encephalitis

Opsoclonus-myoclonus syndrome usually affecting children younger than 4 years, associated with hypotonia, ataxia and irritability

Polymyositis

Lambert-Eaton myasthenic syndrome

3. Hematological paraneoplastic syndromes

Granulocytosis due to the production of G-CSF

4. Endocrine metabolic syndromes:

Hypercalcaemia due to the production of Parathyroid hormone-related protein, typically present in case of squamous cell cancers of the breast and lungs

The Syndrome of inappropriate antidiuretic hormone secretion (SIADH), typically associated with lung small cell and CNS malignancies.

Ectopic ACTH secretion, associated with small-cell lung cancer, carcinoid tumor, thymoma and other cancers.

5. Certain others, not fitting into any of the above categories, f.i.:

Membranous glomerulonephritis, etc.

The causes of PSs associated with underlying cancers are not well known. Only a few cases can clearly demonstrate their etiologic and pathogenetic factors.

Diagnosis: by laboratory studies of the blood, urine and cerebrospinal fluid.

Treatment: by therapies to eliminate the underlying cancer (such as chemotherapy, radiation, surgery, etc) and symptomatically (f.i. by therapies in order to reduce or slow down the neurological degeneration).

RFR method: detects and may eliminate all pathogen microorganisms.

The most frequent HPV resonances are: 314-319, 343-347, 401-410, 418-426, 427-438, 442-448, 452-453, 456-466, 467-479, 488-496, 501-507, 513-521, 525-527, 533-545, 556-564 kHz

The resonances of the most frequently found pathogens attacking the immune system in case of PS are: 297-299, 307-308, 311-315, 321-324, 337-350, 364-367, 370-383, 432-433, 440-452, 493-495, 518-519, 526-530, 569-571kHz

26. TUMORS

26.1. General Aspects of Tumors

26.1.1. Prevention of Tumorous Processes

Tumors consist of cells, which lost their normal control mechanisms and have thus a quick unregulated growth. Tumor can develop from any tissue within any organ. Tumors can be benign or malignant. Malignant tumor or cancer cells grow and multiply forming thus a mass of cancerous tissues that invade the adjacent tissues, can spread, metastasize and spread to all parts of the body.

Cancer cells develop from healthy normal cells within a complex process named precancerous and cancerous transformation. This cancerous process starts with an infection, in case of which, the genetic material of the affected cells or their functioning becomes damaged thus priming the cell to become cancerous. The change in the genetic material of the affected cell is caused by carcinogen *viruses*, which can activate chemical, free radical, or sunlight damages. The infection of a cell with a cancer virus is no cancer yet. Even one or more years after getting infected with a tumor virus there may no cancer process be developed. Several factors, often in combination of a susceptible cell and a direct or indirect carcinogen factor are needed for the development and growth of a tumor. Viruses infiltrate the DNA of the infected cell, but not every virus, only the *retrovirus* can be built into the host's DNA. Other cancer viruses only adhere to the surface of the chromosome, and damage its function. A series of chromosomal changes may by needed for the development of certain malignant tumors. Studies of familial polyposis of the colon suggest how this process may act in case of colon cancer. The normal lining of the colon begins to grow more actively, they hyperproliferate, as the colon cells have no suppressor gene anymore on their chromosome 5, normally controlling the growth of the lining. A slight change in the DNA of the colon cells then promotes changes leading to adenoma formations. Another gene (i.e. the RAS oncogene) can cause a more rapid activ growth of this adenoma. The subsequent loss of a suppressor gene on chromosome 18 stimulates further the growth of the adenoma and the loss of a gene on chromosome 17 finally converts the benign adenoma to cancer. Additional changes may lead to the metastasizing of this cancer. When a cell becomes cancerous, the immune system can often destroy these cells before they replicate to become established as a cancer. Familiar polyposis of the colon suggests that every member of the family is infected with a primitive *retrovirus*.

If the treatment with RFR method eliminates the carcinogen viruses in the affected cells, the cancerous process will turn back. The family history of cancer is an important prognostic factor. Some families, being virus-carriers have a significantly higher risk of developing certain cancers than other families. Exposure to certain common chemicals can greatly increase a person's chance of developing cancer, these substances may inhibit the person's immune system, or activate the cancerogen virus often years later after getting infected. RFR method is able to inhibit the cancerous process by eliminating the carcinogen viruses in the cells.

Cancer prevention means the detection of the cancinogen virus by RFR method, and their elimination by using this method.

26.1.2. The Causes of Tumors

Not all cells are equally susceptible to carcinogens and not all cells can get be virus infected. The genetic flaw of a cell makes it more susceptible to carcinogens. Even a chronic physical irritation can be cancerogenic if the cell is infected with a carcinogen virus. Purely carcinogen substances have no effect on by tumor virus not infected cells.

Several factors, often the combination of a susceptible cell and a carcinogen, are thus needed to cause cancer. In case of a cancerous process the DNA of a normal cell will undergo changes. The changes in the genetic material of a cell are often difficult to detect, but sometimes a change in size or shape of one specific chromosome indicates a certain type of cancer. The cell genome of the host will take the command of the virus to multiply.
The causative factors of a cancer might be the combination of a *virus* and *colonial factors produced by bacteria*. The viral component may be warts i.e. *Human Papilloma Viruses*, *Herpes viruses (i.e. HSV1, HSV2, HZV, CMV, EBV)* and several *Adenoviruses.*
The bacterial components are all colonial factors produced by bacteria.
Genetic changes were identified in brain tumors and in cancers of the colon, breast, lungs and bones.
Within a cancerous process there is a special cellular immunodeficiency present. Cancers are more likely to develop if the immune system does not function normally, f.i. in case of people with AIDS, or with autoimmune diseases. Cancerous cells can escape the immune system's protective surveillance even if it is normally functioning.
The most important cancerogen factor is the viral infection transmitted to each other in the family. Genetic and environmental factors of a host increase the risk of developing cancer, f.i. smoking, exposure to ultraviolet radiation of the sunlight, x-ray, radioactive rays, many chemicals such as asbestos, benzpyrene, etc.
The geographic variations of the risk of cancer are caused probably by a multifactorial combination of genetics, diet and environment.
The important role of the immune system in controlling the development of malformations is exemplified by the astounding statistics stating that cancer is 100 times more likely to occur among people who take immunosuppressive drugs than among those with normal immune system. Cancer starts growing and spreading rapidly in the recipient, whose immune system is suppressed by immunosuppressive drugs.
The cell antigens are to be found on the surface of the cells and a person's immune system does normally not react on his own cells. When a cell becomes cancerous its surface get changed. The immune system may regard these new antigens (named tumor antigens) as foreign, and may leave them untouched or destroy these cancer cells. However, a fully functioning immune system can always destroy all cancer cell. Tumor antigens are identified in several cancers, including malignant melanoma, osteosarcoma and some gastrointestinal cancers. People with these cancers may have antibodies against these tumor antigens. These antibodies are generally characteristic of a type of the immune response. The antibodies seem to be unable to destroy the cancer and sometimes seem even to stimulate its growth.
Tumor cells are able to absorb several bacterial growth factors. The most common tumor markers are, as follows:
Carcinoembryonic antigen (CEA) is a tumor antigen found in the blood of people with cancer of the colon, breast, pancreas, bladder, ovary, or cervix. High levels of this antigen may also be found in heavy smokers and in those having liver cirrhosis or ulcerative colitis. So that, heaving a high CEA level does not always mean that a person has cancer.
Alpha-fetoprotein (AFP), normally produced by fetal liver cells, can be found in the blood of people with liver cancer. More over AFP can be found in people with certain cancers of the ovary or testis and among children and young adults with pineal gland tumors. This factor is perhaps originating from *Shigella bacteria.*
Beta-human Chorionic Gonadotropin (beta-HCG), a hormone produced during pregnancy serving the basis for pregnancy tests, also occurs in women who have a cancer originally in the placenta and in men with various types of testicular cancer. Beta human chorionic gonadotropin is a very sensitive tumor marker.
Prostate specific antigen (PSA) levels can be high in men with non cancerous enlargements of the prostates but considerably higher in men with prostate cancer.

Cancer antigen CA-125, is measurably increased in women with a variety of ovarian diseases, including cancer. Since the ovarian cancer is often difficult to diagnose, this CA-125 tumor marker can be of help.
CA-15-3, a cancer antigen present in breast cancer may be originating from *Salmonella*, **CA-19-5** can be measured in case of pancreatic cancer, **beta-micro globulin** in case of multiple myeloma, **lactat dehidrogenase** in case of testicular cancer, but none of them can be recommended for cancer screening. These, according to Hulda Clark, are originating also from *Salmonella*.
CA-72-4 tumor factor is, according to Hulda Clark, originating from *Shigella*,
Epidermal growth factor is also a non-specific tumor factor. This might be a product of *staphylococcus*.
Cancer Associated Antigen (G1) can be observed in several tumors, and might be a product of *Esherichia coli*.
Platelet Derived Growth Factor (PDGF) is originating from *Salmonella* (Hulda Clark).
Cancer cells are able to adsorb the bacterial colony factors, and are able to grow faster aided by these factors.
In the clinical lab these tumor factors are useful for monitoring the response to treatment of a person already diagnosed with cancer.
Interleukins increase the direct tumor-killing mechanisms and the specific and aspecific immune responses.
Interferon is the best-known and most widely used biological response modifier. Immunotherapy used with it can slow down the cancerous process.
Killer cell therapy: the lymphocytes are exposed to a substance called interleukin-2, activating the Natural Killer cells, which can be reinjected into the person with malignant melanoma.

26.1.3. Comparative Table of Tumor Causing Factors

Tumor Causing Direct Factors
Direct factors have a direct effect on the tumor, and if this factor is eliminated, the tumorous process will regress.

Direct tumor factors	Type of tumors
176 types of HPVs and wart viruses	Different carcinomas
papilloma cervix virus	Cervical cancer
HTLV-1 virus	Leukemia
EBV	Burkitt's lymphoma nasoph.
Hepatitis B, C, D and E viruses	Hepatocellular Carcinoma
KSHV or HHV-8 virus	Kaposi'sarcoma
HTLV-1 virus	Non-Hodgkin's lymphoma
HTLV-2 retrovirus	Hairy cell leukemia
Polioma BK, JC virus	Neural tumors
HPV-5, HPV-8, HPY-17 viruses	Skin cancer
HMTV virus	Mammary tumor
HPV 1, 2, 6, 11 16, 18, 34 virus	Cervical cancer

Tumor Causing Indirect Factors
Indirect factors do not directly cause tumors but accelerate the growing or the development of the tumor. Its elimination will not stop the growing of the tumorous tissues.

Indirect cancer factors	Type of cancer
Helicobacter pylori	Stomach cancer
Schistosomiasis	Bladder cancer
Liver flukes	Liver biliary cancer
Helicobacter hepaticus	Liver cancer

Borrelia B. sensu lato bacteria	Skin and breast cancer
Chlamydia species	Hodgkin's disease
Herpes Virus group	Prostate cancer
HIV virus	All types of cancers
Shigella bacterium	Ovary and testis cancer
Salmonella	Pancreatic cancer
E. coli bacterium	Bowel cancer

26.1.4. Immune Response in Tumorous Processes

All tumor diseases develop only in case of certain immuno deficiency of the affected person. The healthy immune system is able to eliminate the transformed tumor cells. The response of the immune system is a collection of the different protecting mechanisms against diseases by identifying and killing pathogens and tumor cells. The transformed tumor cells express viruses and other new antigens on their surfaces, which antigens are not to be found on normal healthy cells. These antigens are foreign to the immune system, their presence provoke an immune attack against the transformed tumor cells. The antigens expressed by tumors have several sources, some derive from oncogenic viruses f.i. *Human Papilloma Viruses*, which are the cause of all cancers, while others are the own proteins of the body, that exist at low levels in normal cells, but reach high levels in tumor cells. For instance, the tyrosinase enzyme, if expressed at high levels, it transforms melanocytes into melanoma cells. An other possible source of tumor antigens are proteins, normally important to regulate cell growth and its survival, which commonly mutate into cancer inducing oncogenes.

The main response of the immune system to tumors is the destroying of abnormal, virus infected cells using killer T cells, sometimes with the assistance of helper T cells. Tumor antigens are presented on MHC class I molecules similarly to viral antigens. Therefore killer T cells recognize the tumor cells as abnormal cells. NK cells can also kill tumorous cells in a similar way, especially if the tumor cells have less MHC class I molecules on their surface than normal; being a common phenomenon concerning tumors. Sometimes antibodies are producted against tumor cells offering their destruction to the complement system.

Some tumor cells evade the immune system and go on to become cancers. Tumor cells often have a reduced number of MHC class I molecules on their surfaces, avoiding thus the detection by killer T cells. Some tumor cells release products inhibiting the immune response; for example secreting cytokine TGF-β, which suppresses the activity of macrophages and lymphocytes. Moreover, an immunological tolerance may develop against tumor antigens, so that the immune system does no longer attack the tumor cells. A healty immune system is able to eliminate tumorous cells, immunodeficiencies do only occur if one or more of the components of the immune system are inactive. The ability of the immune system to respond to pathogens is diminished concerning very young and elderly persons, immune responses begin to decline in persons at around 50 years of age due to immunosenescence, regarding that an adult person may be infected by *HTLV, HBLV, Epstein-Barr Virus or/and Mycoplasma species*. Obesity, alcoholism and drug abuse are common causes of poor immune functioning. Malnutrition is the most common cause of immunodeficiency in developing countries. Diets lacking sufficient protein are associated with impaired cell-mediated immunity, complement activity, phagocyte function, IgA antibody concentrations and cytokine production. Deficiency of certain nutrients such as iron, copper, zinc, selenium, vitamins A, C, E, B6 and folic acid can also reduce the immune responses. The loss of the thymus at an early age by genetic mutation or surgical removal, results in a severe immunodeficiency and a high susceptibility to infections.

Immunodeficiencies can be inherited and acquired. The most frequent immunodeficiency syndromes include f.i. Job's syndrome (Hyper-IgE syndrome), Chronic granulomatous

disease), Reticular dysgenesis, Chediak-Higashi Syndrome, Shwachman-Diamond syndrome, Congenital agranulocytosis, Kostmann syndrome, neutropenia, complement defect, Leucocyte adhesion defect (LAD), Common variable immunodeficiency (CVID), Wiskott-Aldrich syndrome, other Severe Combined Immunodeficiencies (SCIDs), Adenosine deaminase deficiency (ADA), Nucleoside phosphorylase deficiency (PNP), acquired agammaglobulinemia, Ataxia telangiectasia, IgA deficiency, Chronic mucocutaneous candidiasis (CMC), ZAP-70 defect, Familial hemophagocytic lymphohistiocytosis (FHL) and the severe congenital agranulocytosis. Chronic granulomatous disease, in case of which phagocytes have a reduced ability to destroy pathogens, is an example of inherited, and connatal immunodeficiency. HIV and some types of cancer cause acquired immunodeficiency.

The most frequent and the most important cause of acquired immundeficiency is an infection causing immune damages, blocking the normal immune response. Such infections are caused f.i. by *HTLV, HBLV, EBV, CMV, Mycoplasma pneumoniae, Mycoplasma genitalium, Mycoplasma fermentans, Mycobacteria and others.* These infections inhibit the immune response. The immune deficiency is a part of a tumorous process. Cell-to-cell interactions can cause a cascade of events resulting in T- and B-cell activation, and, ultimately, in host's defense.

Inflammation is one of the first responses. Their symptoms are redness and swelling, caused by increased blood flow into the affected tissue. Inflammation is the product of eicosanoids and cytokines, released from injured or infected cells. Eicosanoids are, f.i. prostaglandins, causing fever and dilation of the blood vessels, associated with the inflammation, and leukotrienes, attracting certain white blood cells. Interleukins are cytokins responsible for the communication between white blood cells; chemokines promoting chemotaxis, and interferons which have anti-viral effects, such as shutting down the protein synthesis in the host cells. Growth factors and cytotoxic factors may also be released that recruit the immune cells to the site of the infection and promote the healing of every damaged tissue following the removal of pathogens. During an immune response there are many systems activated such as the complement system, the cellular barrier system, lymphocytes, killer T cells, helper T cells, B lymphocytes producting antibodies, etc. A normal immune system can eliminate the cell, infected by a cancer virus, but an inherited or acquired immunodeficiency will inhibit this elimination procedure.

A healthy immune system continually tries to recognize and eliminate all tumor cells; but if a tumor cell escapes this immune surveillance and grows to be too large to become killed by the immune system, cancer will be the result. Immune surveillance is most likely to be successful against virus-induced tumors which express foreign peptides. Tumors vary greatly in their immunogenicity, and even tumors with antigens, which can be recognized by the host immune system can evade this immune elimination. The lack of tumor rejection in case of an intact immune system is not always owing to the absence of recognizable antigens or owing to the absence of T cells which could recognize those antigens. Tumor-specific lymphocytes can be found in the blood, draining the lymph nodes. These lymphocytes can kill tumor cells, if the RFR technique eliminates the *infective agents from the immune system* and if RFR method eliminates every *Human Papilloma Virus.*

RFR method is able to eliminate both type of the infective agents, but is not able to eliminate the tumor cell, this restoring process is the task of the healthy immune system. The usage of RFR method and the tumor elimination is usually a long time procedure.

The principle scientific aim of the program of Tumor Immunology and Immunotherapy has to be developed. The RFR method must further be examined to detect and eliminate the pathogens and to apply immunological aspects for the prevention, the diagnosis and the treatment of the diseases, and RFR monitoring of pre-malignant and malignant diseases in order to follow the progression, regression of the disease, and the restoring to health.

Importantly, these four themes are dynamic and interactive, continually evolving to remain focused on clinical needs and translating programmatic data for treatment and prevention strategies, thought it is important to say that RFR method is not able to diagnose the stage of a tumorous process.

26.1.5. Diagnosis

A variety of special tests may be required to the further characterization of a cancer. Its diagnosis can be made by x-ray, CT, ultrasonography, MRI and PETscan, by using fiber-optic-instruments and a variety of laboratory tests such as tumor factor examinations etc.
The RFR method is not able to diagnose cancer!

26.1.6. Cancer Treatment

Immunotherapy
Cancers may be associated with, or leading to an altered immune response of the host, however, the relationship between the cause-and-effect is not yet exactly known. The effect of this type of treatment can be direct, caused by the replacement of normal lymphocytes as in case of chronic lymphocytic leukemia, or indirect, secondary to the treatment required to control the primary tumor, or associated with, as exemplified by situations in which blocking factors can be demonstrated.
To improve the immune system's ability to find and destroy cancer, researchers have developed biologic response modifiers. These substances stimulate the antitumor responses by increasing the number of tumor killing cells or by producing one or more biological mediators such as interleukins. These substances increase the direct tumor-killing mechanisms and the specific and aspecific immune responses.
Interferon is the best-known and most widely used biologic response modifier. This immunotherapy has only a slowly acting effect on cancer.
Killer cell therapy: the lymphocytes are exposed to interleukins-2 in order to produce lymphokine-activated killer cells, which will then be reinjected into the person suffering from malignant melanoma.
Humoral antibody therapy enhances the production of humoral antibodies of the patient. The cancer patient will be treated with the extracts of weakened tuberculosis bacteria.
Apoptosis is a form of programmed cell death, activated under physiological and pathological conditions in a number of immunological processes. A lot of substances such as sulfhydril compounds show to possess apoptosis-modulatory properties.
Surgery
Surgery is one of the oldest forms of cancer therapy. The prognosis of this treatment is determined largely by judging the severity and spreading of the cancer, which process is named staging. Some cancers can be cured by surgery, if treated in the early stages of the tumor, when the viral infection is not yet generalized.
Radiation Therapy
Radiation destroys, preferably the rapidly dividing cells. Usually this means cancer, but radiation damages normal tissues as well, especially tissues of cells normally reproducing rapidly, f.i. cells of the skin, hair follicles, gastrointestinal tract, ovary or testis, the bone marrow, and the reticuloendothelial system (RES). In case of an accurately targeted radiation, the procedure can protect the normal cells as much as possible. Radiation therapy plays a key role in curing certain cancers, including Hodgkin's disease, early stage non-Hodgkin's lymphoma, squamous cell cancer of the head and neck, seminoma, prostate cancer, early-stage breast cancer, early-stage non-small-cell lung cancer and medulloblastoma.
Radiation therapy can reduce symptoms if the cure of the tumor is not possible, as f.i. in some cases of multiple myeloma, advanced cancers of the lung, esophagus, head and neck

and the stomach. Radiation therapy can relieve also the symptoms caused by metastases in the bones or the brain. The insufficiency of radiation therapy is the fact, that it is unable to kill the virus, causing the cancerous process.

Chemotherapy

An ideal anticancer drug would destroy cancer cells without harming the normal cells. There does not exist a drug like this. Anticancer substances are grouped into several categories: biological alkylating agents, antimetabolites, plant alkaloids, antitumor antibiotics, enzymes, hormones and biologic response modifiers. There are often two or more drugs used in combination.

Antimetabolites interfere with some steps of the synthesis of DNA or RNA, in order to prevent pathological cell replications.

Plant alkaloids are drugs stopping the cell division and preventing the formation of new cells. **Hormone therapies** raise or lower the levels of certain hormones in order to limit the growth of cancers that either depend on those hormones, or are inhibited by them. Some breast cancers need estrogens for their growing. An antiestrogen drug Tamoxifen can block the effects of estrogen and thus shrink the cancer. Similarly, prostate cancers may be inhibited by estrogen or antitestosteron drugs.

The use of **biological alkylating drugs** is not adviseable, though they are still used in the clinical practice.

RFR method detects the virus and can eliminate it. Microorganisms should only be treated on their measured resonance frequencies!

Wart Human Papilloma Viruses: have different genetical structures with different resonance frequencies. (researchers determined 80-90 genetic structures of the HPVs).

Adenovirus species have 40-50 different known structures. The number of basis pars changes between 33112-35100.

Here there is only the list of the more frequent onces given.

The resonances of the most frequent HPV and primitive Retroviruses are: 300, 314-319, 343-347, 353, 389-392, 401-412, 418-438, 452-453, 459-464, 476-480, 484-488, 513, 517-521, 525-527, 538-545, 555, 564 kHz

The frequencies of HPVs affecting the cervix are: 402-410, 456, 464 kHz

The most frequent characteristic resonances: 404-405 kHz

The frequencies of plantar HPVs are: 403-408, 467-470 kHz

The frequencies of HPVs causing Condyloma are: 462-466, 476 kHz

The frequencies of Verruca vulgaris are: 329, 392, 403, 486-488 kHz

The frequencies of Adenoviruses are: 370-387, 393 kHz

The frequencies of CMV are: 305, 349, 408-411, 512, 530-536, 543, 548 kHz

The frequencies of EBV are: 337-339, 342-347, 352, 372-382, 403, 422, 491, 516, 518-519, 560 kHz

The frequencies of Herpes Simplex Virus-1 are: 290-294, 328, 336, 344-346, 377, 398, 420, 458, 478, 483-486, 527, 533 kHz

The frequencies of Herpes Simplex Virus-2 are: 341, 350, 352-365, 367-368, 374-375, 380-383, 413, 425, 434, 454, 540-542, 568 kHz

The frequencies of HZV are: 339, 347-349, 372, 383, 396, 409, 416-421, 460, 467, 474, 477, 544-545, 560 kHz

This list is not complete yet, as there are other subspecies having different wave frequencies in this range.

Bacteria producing colonial factors, according to Hulda Clark are as follows:

Escherichia coli: 356-357, 390-394 kHz (producting Cancer Associated Antigen)
(E. coli has a lot of plasmids with higher frequencies).

Staphylococcus aureus: 376-382 kHz (producting Epidermal growth factor)
(Staphylococcus aureus has a lot of plasmids with higher frequencies).

Salmonella: 328-330, 360-365, 382-386 kHz (CA-15-3, CA-125)

Shigella: 390-394 kHz (CA-15-3, PDGF, ILGF, AFP)
Proteus groups: 320-329, 333-339, 345-352, 408-416, 426, 516, 522-529, 535 kHz
Pseudomonas: 331-334 kHz
This list is not complete. There are other subspecies having different wave frequencies.
The cancerogen retrovirus first attaches itself to and then penetrates the host's cell. The virus RNA, the genetic code of the virus will be released into the cell. The enzyme necessary to reproduce and thus to perform the conversion into DNA is named reverse transcriptase. At this point the cancer virus easily becomes mutated, as this reverse transcriptase is prone to errors during the conversion of viral RNA to DNA. The viral DNA then enters the cell's nucleus. With the help of an enzyme named integrase, the viral DNA becomes integrated in the DNA of the cell. The DNA now replicates and reproduces RNA and proteins. The proteins form a long chain that must be cut into pieces after the virus leaves the cell. Every time a host cell divides; there will a new copy of the integrated viral DNA produced as well, which can take-over the functions of the cell, causing the cell to produce new virus particles. These new viruses are released from the infected cell to invade other healthy cells. A new virus is assembled from RNA and short pieces of protein. The virus travels through the cell membrane, wrapping itself in a fragment of the cell membrane. To become infectious to other cells, another viral enzyme must cut structural proteins within the budded virus causing them to be rearranged into the mature form of a cancer virus. The cancer virus is not permanently built up in the DNA of the host cell.
If these pathogens can interfere with the genetic material of a normal host cell and if the genetic information of the pathogen can embed itself partially or fully in that cell, the originally normal cell will produce mutated cells. Moreover, if the DNA repair function of the cell is blocked, the new genes in the cell induce an infinite multiplication of cells that will become tumors. Any process that disturbs the natural repairing mechanism of the host DNA (meaning the elimination of the defective gene parts and the re-synthesis of the original gene), helps the formation of tumors.
The loss of genetic information in the tumorous cells occurs as a consequence of the defective repairing mechanism.
In case of the existence of tumorous cells in the body, the immune system may still own its eliminating capacity, though it is usually limited, especially in case of patients with a suppressed immune system due to various causes. If the mutated cells can live and multiply, they will more and more get out of the control of the immune system.
It seems as if the presence of viruses would be necessary to maintain the cancerous process. If the cancer viruses (by their all pathological frequencies) are eliminated the tumor cells will die!
Complications caused by the treatment of cancers or sarcomas can arise, f.i. by the elimination of the primitive retroviruses with RFR method. For example in case of an intestinal tumor, or a brain tumor, the inflammatory edema may cause fatal pressure. An other complication of the RFR method can occur when the tumorous tissue get disintegrated. Perforation may also occur and cause bleeding f.i. in case of lung tumor.

26.1.7. The Most Frequent Resonances of Various Tumors

Adenoma: 442 kHz
Adenocarcinoma: 313, 368, 402-410, 426-438, 533, 552-558, 568 kHz
Astrocytoma: 343, 354, 436, 438, 450, 453 kHz
Basal cell (skin) carcinoma: 314, 389, 522-524, 540-542, 553-557, 583 kHz
Bladder tumor: 342-347, 402-412, 438-448, 524, 545 kHz

Breast tumor: 313-321, 343-346, 374, 397, 402-413, 427-436, 484, 502, 524, 540-543, 552-557, 578 kHz
Bronchial and lung tumor: 294-297, 372-376, 402-409, 426-439, 524, 538-540, 548 kHz
Carcinomas: 343-345, 400-410, 426-444, 458-469 kHz
Cervical polip: 294, 351, 379-384, 380, 443, 564 kHz
Cervical carcinoma: 392, 402-410, 412, 426-438, 443-448, 459-464, 476, 500 kHz
Cervix adenoma: 440-448 kHz
Cholesteatoma: 316, 404-408, 462 kHz
Cholesteatoma of the mastoid region: 316, 324, 406, 463 kHz
Chondrosarcoma: 513-521, 524-534 kHz
Colon carcinoma: 312-314, 335-346, 356, 392-393, 426-438, 524, 557 kHz
Droglioma: 436 kHz
Endometrium tumor: 426-436 kHz
Eosophagus tumor: 372, 426-439 kHz
Ewing's sarcoma: 318, 348-353, 370-372, 395-406, 450, 512-519, 523-527, 530-534 kHz
Feline (cat) leukemia: 338, 380-392, 422-434, 461-469, 510, 528 kHz
Fibroadenoma mammae: 354 kHz
Fibroma: 340, 353, 372, 395-397, 476, 513, 544 kHz
Fibrosarcoma: 342, 445-447 kHz
Gastric adenocarcinoma: 343-348, 426-438 kHz
Glioblastoma: 372, 402-409, 418-429, 437-439, 444, 459 kHz
Glioma: 438-448, 470-476, 554-556 kHz
Hairy cell leukemia: 318, 320, 399, 477, 493, 496-500 kHz
Hodgkin's lymphoma: 389-390, 564 kHz
Hypernephroma: 300, 324, 389-392, 402-409, 426-444, 448, 459-469, 540, 564 kHz
Kaposi's sarcoma: 331, 426, 508 kHz
Laryngeal polip: 391, 412 kHz
Larynx tumor: 370-376, 536, 580 kHz
Leucoplakia: 340-342, 353, 476, 513, 544 kHz
Leukemia B cell tumor: 486-487 kHz
Leukemia feline: 424-436, 461, 469 kHz
Leukemia lymphatic: 402-409, 426-432, 488 kHz
Leukemia myeloid: 418-422, 426-429, 432, 450 kHz
Leukemia T cell: 311, 330, 370-374, 420, 432-433, 452, 496, 536 kHz
Leukemia B cell: 307, 317-319, 329, 399, 435, 486-487, 523-525, 561-563, 574 kHz
Leukemia: 340, 353, 372, 402, 450, 513, 544, 567 kHz
Lipoma: 309-312, 360-363 kHz
Liposarcoma: 450, 488-496, 514, 520-527, 531-536, 550, 560-576 kHz
Liver carcioma: 324, 336, 343-347, 352-354, 372-382, 390-402, 420-427, 456, 475-479, 490, 510-514, 532, 538, 541, 561, kHz and the frequencies of the Hepatitis viruses.
Lymphosarcoma: 486, 492 kHz
Melanoma: 294-300, 322-328, 396, 442-448, 451-456, 465-470, 480-489, 490-496, 501-507, 533-545, 554-563 kHz
Meningioma: 390, 546-548 kHz
Multiple Myeloma: 372, 486 kHz
Myoma: 425-428, 460-464, 516 kHz
Myosarcoma: 404-408, 514 kHz
Nasopharyngeal tumor: 372-382, 408-410 kHz
Ovary tumor: 390-394, 402-410 kHz
Pharyngeal tumor: 407-411, 436-439, 538-542 kHz
Plasmacytoma: 486 kHz
Polyp nasal: 367-369, 550-554 kHz

Polyps in different tissues: 296-312, 318-319, 332-340, 348, 352-354, 366-368, 372, 409, 452-453, 459-464, 476-479, 513, 534, 544, 550-556 kHz
Precancerous state polyposis: 296-312, 318-319, 332, 340-348, 352-353, 367, 372, 452-453, 459-464, 476-479, 544, 554-555 kHz
Precancerous state: fibroid cysts in breast: 340, 353, 372, 396-400, 402, 410, 450, 544 kHz
Prostate adenoma: 292, 352, 382, 402-410, 452, 479-480 kHz
Prostate, other tumors: 310, 343-345, 353, 362, 372, 392, 402-410, 426-437, 470, 479-480, 506-513, 542-547 kHz
Rhabdomyosarcoma: 372, 401-411, 450, 459-464, 511-524, 533, 535-544, 558-559, 567 kHz
Salivary gland tumor: 372-383, 402-410, 426-439, 518-519, 526-538 kHz
Sarcoma: 470-476, 501, 510-515, 542-545 kHz
Skin cancers: 330, 332, 378-382, 544-545 kHz
Stomach carcinoma: 343-346, 372, 402-412, 418-422, 426-448, 458-466 kHz
Testis tumor: 344, 372, 390-394, 402-408 kHz
Thyroid carcinoma: 372, 402-412 kHz
Urinary tract tumor: 344-346, 372, 402-412 kHz
Uterine tumor: 426-437, 520 kHz
Vaginal carcinoma: 426-444 kHz
Wart, condyloma acuminatum: 462-478 kHz
Wart, plantar: 404-407, 468-477 kHz
Wart, verruca acuminata or filiformis, or peruviana: 329, 352, 392-396, 402-410, 448, 485-491, 502-510 kHz
This list is not complete, there are other subspecies having different wave frequencies.

26.2. Rare Tumors Caused by Simian Vacuolating Virus 40 (SV-40)

Simian vacuolating virus 40 (SV-40) is a polyomavirus infecting monkeys and humans. Between 1955 and 1963 SV-40 entered the human population as a contaminant of poliovirus vaccine in several countries, as the vaccines were prepared in cultures of rhesus monkey kidney cells. (The subsequent vaccines are already free of SV-40). SV-40 is thought to be widely distributed in the human population.
It is a DNA virus that can be a potential cause of tumors, but persists most often as a latent infection. Its virion adheres to the cell surface receptors of MHC class 1 and can penetrate into the cell through a caveolin vesicle. The eventual release of the viral particals occurs causing cytolysis and thus the death of the cell. The role of SV-40 infections in the tumorgenesis is yet unclear, but this virus can but very seldom be detected in human tumors, f.i. in ependymomas, mesotheliomas, non-Hodgkin lymphomas, osteosarcoma and choroid plexus carcinoma.
Diagnosis: by PCR and other virus examinations.
Treatment: there exists no specific virus killing therapy.
RFR method: can detect ad eliminate SV-40.
The resonant frequencies of SV-40 are: 331-332, 338-339, 343-345, 360-362, 379-380, 385-387, 405-407, 425-426, 440-443, 447, 450-451, 453-457, 480-481, 467-489, 494-498, 552-554 kHz

26.3. Brain Tumors

A **benign brain tumor** is an abnormal, but not malignant growth of tissue in the brain.

Several types of benign tumors can grow in the brain.
Schwannomas originate in Schwann cells which form a wrapping around the nerves.
Ependymomas originate in cells lining the interior surface of the brain, f.i in the walls of the fourth ventricule, affecting children and if being in the laterial ventricles and the spinal cord they affect mostly adults. This tumor is not sensitive enough to x-ray so that only the surgical removal can offer the chance of survival. The cause of this tumor is a *viral* infection.
Meningiomas originate in the meninges, lining the other surfaces of the brain. **Adenomas** originate in the gland cells, i.e. the **pituitary adenomas**, common particularly in late adulthood, and is discovered mostly when a patient begins to complain of visual disturbances. A partial or complete bitemporal hemianopsia progressing to blindness, with optic atrophy is the usual finding accompanied by the symptoms of an endocrine disorder. As the growth gets enlarged and extends laterally, an oculomotor palsy can occasionally be seen. **Osteomas** originate in the bony structures of the skull, while **hemangioblastomas** in the blood vessels.
Certain benign brain tumors, such as **craniopharyngiomas, chordomas, germinomas, teratomas, dermoid cysts** and **angiomas** may be present even already at birth.
Malignant brain tumors are all those that are able to invalidate and destroy the neighbouring tissues or which spread metastasized from elsewhere via the bloodstream to the brain.
The most common **malignant brain tumors** are **metastases** from cancers originating in another part of the body, these tumors may grow in one single locus of the brain or in several different parts.
Primer brain tumors originate within the brain. They are most often gliomas, growing from tissues surrounding and supporting the nerve cells. Certain types of **glioma** are malignant; f.i. glioblastoma multiforme.
This highly malignant **glioblastoma multiforme** can infiltrate the brain and grow enormously before being diagnosed. This tumor can be mottled gray, red, orange, or brown, depending on the degree of its necrosis and hemorrhage and whether they new or old. This highly vascular tumor can sometimes be mistaken for a hemangioma. Its characteristic pathologic findings are f.i. a number of pleomorphic cells with hyperchromatic nuclei, identifiable astrocytes with fibrils in combination with astroblasts, tumor giant cells, mitotic cells, necrosis, hemorrhage and thrombosis of the vessels.
Astrocytoma may occur anywhere in the brain or in the spinal cord. Its favored loci are the cerebrum, cerebellum, thalamus, optic chiasm and the pons. It is a slowly growing infiltrative tumor forming large cavites and pseudocysts. Calcium can often be deposited in this tumor and be experienced by x-ray of the skull. The tumor tissue contains well-differentiated astrocytes of various (fibrillary, protoplasmic and transitional) types. Its fast growing type is the anaplastic astrocytoma, being a most malignant astrocytoma and the glioblastoma multiforme, which all can increase the pressure in the brain causing thus headache, slowed thinking, and in severe cases sleepiness and coma as well.
The relatively rare **oligodendrocytoma** is usually a soft, solid tumor often calcifying. Some of the tumors are mixtures of astrocytoma and oligodendrocytoma.
Ependymoblastoma is usually a solitary mass in a cerebral hemisphere of adults, presumably arising from the wall of the lateral ventricle.
Reticulum cell sarcoma may be primary in the brain (microglioblastome) its symptoms being nearly identical to those of glioblastome multiforme, though the former is radiosensitive. Its cause is a viral infection.
Medulloblastomas are rare tumors usually affecting children before puberty.
Sarcomas and **adenocarcinomas** are unusual cancers growing from cells other then nerve cells. Brain tumors even those destroyed often cause intracranial hypertension, which fact must be considered when treating tumors. The prognosis of a patient with an intracranial

tumor is depending of the nature of the growth, location, and other factors of the tumor nevertheless, almost all intracanal tumors can end fatally. Death is often preceded by a critical rise in the intracranial pressure and a tentorial or foramen magnum herniation. The most malignant tumors, i.e. glioblastoma multiforme, medulloblastoma and metastatic carcinomas end fatally within few months, whereas the slowly growing meningiomas and astrocytomas often permit a survival for many years.

RFR method can cause necrosis in the tumor, peripherial edema and the increase of the intracranial pressure. These states may need a long-term corticosteroid therapy and treatment with diureticum.

Pseudotumor cerebri syndrome is a condition, in which the patients, mostly obes women of childbearing ages, complain of headache lasting for weeks, have a papilledema, slightly constricted visual fields and enlarged blind spots. A vague dizziness, diplopia due to a slight abducens weakness, or paresthesias of the upper extremities can also come about. Theories point to the increased resistance to cerebrospinal fluid outflow at the arachnoid granulations lining the dural venous sinuses. Cerebral venous outflow abnormalities may produce an idiopathic intracranial hypertension. The cerebrospinal fluid is acellular with normal protein content. The cause of this condition is a viral infection.

The **symptoms** of *hypertensive encephalopathy* are an extrem hypertension, retinal arteriolar changes with hemorrhages and exudates in the periphery of the optic fundi, headache, convulsions, confusion, stupor or coma. Signs of a renal disease are also present.

The **symptoms of a brain tumor**, either benign or malignant, depend on its size, growth rate and location. Symptoms include headache, poor balance in coordination, dizziness, double vision, nausea and vomiting. Extreme fluctuations in the blood pressure may occur. If the brain tumor is a metastasis from a distant cancer, the patient may have symptoms related to that cancer as well.

Diagnosis: symptomatically, by CT, MRI scan and x-ray.

Treatment: depending on the location and type of the brain tumor. If possible, the tumor should be removed surgically.

The RFR method of a brain tumor depends on the found resonance frequencies. The pathological resonances must be detected and the treatment should happen on these frequencies.

The most frequent resonant frequencies of brain tumors are concerning.

Astrocytoma: 343, 354, 436, 438, 450, 453-454 kHz

Glioma: 438-448, 476, 554 kHz

Glioblastoma: 328, 339, 368, 372, 402-409, 416-429, 438-439, 444, 459, 472-476, 513, 544, 554, 557-560 kHz

Droglioma: 436 kHz

Meningioma: 390, 546-548 kHz

The most frequent resonances of ependymoma are: 440-446, 464-470, 537 kHz

The most frequent resonances of the Pituitary adenoma are: 426-438 kHz

The most frequent resonances of ependymoblastoma are: 426-438, 440-446, 464-470, 537 kHz

The most frequent resonances of reticulum cell sarcoma are: 335-338, 470-471, 500-506, 510-515, 542-550 kHz

26.3.1. Meningioma

Meningiomas are the most common benign and malignant tumors of the brain, may occur intracranially or within the spinal canal. They arise from the arachnoidal cap cells, which reside in the arachnoid layer covering the surface of the brain. Meningiomas may be found at the surface of the brain, either over the convexity or at the skull base. In rare cases, they can develop in an intraventricular or intraosseous location as well. The classification of meningiomas is problematic as the arachnoidal cells may express mesenchymal and

epithelial characteristics, too. Similar tumors f.i. hemangiopericytomas or sarcomas may arise also from other mesodermal structures. The unequivocal meningiomas can be separated from other, less well-defined brain neoplasms. The exact genomic aberrations, responsible for their specific neoplasms can be examined by the methods in molecular biology.

The possible causative agents of the development of meningiomas can be a trauma, f.i. irradiation together with combined viral infections. Infections caused by *Simian vacuolating virus 40 (SV-40), Adenoviruses* and *HPVs* can be in close association with the development of meningiomas.

A genetic predisposition can play a role as well. The best-characterized and most common genetic alteration is the loss of the NF-2 gene on the chromosome 22q. This gene encodes a tumor suppressor protein.

The loss of chromosome 10 is associated with increased tumor grade, shortened time of recurrence and shortened survival. Progression into an anaplastic meningioma is associated with the involvement of the chromosomal site 17q.

The potential of the invasiveness of meningioma cells depends on the balance between the expression of matrix metalloproteinases (MMPs) and the tissue inhibitors of MMPs (TIMPs).

The increased incidence concerning women versus men and the presence of estrogen, progesterone and androgen receptors on some of these tumors may suggest an association between hormones and the risk of getting meningiomas.

Affecting children, the more common locations of meningiomas include the orbit, the temporal region, the foramen magnum, the tentorial region, the subfrontal base, the sellar region and the ethmoidal air sinus. As compared to adults, these tumors of children tend to be more aggressive regarding their growth rate, size, propensity to undergo malignant changes and recurrence rate.

Based on their behavior 3 different types of meningioma exist:

The benign (grade I) type does not invade the parenchyma of the brain but can invade the nearby bony structures

The atypical (grade II) type is a more agressively growing semimalignant form, with a higher recurrence rate, and

The malignant (grades III and IV), but rarely experienced form invading the parenchyma of the brain and showing the histologic signs of malignancy with a recurrence rate more than 70%: Its papillary type affecting mostly children is malignant, i.e. is invading agressively.

The formerly used term angioblastic meningioma is distinctly different from a meningioma, and is renamed hemangiopericytoma, a sarcoma with high recurrence rate and ability to metastasize.

Symptoms and signs of meningiomas are caused by the raised intracranial pressure, the involvement of the cranial nerves, compression of the underlying parenchyma and the involvement of the bone and subcutaneous tissues. A raised intracranial pressure can lead to papilledema, decreased mentation and even to brain herniation. Involving the cranial nerves anosmia, visual field defects, optic atrophy, diplopia, decreased facial sensations, facial paresis, decrease in hearing, deviation of the uvula and hemiatrophy of the tongue can come about. Compression of the surrounding parenchyma can cause pyramidal signs exemplified by pronator drift, hyperreflexia, positive Hoffman sign and the presence of the Babinski sign.

Optic nerve sheath meningiomas arise either from the cells of the arachnoid surrounding the intraorbital or the intracanalicular optic nerve, or, more often, are extensions of an intracranial meningioma into the orbit.

Their symptoms are usually a painless progressive visual loss with proptosis. Exophthalmos, headache, decreased visual acuity, ptosis diplopia can also come to pass.

Diagnosis: symptomatically. There are no specific laboratory tests to screen for meningioma. By CT, MRI and EPM. By biopsy. Enveloped in a thin capsule of the adjacent meninges, meningiomas are separated from the brain and the spinal cord.
Differential diagnosis: by distinguishing it from lesions associated with focal hyperostosis: osteomas, Paget's disease, neurosarcoidosis, tuberculosis, lymphoma, fibrous dysplasia, etc
Treatment: by surgical resection, radiotherapy, chemotherapy, and symptomatically f.i. by administering antiepileptic drugs, hydroxyurea and IFN-alpha, corticosteroids in order to reduce edema around the tumor, etc. Incompletely excised, malignant and multiple tumors are most likely to recur. The prognosis is excellent concerning patients whose meningiomas are completely resected and their causative viral infection is eliminated.
RFR method: detects and may eliminate all viral components of the tumor
The most frequent resonances are: 338, 344, 354-362, 379, 390, 406, 425-426, 428-438, 448-449, 480, 546-548 kHz

26.3.2. Ependymoma

Ependymoma is a neuroepithelial tumor arising from the ependymal lining cells of the ventricles. Its intracranial form is most common among children, while its spinal cord location occurs usually among adults. Intracranial ependymomas are intraventricular masses, while spinal ependymomas can be either intramedullary masses arising from the central canal or exophytic masses at the cauda equina.
Ependymoblastomas are primitive neuroectodermal tumors, distinct from ependymoma.
Based on their histologic characteristics these tumors include:
Grade I: myxopapillary ependymoma and subependymoma occuring in the region of cauda equina;
Grade II: ependymoma (with cellular, papillary, and clear cell variants);
Grade III: anaplastic ependymoma.
Ependymomas arise from oncogenetic events, in case of which *HPV and/or SV-40* viruses transform the normal ependymal cells into tumor phenotype cells.
Molecular heterogeneity can exist among histologically identical tumors. Studies identifying genetic defects (f.i. a loss of locus on chromosome 22, a mutation of p53 in malignant ependymoma, a recurring breakpoint at band 11q13, abnormal karyotypes frequently involving chromosome 6 and/or 16, and NF2 mutations) and the clustering of ependymomas in one family suggesting the loss of an ependymoma tumor suppressor gene in the region of the chromosome 22 locus 22pter-22q11.2) point to the role of genetic predisposition. This predisposition and the *viral infection* can cause together the development of ependymoma.
Symptoms varies depending upon the age of the patient and the location of the ependymoma. The neurologic signs of an intracranial ependymoma can be general or focal. The presence of symptoms prior to diagnosis usually varies from 3-6 months. Children with ependymoma in the fourth ventricle suffer headache, progressive lethargy, nausea and vomiting caused by the secondary developed obstructive hydrocephalus. Cranial-nerve palsies (VI-X) and cerebellar dysfunctions can also occur. Changes in mood, concentration and personality are often experienced.
Spinal ependymomas cause pain and progressive neurological damages of the ascending or descending nerve tracts. Papilledema, nystagmus and ataxia are common signs of infratentorial ependymomas. Hemiparesis, sensory loss, visual loss, aphasia, and cognitive impairment may be the symptoms of supratentorial lesions.
Cervical/thoracic spinal ependymoma can cause pain and paresthesia in the occipital and cervical regions, stiffness, weakness and the wast of the neck muscles and underneath of this tumor spastic tetraplegia or hemiplegia can also develop.

Thoracic ependymoma can be detected by sensory examinations as the testing the strength of intercostal muscles is difficult.
Lumbar ependymomas cause radicular pain and weakness associated with nerve root compression.
Tumors of the conus and cauda equina can cause pain in the back, the rectal area, or in the legs. Bladder dysfunction and impotence are early signs of patients with conus medullaris lesions. Conus lesions can but rarely cause a spontaneous pain, the pain of a cauda equina lesion is, in contrast, severe, involving the perineum, thighs and legs. Motor dysfunctions are symmetric in case of conus lesions and asymmetric in case of cauda equina lesions.
Diagnosis: symptomatically, by CT scan and MRI with and without the administration of an intravenous contrast material. By biopsy and histological analysis.
Differential diagnosis: by distinguishing it from astrocytoma.
Treatment: by conventional radiation therapy, radiosurgery, surgery, chemotherapy. By administering corticosteroids in case of peritumoral edema, and anticonvulsants in case of a supratentorial ependymoma. By administering combination chemotherapy regimens by the collaboration of a neurologist, neurosurgeon, neurooncologist and radiation oncologist in order to coordinate the treatment strategy.
RFR method: detects and may eliminate HPV and other pathogen microorganisms.
The most frequent resonances are: 338-339, 344, 354-362, 370-373, 379, 388-393, 406-408, 425-435, 437-439, 442-451, 543-548 kHz
Following the medical treatment the elimination of all viral agents is important and necessary. If the virus remains alive in the patient, the tumorous process will start again and proceed. Predictors of long-term survival are the extent of the surgical resection, the amount of the residual tumor observed postoperatively. Although lower tumor grade, infratentorial location in case of children, absence of tumor invasion within the brainstem, absence of metastases, an improved state and older age in case of childhood ependymoma are good prognostic signs, these factors are not significantly correlated with the long-term survival.

26.3.3. Astrocytoma

Astrocytomas are CNS neoplasms consisting predominantly cells, derived from immortalized astrocytes. Several types of astrocytic tumors are recognized, f.i. pilocytic astrocytoma, subependymal giant cell astrocytoma, pleomorphic xanthoastrocytoma, which can be also named low-grade astrocytoma, anaplastic astrocytoma and glioblastoma. There are numerous grading schemes created based on histopathologic characteristics, such as the Bailey and Cushing grading system, Kernohan grades I-IV, WHO grades I-IV, and St. Anne/Mayo grades 1-4.
WHO grade I corresponds to pilocytic astrocytoma,
WHO grade II corresponds to low-grade (diffuse) astrocytoma,
WHO grade III corresponds to anaplastic astrocytoma, and
WHO grade IV corresponds to glioblastoma multiforme (GBM).
The regional effects of astrocytomas are the compression, invasion and destruction of the brain parenchyma. These regional effects lead to the ruining of the normal parenchymal function, an elevated intracranial pressure (ICP), an increased blood volume and to an increased cerebrospinal fluid volume, causing clinical neurological signs and symptoms.
Such **focal neurological symptoms** are f.i. weakness, paralysis, sensory deficits, cranial nerve palsies and seizures which all can point to the locus of the tumor. The first symptoms are mostly headache, a depressed mental state and other focal neurological problems. The signs of an increased ICP include headache, nausea, vomiting, decreased alertness, cognitive impairment, papilledema, ataxia, etc. Localizing and lateralizing signs, including cranial nerve palsies, hemiparesis, sensory levels, alteration of deep tendon reflexes and

the presence of pathological reflexes (f.i. Hoffman and Babinski signs) can also come to pass.
Familial clustering of astrocytomas is well known and described as inherited neoplastic syndromes, f.i. Turcot syndrome, Neurofibromatosis type 1 (NF1) syndrome, and p53 germ line mutations (f.i. Li-Fraumeni syndrome).
Mutations in specific molecular pathways, such as the p53-MDM2-p21 and p16-p15-CDK4-CDK6-RB pathways, are associated with the development and progression of astrocytomas [redacted]
The etiologic factors of **juvenile pilocytic astrocytomas** are a genetic predisposition and *combined viral infections*. The transformation into a malignant high-grade tumor can be caused by a new, other pathogenic virus, though a secondary infection like this does but rarely occur.
Juvenile pilocytic astrocytoma is associated with neurofibromatosis type 1 (NF1), an autosomal dominant disorder characterized by the development of benign and some malignant tumors. Optic gliomas, 60% of which represent pilocytic astrocytomas, are common tumors. The tumor can be solid, with or without a cystic degeneration. This brain tumor consists of well-differentiated pilocytes and microcysts containing some mucopolysaccharide material.
The signs and symptoms depend on the location of the tumor. The most common symptoms are caused by the increased intracranial pressure as a result of mass effect or hydrocephalus. These include nausea, vomiting, headache, ataxia and visual complaints.
Diagnosis: by laboratory examinations including those of the basic metabolic panel (CHEM-7), CBC, prothrombin time (PT), and activated partial thromboplastin time (aPTT). By CT scans and MRI with and without any contrast material. By imaging methods including the pretreatment of the patient with tumor-specific proteins tagged with fluorescent molecules. By PET scan, SPECT and technetium-based imaging in order to distinguish a solid tumor from an edema, to differentiate tumor recurrence from radiation necrosis, to localize structures etc. By biopsy.
Treatment: symptomatically in case of seizures by administering phenytoin, carbamazepine, etc. By administering corticosteroids in case of vasogenic edema around the tumor. By surgery in order to remove or debulk the tumor, provide tissue for histologic examinations, etc. By stereotactic biopsy.
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequent resonances are: 328, 340-345, 353-355, 370-374, 436-439, 442-453, 555-558 kHz
An effective RFR method can lead to tumor necrosis and to a peripheral edema increasing the pressure in the brain, so that this therapy must be done very carefully by administering diuretics and corticosteroids if necessary. The most common sign of an effective RFR method is headache.

26.3.4. Glioma

Brainstem gliomas (BG) are primary brain tumors, which seldom metastasize or spread to affect another part of the body. BG are tumors occuring in the region of the brain referred to as brain stem, which is the area between the aqueduct of Sylvius and the fourth ventricle. These tumors can be sorted, according to their distinct anatomic locations, into diffuse intrinsic pontine, tectal and cervicomedullary gliomas. Intrinsic pontine gliomas have a graver prognosis than the tectal and cervicomedullary ones.
Optic nerve glioma is the most common primary neoplasm of the optic nerve. Together with the reduction of the visual acuity in the affected eye, the tumor sometimes produces additional symptoms as it grows. Its less serious form, the benign optic glioma affects mostly children. Another form, the aggressive glioma, affects adults, is frequently fatal even if treated.

A glioma is characterized on the basis of either its locus of origin, or its focality, direction and the extent of its growth, degree of brainstem enlargement, degree of exophytic growth, and the presence or absence of cysts, necrosis, hemorrhage and hydrocephalus. Most of them are located in the pons; though the medulla and midbrain can also be involved. Brainstem gliomas and optic nerve gliomas are highly aggressive brain tumors. The pathophysiological manifestation of the tumor is determined by its anatomic location. In case of tectal gliomas, the compression of the fourth ventricule can result in hydrocephalus. Cranial nerve and long tract signs can commonly be observed in case of pontine and cervicomedullary gliomas.

Their symptoms can be double vision, headache, nausea and vomiting, lack of facial control (droopy eyelids), weakness and fatigue, papilledema and seizures. These signs can develop but slowly or subtly, and may go unnoticed for months. In other cases, the symptoms may arise abruptly. A sudden onset of symptoms occurs in case of more rapidly growing, high-grade tumors. A painless proptosis is usually the sign of young patients with optic glioma. Optic atrophy and a later developing reduced visual acuity are frequently present. A large lesion may compress the optic chiasm, causing nystagmus or other symptoms. Hypothalamic symptoms, such as the altering of appetite or sleep, can also occur. Massive lesions may compress the third ventricle, resulting in obstructive hydrocephalus accompanied by headache, nausea and vomiting. Optic nerve gliomas develop in stages, beginning as generalized hyperplasia of glial cells in the nerve, leading to a complete disorganization with the loss of neural characteristics in the nerve and nerve sheaths. It is difficult to distinguish an optic nerve glioma from a perioptic meningioma. The exact origin of benign optic gliomas is uncertain.

Brainstem glioma and optic nerve glioma can be caused by a combined infection with *Mycoplasma* species, *HPV*, *HTLV* and *other infectious agents*.

Diagnosis: by MRI, and/or PETscan. Sometimes by surgery and biopsy.

Treatment: Mostly by chemotherapy and/or radiation therapy, seldom by neurosurgery.

RFR method can detect and eliminate all the infectious agents.

The most frequent resonances of gliomas are: 370-376 *(HTLV)*; 438-448 *(HPV)*; 442-451 *(Mycoplasma)*; 476-479 *(HPV)*; 543-545 *(HPV)*; 554 kHz

The most frequent resonances of glioblastomas are: 328, 339, 368, 370-376 *(HTLV or EBV)*; 402-409 *(HPV)*; 418-425 *(HPV)*; 437-448 *(HPV)*; 442-451 *(Mycoplasma fermentans)*; 476-479 *(HPV)*; 512, 543-545 *(HPV)*; 554-558 kHz

26.3.5. Glioblastoma

Glioblastoma multiforme (GBM), composed of a heterogenous mixture of poorly differentiated neoplastic astrocytes, is the most common and aggressive type of primary malignant glial brain tumors, accounting for more than 50% of all primary brain tumor cases and 20% of all intracranial tumors. This brain tumor located mostly in the cerebral hemispheres, primarily affects adults. Less commonly it can develop in the brain-stem in case of children and in the spinal cord. These tumors may develop from lower-grade astrocytomas. Glioblastomas can be classified as primary or secondary. The rapidly growing primary GBM cases account for about 60% of glioblastoma cases and affect adults older than 50 years. Secondary GBM cases develop typically in younger patients from a low-grade or an anaplastic astrocytoma (WHO Grade II and III).

Among the astrocytic neoplasms, glioblastomas contain the greatest number of genetic changes due to the accumulation of multiple mutations, such as the

Loss of heterozygosity (LOH): found on chromosome arm 10q, the most frequent gene alteration of the primary and secondary glioblastomas. This mutation appears to be specific for GBM and is rarely found in other tumors. This mutation is associated with short-term survival. LOH at 10q and one or two additional gene mutations are frequent alterations and important factors in the development of glioblastomas.

Mutations in p53, a tumor suppressor gene, were among the first genetic alterations identified in astrocytic brain tumors. This p53 immunoreactivity is associated with tumors affecting younger patients.
Epidermal Growth Factor Receptor (EGFR) gene plays a role in the control of cell proliferation. Its multiple genetic mutations are apparent, including the overexpression of the receptor as well as its rearrangements resulting in truncated isoforms.
MDM2: the amplification or overexpression of this protein, bound to p53 lessening its activity, constitutes an alternative mechanism to escape from the p53-regulated control of cell growth.
Platelet-derived growth factor–alpha (PDGF-alpha) gene can act as a major mitogen for glial cells by producing PDGF-alpha binding to the PDGF receptor (PDGFR). Amplification or overexpression of this factor is a pathway leading to secondary glioblastomas.
There are several other known gene mutations (f.i. PTEN, MMACl-El, MAGE-El, etc.) all resulting in an aberrant, enhanced proliferation of astrocytes.
Glioblastoma multiforme is characterized by small areas of necrotizing tissues surrounded by anaplastic cells (pseudopalisading necrosis). The characteristic presence of hyperplastic blood vessels can differentiate it from astrocytoma Grade 3.
An inherited or acquired predisp osition and combined *infections with different viruses* play an important role in the etiology of gliomas.
The symptoms usually include nausea, vomiting, headache and hemiparesis. A progressive memory loss and personality changes caused by the involvement of the temporal and frontal lobes are characteristic. The symptoms depend highly on the location of the tumor, more than on its pathological properties. The tumor patient can quickly develop symptoms, but may occasionally remain asymptomatic until this tumor reaches an enormous size.
Diagnosis: By MRI examinations glioblastoma in its early stage may mimic more benign brain lesions. In case of a suspected GBM observed by CT or MRI a stereotactic biopsy or a craniotomy should be made, by which latter as much of the tumor as possible should be removed at the same time.
The histopathology of GBM is extremely miscellaneous. These tumors are composed of poorly differentiated, pleomorphic astrocytic cells with nuclear atypia and brisk mitotic activity. Necrosis is characteristic and microvascular proliferations are common.
Treatment: of glioblastomas is palliative including surgery, radiotherapy and chemotherapy (Temozolomide). Symptomatically by administering anticonvulsants (f.i. phenytoin, carbamazepine, tegretol, etc.) and corticosteroids.
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequent resonances are: 328, 339, 368, 370-374, 406-411, 424-426, 437-439, 442-451, 472-476, 512-515, 540-545, 555-558 kHz
An effective RFR method can lead to tumor necrosis and a peripheral edema increasing the pressure in the brain, so that this therapy must be done very carefully by administering diuretics and corticosteroids if necessary. The most common sign of an effective RFR method is headache.

26.4. Adenocarcinoma in General

Adenocarcinoma is a tumor originating in glandular or epithelial tissues. Epithelium is derived embryologically from the ectoderm, endoderm and mesoderm. Epithelial tissues include the skin, glands and the tissues lining the cavities and organs of the body. To be classified as adenocarcinoma, the cells of the cancer must have secretory properties, but do not necessarily need to be part of a gland. Adenocarcinomas can arise in many tissues of the body due to the ubiquitous nature of glands within the body. The well differentiated adenocarcinomas resemble the glandular tissues which they are deriving from, while the

less differentiated ones do not. Though these glands do not secrete the same substances, but as long as a cell has an exocrine function, it is considered to be glandular and, therefore, its malignant form is an adenocarcinoma. Endocrine gland tumors, such as insulinoma, pheochromocytoma, VIPoma (i.e. a vasoactive intestinal peptide secreting tumor), etc, are typically not referred to as adenocarcinomas, but rather as neuroendocrine tumors. If the glandular tissue of a tumor is abnormal, but benign, it is defined as adenoma. Benign adenomas typically do not invade other tissues and but rarely metastasize. Malignant adenocarcinomas invade other tissues and often metastasize if given enough time.

The immune system of the body attacks and eliminates not only bacteria, viruses and other foreign substances, but also cancer cells. A cancer cell is not a foreign on, far rather is an altered cell which does not respond anymore to the body's normal system of controlling cell growth and reproduction, as this cancer cell is infected with one or more viruses, and its genetic substance is mutated in a precancerous or cancerous manner. The abnormal cells can continue to grow, resulting in cancer processes. If one's immune system is suppressed the cancer may be manifested. The development into a precancerous state occurs usually slowly, but getting into this state the further carcinomatous progression into an adenocarcinoma can be very quick.

The most frequent parts of the body, where adenocarcinomas may arise are the brest, colon, lung, prostate, stomach, pancreas, vagina and the uterine cervix.

Diagnosis: by physical examinations, by x-ray, CT, MRI, ulrasound exams and by biopsy followed by histological examinations, etc.

Treatment: (depending of the stages of adenocarcinomas) by surgery, radiology and chemotherapy.

RFR method: detects and may eliminate all pathogen viruses and bacteria.

The self-antigens are on the surface of the cells, but a healthy immune system does not attack its own cells. If a cell becomes infected by a new cancerogen virus, this will be expressed on the surface of the infected cell. The immune system may take these new antigens as if they were foreign and can be able to destroy these cancerous cells. However, even a normally functioning immune system may be unable to destroy and eliminate all cancer cells. Certain colonial factors originating from bacteria increase the growth of cancer cells.

The most frequent resonances found in case of adenocarcinoma are: 314-319, 343-347, 426-438, 442-451, 525-527, 543-545 kHz

The most frequent resonances found in case of adenoma are: 438-442 kHz

26.5. Head and Neck Cancers

The term head and neck cancer refers to a group of biologically similar cancers originating from the upper aerodigestive tract, including the lips, the oral cavity (mouth), the nasal cavity, the paranasal sinuses, pharynx and larynx. Head and neck (HN) cancers are strongly associated with certain environmental and lifestyle risk factors, including irritation (f.i. the gastroesophageal reflux disease is held to be a significant risk factor for cancer of the larynx), wood dust, tobacco smoking, exposures to paint and gasoline fumes, plastic byproducts, asbestos, alcohol consumption, and certain strains of the sexually transmitted *HPVs*. Some head and neck cancers may have a viral etiology. The presence of DNA of *HPV* in the tissue of oral and tonsil cancers, can point to its predisposing potency to develop oral cancer even in case of tobacco non smokers and those who are no alcoholists. *HPV, EBV, CMV, HTLV* and *Mycoplasma fermentans or penetrans* infections are predisposing factors to develop head and neck cancer. Mutations in the p53 gene, found to be associated with SCC, correlate with the drinking and smoking habits of the patients.

If detected early, head and neck cancers are well curable by a combination of chemotherapy and radiation therapy, though surgery can also be effective.

Squamous Cell Carcinoma (SCC) represents more than 90% of all head and neck cancers. This malignant tumor has a regional distribution involved in the biological activity of the neoplasm. Its behavior depends on the locus of its origin. This type of carcinoma arises from keratinizing or malpighian epithelial cells. According to histologic evaluations SCC is characterized by the presence of keratin or „keratin pearls". These are well-formed desmosome attachments and intracytoplasmic bundles of keratin tonofilaments. Epidermoid can be substituted for squamous.

Symptoms: The most common loci for Squamous Cell Carcinoma are the tongue, the soft palate, the anterior tonsillar pillar, the floor of the mouth and the retromolar region.

SCC usually begins as a superficial circumscribed erythematous slightly elevated asymptomatic alteration of the skin (i.e. erythroplakia), which can be either a carcinoma in situ or an invasive carcinoma. Squamous Cell Carcinoma in situ is named also Bowen's disease. Some precarcinomatous lesions are pure white; known as leukoplakia, 10% of which usually develop into carcinoma in situ or invasive carcinoma. Squamous Cell Carcinoma s may appear as plaques, nodules, or verrucous papules as well. They may be scaly or ulcerated, white, red, or brown. Verrucous carcinoma has a more favorable prognosis because its low propensity for lymph nodal and distant metastasis. Painful and tender lesions point usually to the perineural invasion of this tumor.

Oral squamous cell cancers are common in the oral cavity, including the inner lip, tongue, floor of the mouth, gingiva and the hard palate. Cancers of the oral cavity are strongly associated,with tobacco use, especially with the use of chewing tobacco or „dip", as well as with heavy alcohol use. Cancers of this region, particularly the tongue, are more frequently than other head and neck cancers treated by surgery.

Nasopharyngeal cancer arises in the nasopharynx, the region where the nasal cavities and the Eustachian tubes are connecting the upper part of the throat. While some nasopharyngeal cancers are biologically similar to the common HNSCC, the „poorly differentiated" nasopharyngeal carcinoma is distinct in its epidemiology, biology, clinical behavior and treatment, and is by many experts treated as a separate disease.

Oropharyngeal cancer begins in the oropharynx, the middle part of the throat that includes the soft palate, the base of the tongue and the tonsils. Squamous cell cancers of the tonsils are more often associated with *HPV* infections than cancers of other regions of the head and neck.

Hypopharynx includes the pyriform sinuses, the posterior pharyngeal wall and the postcricoid area. Tumors of the hypopharynx frequently are in an advanced stage when diagnosed, and have the worst prognosis of all pharyngeal tumors. They tend to metastasize early owing to the extensive lymphatic network around the larynx.

Laryngeal cancer begins in the larynx or „voice box." Cancer may occur on the vocal cords themselves („glottic" cancer), or on tissues above and below the true cords („supraglottic" and „subglottic" cancers respectively). Laryngeal cancer is strongly associated with tobacco smoking.

Its surgeries can include partial laryngectomy (removal of a part of the larynx) and total laryngectomy (removal of the whole larynx). If the whole larynx is removed, the person is left with a permanent tracheostomy opening and has to learn to speak again in a new way by help of intensive teaching and speech therapy and/or an electronic device.

Tracheal cancer is a rare malignant tumor, biologically similar in many ways to other head and neck cancers, and is sometimes classified as such.

Most tumors of the salivary glands differ from the common carcinomas of the head and neck in their etiology, histopathology, clinical presentation and therapy.

Other rare tumors arising in the head and neck include teratomas, adenocarcinomas, adenoid cystic carcinomas and mucoepidermoid carcinomas. Malignant melanoma and lymphomas of the upper aerodigestive tract develop but very rarely.

Dissemination: In advanced cases dissemination to the ipsilateral submandibular or/and the jugulodigastric lymph nodes can occur, the sign of which will be a mass in the neck. Remote bone and organ metastases are usually not associated with early oral primary squamous cell cancers, in these cases a second, more advanced primary cancer (f.i. an upper aerodigestive one or a lung cancer) can be responsible for them.
Diagnosis: symptomatically, by CT, MRI, biopsy and by histological examinations.
Treatment: by surgery, x-ray, by administering citostatics (and antibiotics, if needed).
(Surgery is mostly used to resection (removal) some of the lymph nodes in order to prevent the further spreading of the illness). Radiation therapy (i.e. Intensity-modulated radiotherapy) is the most common form of its treatment. If the cancer has metastasized or is widespread, the older form of radiation treatment will most effectively slow down or stop the progression of the disease.
RFR method: detects and may eliminate the pathogens causing the cancer!
Surgery and x-ray are not able to stop the systemic infection caused by *HPV, HTLV, EBV, mycoplasma* and *other microorganisms*.
The frequencies of HPV and other Papova viruses found in case of various HN tumors are:
Regarding squamous cell carcinomas: 343-345, 400-410, 426-438 kHz
Regarding adenocarcinomas: 427-438 kHz
Regarding the adenoid cystic carcinomas: 343-345, 400-410, 426-438, 440-444, 458-469 kHz
Regarding mucoepidermoid carcinomas: 370-378, 408-411, 427-437, 518 kHz
Regarding melanoma: 279-300, 322-328, 442-456, 465-470, 480-489, 490-496, 501, 533-543, 544, 554-563 kHz
As to lymphomas: see its special Chapter.
As to teratomas: 329-332, 442-451, 543-545, 568-572 kHz
The most important EBV frequencies are: 372-383, 518-519 kHz
The frequencies of CMV are: 406-410, 530-536 kHz
The frequencies of HTLV are: 297-299, 307, 311-315, 320-340, 354, 359, 365-367, 370-376, 382-383, 397-400, 406, 416, 428-439, 453-455, 474-476, 480-482, 484, 487-490, 493-504, 523-530, 540-545, 570-578 kHz
The frequencies of Mycoplasma species are: 307-308, 321-324, 442-451, 491-495 kHz (one of them can always be found)
In case of these diseases the usage of RFR method must last for a long time.

26.6. Salivary Gland Tumors

Salivary gland tumors (SGT) can be benign tumors, tumorlike conditions or malignant neoplasms. These glands are sorted into the major and minor salivary gland groups. The parotid, the submandibular and the sublingual glands belong to the major salivary glands. The minor glands, can sometimes be seen only by microscopy, are located everywhere in the upper aerodigestive submucosa. SGTs affect mostly persons about 50 years. About 80% of the SGTs are tumors of the parotid gland. Neoplasms of the submandibular gland develop but very seldom. The most common benign tumor is the benign mixed tumor, or pleomorphic adenoma-adenocarcinoma.
EBV, CMV, Mycoplasmas and in addition one of the *HPVs* cause the development of lymphoepithelial tumors of the salivary glands. Genetic alterations, such as allelic loss of tumor suppressor genes, monosomy and polysomy of chromosomes and structural re-arrangements may also be co-factors causing the development of SGTs. Certain kinds of radiation therapies to the neck and head can increase the risk of salivary gland cancer.
According to the multicellular theory of SGTs the pleomorphic adenomas originate from the intercalated duct cells and the myoepithelial cells; while the oncocytic tumors from the striated duct cells; the acinic cell tumors from the acinar cells; and the mucoepidermoid

and squamous cell tumors from the excretory duct cells. Different tumors are caused by different *Human Papilloma Viruses* (their resonances being 343-347, 405.5 kHz and 402-410 kHz or in case of an *adenocarcinoma* the resonance frequencies being: 427-438 kHz). *Mycoplasma pneumoniae*, or *Mycoplasma fermentans* is the most frequent coinfection found in case of SGTs. A subtype of *HPVs* is the predominant cause of the tumorgenesis, the *mycoplasma* coinfection supports this tumorous process.

The classic presentation of a benign SGT is a painless, slow-growing mass on the face (parotid gland), on the angle of the jaw (parotid tail, submandibular gland), or on the neck (submandibular gland) or a swelling at the floor of the mouth (sublingual gland). A sudden increase in size may indicate an infection, a cystic degeneration and a hemorrhage inside the mass, or a malignant degeneration. Benign SGTs are freely mobile, and the facial nerve function remains normal.

Benign epithelial SGTs include the pleomorphic and monomorphic adenomas, Warthin tumor, the intraductal papilloma, oncocytoma and the sebaceous neoplasms.

Benign nonepithelial tumors (of mesenchymal origin) include hemangioma, angioma, lymphangioma (cystic hygroma), lipoma and neural sheath tumors. Hemangiomas are the most common SGTs affecting children usually involving the parotid and less often the submandibular gland. These vascular tumors are present early in life, grow rapidly affecting infants from 1-6 months and involute gradually in 1-12 years. Lymphangiomas are located mostly in the head and neck region of infants and children and develop due to lymphatic sequestration of primitive embryonic lymph ducts.

Pleomorphic adenomas (i.e. benign mixed tumors) are the most common tumors of the salivary glands most often located in the tail of the parotid gland. If found in the minor salivary glands, the hard palate and the upper lip are their frequent locations.

Malignant tumors of the salivary glands occur less commonly, are characterized by a sudden and rapid growth and by its early spreading into the whole neck area through the lymphatic vessels and the blood vessels. They are usually different carcinomas, such as adenocarcinoma, acinic cell carcinoma, adenoid cystic carcinoma, malignant salivary mixed tumor, mucoepidermoid carcinoma, etc. Most small salivary gland tumors begin in the palate.

Symptoms of cancers are usually a painless or painful lump mostly in the area of the ear, jaw, lip, or inside the mouth. Difficulty in swallowing or by widely opening the mouth, weakness of the face muscles, permanent pain in the affected head or neck area. Other characteristic signs of a malignant lesion are rapid growth, paresthesia, hoarseness, skin involvement, a fixed lesion and cervical lymphadenopathy.

Diagnosis: by physical examinations and by the patient's hystory, by CT, MRI, PETscan, ultrasound examinations, by endoscopy, and by biopsy (fine needle aspiration) and histological examinations, by virus and mycoplasma detection with PCR or other techniques.

Treatment: must be planned by doctors expert in that field. Salivary gland excisions are indicated if symptomatic and if the recurrent chronic gland infections prove to be refractory to conservative treatments. The standard therapies are radiation, surgery and chemotherapy. The mode of treatment is depending usually on the stage and location of the tumor and the patient's general state of health.

RFR method: detects and may eliminate the pathogens causing the tumor.

The resonances of HPVs are: 314-319, 343-347, 401-410, 418-426, 427-438, 442-448, 452-453, 456-466, 467-479, 488-496, 501-507, 513-521, 525-527, 533-545, 556-564 kHz

The resonances of Mycoplasma species are: 321-324, 442-451 kHz

The resonances of EBV are: 372-373, 518-519 kHz

The resonances of CMV are: 408-410, 530-536 kHz

One or more HPVs accompanied by EBV, CMV and Mycoplasmas are usually the cause of these tumors. If a new subtype of HPVs infects the tumor, the type and the histological findings of the tumor may change.

26.7. Lingual Cancer

The tongue is the most common intraoral locus of cancer. The incidence of oral cancers widely varies in the world. The cancer of the tongue is a serious public health problem with significant mortality and morbidity. The incidence of oral cavity cancers is among persons who smoke approximately 6 times higher than among those, who do not. An exposure to tobacco causes progressive sequential histological changes in the oral mucosa. These changes eventually lead to neoplastic transformation in case of a prolonged period of exposure, but can be reversible if the tobacco exposure is discontinued. Some people seem to be more susceptible than others concerning the effects of these irritants. This increased vulnerability can be genetic, familial and acquired (f.i. due to immunosuppression, syphilis, Plummer-Vinson syndrome, chronic candidiasis). These risk factors and the underlying pathogenesis do usually not disappear following surgical excision and/ radiation cancer therapy.

Mutations in tumor suppressor genes in patients with cancers of the oral cavity were reported in studies. Nitrosamines constitute the most abundant carcinogens present in tobacco. These agents can damage the DNA, by leading to point mutations. These point mutations lead to the deregulation of tumor suppressor genes, the best characterized of them being TP53 located on chromosome 17. Other oncogenes associated with oral squamous cell cancers are c-myc and erb -b1.

CMV, EBV and *HPV*, the last being an epitheliotropic DNA virus, are other possible etiologic agents for carcinogenesis, transforming the cells to malignant phenotypes. *Human Papilloma Virus* was detected in various amounts in persons with oral dysplasia, leukoplakia and malignancy. See also Human Papilloma Virus in its special Chapter. *Mycoplasma species and/or Human T-cell Lymphotropic Viruses* also count always as coinfectors.

Riboflavin and iron deficiency are known to produce dysplastic changes in the oral mucosa. This may partly explain the relationship of oral cancer with alcoholism, which can result in riboflavin deficiency.

More than 90% of the oral cavity cancers are **squamous cell carcinomas**. The other ones are of minor salivary gland origin. Lymphoma, melanoma and sarcoma do rarely occur in the tongue.

Pleomorphic adenoma being of dual origin from epithelial and myoepithelial elements is the most common, benign neoplastic tumor of the salivary gland and the parotid gland. Areas of squamous metaplasia and epithelial pearls may be present in it. This tumor is not enveloped, but is surrounded by a fibrous pseudocapsule of varying thickness. This tumor extends across the normal glandular parenchyma in form of a finger-like pseudopodia, which, nevertheless, is not a sign of malignant transformation. This tumor often displays characteristic chromosomal translocations between chromosomes 3 and 8. Pleomorphic adenoma may develop into **adenocarcinoma**.

Leukoplakia and erythroplakia are lesions most likely to get transformed and become malignant. The term leukoplakia is defined as a clinical white patch of the mucosa that cannot be characterized clinically or pathologically as any other disease. Leukoplakia is a premalignant alteration caused by a chronic irritation of the mucous membranes, resulting in an increased rate of epithelial and connective tissue proliferation. The head and neck region has a rich vascularity, which is an advantage for treating cancer in this region. This vascularity, nevertheless, increases the chance of metastasis. The shells of tumors that metastasize to the head and neck are richer in vascularity than the tumor tissue itself.

Squamous Cell Carcinoma is by far the most common malignant tumor of the tongue, having typically 3 morphologic growth patterns: exophytic, ulcerative and infiltrative. The infiltrative and ulcerative types are mostly observed on the tongue. Early carcinomas smaller than 1 cm may be detected only by routine clinical examination. As to symptomatic tumors, their most common sign is an indurated, ulcerated area of the tongue. The induration may extend deep into the tongue musculature and the root of the tongue. In many cases a regional lymphadenopathy is also present. There is a correlation recognized between tumor size, nodal presence, metastasis, and their eventual prognosis.
Diagnosis: by biopsy and histological examinations.
Treatment: surgically and with radiation therapy.
RFR method can detect and may eliminate all pathogen microorganisms.
The most frequent resonances are: 317-319, 321-324, 343-347, 354, 370-383, 393, 404, 408-410, 427-438, 442-451, 493-495, 518-519, 538, 543-545, 572-586 kHz

26.8. Tumors of the Ocular System

The orbit is an anatomically complex structure containing the globe; the extraocular muscles; fat; and the vascular, glandular, nerve and connective tissues. Eye cancers can be **primary** ones, startig within the eyes and **metastatic cancers** spreading to the eye from an other organ. Breast cancer and lung cancer are the two most common cancers spreading to the eye from an other organ. Other less common sites of origin are f.i. leukemia, prostate, kidney, thyroid and skin cancers and colon lymphoma.
Tumors in the eye and orbit can be benign, as f.i. dermoid cysts, or malignant, as f.i. rhabdomyosarcoma and retinoblastoma.
Basal Cell Carcinoma is the most common eyelid tumor. This tumor can grow around the eye but rarely spreads to other parts of the body. Other types of common eyelid cancers include **squamous carcinoma, sebaceous carcinoma** and **malignant melanoma.**
The most common malignant primary intraocular tumor is the **uveal melanoma** and the **ciliary body melanoma.**
Retinoblastoma is the most common malignant intraocular tumor of children. **Medulloepithelioma** again, occuring either in the ciliary body or in the uvea of the eye, is an other eye cancer of children.
The most common orbital malignant tumor is the **orbital lymphoma.** This tumor can be diagnosed by biopsy with histopathologic and immunohistochemical analysis. Most patients with orbital lymphoma can be treated with chemotherapy or radiation therapy.
The Causes of intraocular tumors are a genetical predisposition and a combined infection caused by *Mycoplasma species*, *HTLV*s and *HPV* species.
Symptoms: Protrusion of the eye is an important clinical manifestation of **orbital diseases.** (This ocular phenomenon is referred to as proptosis or exophthalmos. According to Henderson, exophthalmos describes the orbital manifestations of endocrinopathies, while proptosis is used to describe the change in the anteroposterior axis of the eye resulting from orbital masses. Besides proptosis, one should also note the displacement of the eye in planes other than the anteroposterior dimension. Hertel's exophthalmometry is a well-accepted tool to quantify proptosis.)
In their early stage, choroidal, ciliary body and uveal **melanomas** may show no symptoms. As the tumor grows, its symptoms can be blurred vision, decreased vision, double vision, eventual vision loss and if they continue to grow, the tumor can cause retinal detachment. Sometimes the tumor can be visible through the pupil.
Nevi are benign frecklese in the eye. These should be investigated regularly in order to rule out its development into a melanoma.
Iris and conjuctival melanomas are dark spots. Every spot continuing to grow on the iris and the conjunctiva must be examined.

Symptoms of **retinoblastoma** are: strabismus (crossed eyes), a whitish or yellowish glow through the pupil, decrease/loss of vision. Sometimes the eye may be red and painful. Retinoblastoma can occur in one or both eyes, and affects babies and young children. When a photograph is made of a child's eye, a white/yellow dot instead of the red eye reflex can indicate a tumor or some other kind of eye disease.
Intraocular tumors cause sometimes glaucoma. In cases of an unexplained glaucoma, the possibility of an ocular tumor must be considered.
The epidemiology, prognosis and therapy depends on the specific tumor type. In many instances the tumor is not directly visible so that different methods of indirect visualization may be needed and used.
Diagnosis: by complex ophthalmic examinations, by biopsy
Treatment: by laser therapy, chemotherapy, plaque therapy, radiotherapy and surgery.
(Surgery: enucleation, evisceration, exenteration, iridectomy, choroidectomy, iridocyclectomy and eyewall resection.)
RFR method can detect and eliminate all specific pathogen microorganisms.
The most frequent resonances of Human Papilloma Viruses are:
in case of Rhabdomyosarcoma: 401-408, 513-521, 525-527, 533-538, 543-545, 558 kHz
in case of Basal cell carcinoma: 541-545 kHz
in case of Squamous cell carcinoma: 543-545 kHz
in case of Malignant melanoma: 501-507, 533-543, 556-562 kHz
in case of Retinoblastoma and Medulloepithelioma: 452-453, 525-527, 538, 543-545 kHz
in case of Lymphoma: 404-406, 420-426, 488 kHz
in case of Sarcoma: 446-447, 470-473, 488-496, 513-534 kHz
The most frequent resonances of HTLV are: 330, 370-376, 432-433, 454-455, 496 kHz
The most frequent resonances of Mycoplasma are: 321-324, 365-366, 440, 442-451, 493-495 kHz

26.9. Lung Cancers

Most cancers in the lungs originate either in the cells of the lungs, or are metastases spreading to the lung from a primary tumor present in some other part of the body.
More than 90 percent of lung cancers start in the bronchi. Their type can be **squamous cell carcinoma, small cell,** or **oat cell carcinoma, large cell carcinoma** and **adenocarcinoma**. Alveolar cell carcinoma originates in the air sacs of the lung. Though this cancer can be a single tumor, it often develops at the same time in more loci of the lungs. Less common lung tumors are the bronchial adenoma, that may be cancerous as well, chondromatous hamartoma and sarcoma. Lymphoma involving the lungs is a cancer starting in the lymphatic system. Many cancers starting elsewhere in the body may spread to the lungs. These start mostly from the breast, colon, prostate, kidney, thyroid, stomach, cervix, rectum, testis, bone and the skin.
Lung cancers can caused by simultaneously present Human Papilloma Viruses, Human T-cell Lymphotropic Viruses, Epstein-Barr Virus and different mycoplasmas. Genetic predisposition also plays a role in manifesting the disease, while other predisposing factors are smoking, uranic substances and silicosis.
The **symptoms** of a lung cancer depend on its type and location. The main symptom is usually a chronic cough. People with chronic bronchitis who develop lung cancer often notice that their coughing becomes worse. Cancer can start from TBC of the lungs. The sputum coughed up may be streaked with blood. Forced or abdominal respiration, wheezing caused by narrowing the airways in which or around the tumor is growing, collapse of the part of the lung caused by the blockage of the affected bronchus, chest pain caused by pleuritis are typical signs of developed large lung tumors. If the cancer invades the blood vessels, it may cause severe bleedings. Symptoms can include weakness, loss of

appetite and weigh. Fluid accumulations around the lung leading to shortness of breath, hypoxia and heart failure may also be caused by lung cancers.
Horner's syndrome is characterized by a drooping eyelid, small pupil, sunken eye and reduced perspiration on the affected side of the face all caused by lung cancer growing into certain nerves in the neck area. Cancers at the top of the lung may grow also into the nerves that supply the arm, causing pain, weakness or numbness of the affected arm. Nerves supplying the voice area may also be damaged, so that the voice get hoarsen. A cancer may grow directly into the esophagus, or may put pressure on it. A lung cancer growing into the heart can lead to arrhythmias, enlargement of the heart and pericarditis.
The cancer may grow into or around the superior vena cava. The obstruction of this vein can cause shortness of breath, headache, distorted vision, dizziness and drowsiness.
Lung cancers can cause paraneoplastic syndromes, too, i.e. metabolic changes, diseases and symptoms of the nerves, muscles etc. far away from the lungs, not related to the size or location of the lung cancer [redacted] Some lung cancers secrete hormones or hormonlike substances (see below).

26.9.1. Squamous Cell Carcinoma of the Lungs

Squamous Cell Carcinoma of the lung accounts for 30-40% of cases of bronchogenic carcinoma, and it has a strong association with smoking. The lesion is usually located centrally, and it is among all bronchogenic carcinomas most likely to cavitate. Squamous cell carcinomas grow intraluminally and but seldom metastasize distantly (less than 20% of cases). The way of their spreading is direct extension to the local lymph nodes. This type of carcinoma is commonly associated with hypertrophic osteoarthropathy. Hypercalcemia due to a parathormone-like peptide created by the tumor can also be often observed. Tumors of squamous histology can sometimes elicit a sarcoid reaction in nodes, resulting in a nodal enlargement without metastatic spread.
The RFR method detects the most frequent resonances, such as: 321-324, 370-376, 402-410, 420-426, 428-437, 442-451, 494, 544-546 kHz

26.9.2. Adenocarcinoma of the Lungs

Adenocarcinoma occurs with a frequency of 30-40%, surpassing the incidence of squamous cell carcinoma. Its lesion is located peripherally in approximately one half of the cases, and it is associated with smoking. Adenocarcinoma may arise from a previous scar, it rarely forms a cavity, and an eccentric pattern of calcification can be observed. An early propensity of metastases to the lymph nodes, pleura, adrenal glands, central nervous system (CNS) and the bones is noted.
The RFR method detects the most frequent resonances, such as: 316-319, 321-324, 370-374, 376-387, 393-394, 408-410, 426-438, 442-451 kHz

26.9.3. Bronchoalveolar Cell Carcinoma

Bronchoalveolar Cell Carcinoma is a subtype of adenocarcinoma accounting for 5% of bronchogenic carcinomas. Although an association with smoking has not been established, a substantial percentage of patients have a significant smoking history. The incidence of bronchoalveolar cell carcinoma is increased in patients who have an underlying interstitial lung disease, parenchymal scaring and exogenous lipoid pneumonia. Bronchoalveolar cell carcinoma is classified on the basis of histopathologic features as being **mucinous** and **nonmucinous**. The mucinous variant is the most common (80%) and arises from columnar mucus-containing cells. This variant is often multicentric, appears occasionally with bronchorrhea, and has a bad prognosis. The nonmucinous form arises from type II pneumocytes or Clara cells, is more often localized, and has a better prognosis. Bronchoalveolar carcinoma may spread to the other lung by means of transbronchial

spreading called aerogenous spread. These tumors can also grow along the pulmonary interstitium without destroying the architecture of the lungs (lepidic growth).
Bronchoalveolar carcinoma may appear in a variety of forms, such as solitary pulmonary nodule (45%), multiple nodules (25%), and consolidated (30%). Its presentation as a solitary pulmonary nodule is associated with the best prognosis. These nodules can be sharp or poorly defined, and may be cavitated. In case of 30% of patients, an associated pleural effusion is noted, as well as a hilar or mediastinal lymphadenopathy.
The RFR method detects the most frequent resonances, such as: 299, 316-319, 321-324, 337-344, 370-384, 408-410, 426-438, 442-451, 530-536 kHz

26.9.4. Large Cell Carcinoma of the Lungs

Large cell carcinoma accounts for only 5-10% of bronchogenic carcinomas and is strongly associated with cigarette smoking. The lesion occurs peripherally growing rapidly, with early metastases and a poor outcome. A subtype of large cell carcinoma is the giant cell carcinoma. It is highly malignant and has a poor prognosis.
The RFR method detects the most frequent resonances, such as: 321-324, 370-374, 427-438, 442-451, 536 kHz

26.9.5. Small Cell Lung Cancer

Small Cell Lung Cancer (SCLC) has been found to be strongly associated with cigerette smoking. SCLC is a most aggressive tumor. Despite its neuroendocrine features, it is not yet clear, whether the origin of this tumor is a neuroendocrine cell (Kulchitsky cell) or an undifferentiated airway epithelial cell. Small cell lung cancer arises in peribronchial locations and infiltrates the bronchial submucosa. Widespread metastases occur early in the course of the disease, spreading often to the mediastinal lymph nodes, liver, bones, adrenal glands and brain. In addition, the production of a variety of peptide hormones leads to a wide range of paraneoplastic syndromes. The most common paraneoplastic syndromes are the syndrome of inappropiate secretion of antidiuretic hormone (SIADH) and the syndrome of ectopic adrenocorticotropic hormone (ACTH) production. Moreover, the autoimmune phenomena may lead to various neurological syndromes. SCLC mostly originates in the proximal airways such as the lobar bronchi or main bronchi. A small percentage of these tumors originate in the peripheral areas of the lung. This tumor is provoking a limited fibrotic or inflammatory response. Consequently, the tumor spreads to the submucosal and perivascular connective tissues, to the lymphatics, and to the blood vessels at an early stage, resulting in early nodal and metastatic deposits.
Extrathoracic nonmetastatic manifestations and paraneoplastic syndromes (Peripheral neuropathies) are more common in case of SCLC than in case of NSCLC. The neuromuscular neuropathies include the Lambert-Eaton myasthenic syndrome and polymyositis. The associated neuropathy can involve the peripheral and autonomic nerves and is associated with anti-Hu antibodies. Cerebellar degeneration, encephalomyelitis and limbic encephalitis (rare) has also been associated with SCLC. The cerebral symptoms of patients with SCLC are often caused by brain metastases. There have been described many cases of cutaneous peripheral neuropathies; including acanthosis nigricans and hypertrichosis lanuginosa. Endocrine syndromes associated with SCLC are f.i. Cushing's syndrome caused by secreted adrenocorticotropic hormones, the syndrome of inappropriate antidiuretic hormone (ADH) secretion, hyperparathyroidism, carcinoid syndrome, gynecomastia, caused by secreted gonadotropins, hyperpigmentation, caused by secreted melanocyte-stimulating hormones, hypoglycemia caused by secreted insulin-like substances, and hypocalcemia, caused by secreted calcitonin.
Many changes have been noted in the cellular genetics of SCLC, for example an abnormal deletion in chromosome 3, alterations in the retinoblastoma gene on chromosome 13 and

the presence of BCL2, C-myc, or N-myc oncogenes. Until now, none of these changes could replace the traditional morphologic criteria used to diagnose SCLC.
The 5-year survival rate of small cell lung cancer is 1-5%, its median survival being about 6-10 months.
RFR method detects mostly the following resonances: 294-297, 321-324, 397, 402-410, 427-438, 434-436, 440-451, 470-473, 476-479, 488-496, 513-519, 533-548, 556-558, 589-591 kHz
The most frequent resonances found in case of bronchial adenomas are: 321-324, 434-451, 513-534 kHz
The most frequent resonances found in case of lung sarcomas are: 404-408, 440-452, 446-447, 470-473, 499-496, 513-534 kHz
Diagnosis: symptomatically, by x-ray, CT, PET, PET with fluorodeoxyglucose (FDG), bronchoscopy. By biopsy, by analyzing exudates on tumor cells, etc.
Treatment: is depending on the stage, location and the patient's general state of health. By surgery, radiation therapy, chemotherapy (in case of small cell lung cancer) and their combination. By treating the metastases and the primary tumor, symptomatically and palliatively as well.
RFR method: can detect and eliminate the causative pathogens! Lung cancers are caused *by primitive retroviruses,* which must be eliminated.

26.10. Fibrocystic Breast Disease

Fibrocystic breast disease (named also fibroglandular changes, fibrocystic changes, chronic cystic mastitis, mammary dysplasia or benign breast disease) is characterized by fibrocystic breast lumps i.e. fibroadenomas, which are small, noncancerous, solid nodules composed of fibrous and glandular tissues. This benign fibrocystic breast disease can cause pain of the breast, cysts, and non-cancerous breast lumps often affecting women, mostly young ones and teenagers, but rarely men. The vast majority of breast lumps are benign. Many women, mostly between 35-50 years, experience generalized lumpiness in their breasts, usually in the upper, outer area. It usually disappears with menopause though may continue in women taking estrogen for hormone replacement. This kind of lumpiness is quite common and does not mean an increased risk of developing breast cancer.
Many breast lumps are actually cysts (fluid-filled sacs) that may grow bigger at the end of a woman's menstrual cycle, when her body is retaining more fluid. Not all breast lumps are cysts, they may also be fibroadenomas, usually found in younger women. Infections or severe injuries can also cause lumps in the breast. Tumors of fatty tissues named lipoma and blocked milk ducts named intraductal papilloma are also benign tumors of the breast.
The cause of cysts and fibroadenomas is a viral infection caused by *Human Papilloma Viruses.*
The signs and symptoms of fibrocystic breast diseases are one or more lumps in the breasts, may or may not be painful, as well as discharge from the nipple, tenderness and/or sensitivity of the breast. Some cysts are very small, others can be as large as a hen's egg. If applying pressure, larger cysts may change their shape slightly and can be moved around a bit under your skin. Most fibroadenomas have a firm, smooth, rubbery feeling and a well-defined shape.
Diagnosis: by physical examinations, by mammography, ultrasound, CT, MRI and biopsy.
Treatment: by administering high dose vitamine-E, symptomatically.
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances are: 340, 353-354, 370-373, 396, 402-410, 440-453, 476, 513, 544 kHz
The virus causing breast fibroadenomas has a special frequency, such as: 354, 442-451 kHz

26.11. Breast Cancer

Breast cancer is a very frequently developing tumor. It is classified by the tissue it originates and by the extent of its spreading. Cancer may start in the milk glands, milk ducts, connective tissue and but very rarely in the fatty tissue as well. Different types of breast cancers progress differently. Some grow very slowly and spread to others parts of the body only after getting enlarged. Others are more aggressive, growing and spreading quickly. However, the same type of cancer may progress differently concerning affected women.

Breast cancer is a complex infection caused by cancerogen viruses (i.e. by *primitive retroviruses*) and by *bacteria* producing colonial growth factors. The often found CA-125 and CA 15-3 tumor markers are originated from bacteria as colonial grow factors, the tumor adsorb them so that they get concentrated on the surface of the cancerous cells. Their tumorgenesis lasts from the beginning of the humanity as evidence by our body's ability to produce tumor necrosis factors and tumor suppressor genes, and to repair DNS mutations. These abilities and respond mechanisms of the patients developing breast cancers get easily exhausted being damaged by these *primitive retroviruses*.

In situ carcinoma, meaning cancer in loco, is an early cancer without invading or spreading other tissues than it is originating from.

A ductal carcinoma is most frequently an in situ tumor, starts in the epithelial cells of milk duct. This type of cancer only occasionally can be felt as a lump and may appear as tiny specks of calcium deposits i.e. micro calcifications detected by a mammogram. This calcium deposit is a result of immune protection.

A lobular carcinoma in situ, starts to develop usually before menopause in the epithelium of the milk glands.

Invasive breast cancers, which are able and used to spread into and destroy other tissues, may remain localized or metastasized within other organs, mostly the bones.

Risk factor for breast cancer are atypical hyperplasia, chronic inflammation of the mammal tissues, prolonged administration of oral contraceptives or estrogen replacement therapy, breast cancer gene, familiar breast cancer history, previous breast cancer, obesity after menopause and over-age.

Breast cancer affecting a first degree relative person (i.e. mother, sister, daughter, grandmother) increases a woman's risk to developing brest cancer, as the breast cancer gene causes a familial disease. The other important cause of breast cancer is a viral infection which can be found in the family. Men can also develop breast cancer, but their chance of developing it is very little.

Recently, there are two different genes (may be of retrovirus origin) identified from breast cancers occuring in two separate small groups of women. Woman having one of these genes, has a very high risk for developing breast cancer. However, if this woman develops breast cancer, her risk for dying of breast cancer is not necessarily greater than concerning other women with breast cancer. The incidence of ovarian cancer is also increased in families which fact is associated with one of the breast cancer genes. I think, that the breast cancer gene has an affinity to the *breast cancer virus*.

Women who have increased number of milk ducts are at increased risk to develop breast cancer. Even in case of these women, the risk is moderate unless an abnormal tissue structure, atypical hyperplasia, fibroid cystosis, mastopathia fibrosa cystica is found by biopsy.

Symptoms the evaluation of symptoms is difficult, as breast pain is not a sign of breast cancer, though about 20 percent of women suffering this cancer have pain without a lump. Usually the first symptom is a lump, which levels different from the surrounding breast tissue. Scattered, lumpy changes in the breast, especially the upper, outer region, can be precancerous. In its early stage, the lump may move freely beneath the skin when pushed with the fingers. Later on, the lump can not be moved at all. In case of advanced cancers

swollen bumps, festering sores and other skin alteration may be experienced. Chronic mastopathia with inflammation is a precancerous state. In case of an inflammatory breast cancer the breast looks as if infected, i.e. is warm, red and swollen.
Diagnosis: by physical examination and familial history, by mammography, ultrasound scanning, by biopsy. By x-ray, CT, MRI in order to find metastases.
Treatment: depends on the type and stage of the tumor. By surgery, radiation therapy, chemotherapy, by administering hormone blocking drugs and their combination. By BMRs (biologic response modifiers such as INF, IL-2, LAK TNF etc.)
RFR method: detects and eliminates the cancer virus and the pathogenic bacteria! One must treat with RFR method for only three weeks to see, whether RFR method was effective, or not.
Mastopathia cystica may be a precancerous state, in 10-20% of these cases a tumor will later on develop.
The most frequent resonances of breast cancer HPV in case of milk duct wall cancer are: 315-321 kHz
The most frequent resonances of breast cancer HPV in case of adenocarcinoma are: 426-438 kHz
The most frequent resonances of mastopathia cystica are: 314-321, 340, 353-354, 372, 396-402, 410-413, 442-451, 476, 513, 525-527, 538, 543-545 kHz
The most frequent resonances of atypical mastoplasia are: 314-321, 340, 353-354, 373, 400, 410-413, 442-451, 476, 513-515, 550-551, 565, 579 kHz
The most frequent resonances of ductal cancer are: 314-319 kHz
The most frequent resonances of acinar cancer are: 426-438 kHz
The other resonances of breast cancer are: 314-321, 324, 335-336, 346, 353, 370-374, 394, 432, 440-451, 454, 482, 488, 502, 504-507, 578 kHz
This frequency list shows the way of the cancerous process.

26.12. Thyroid Cancers

Thyroid cancer can be any one of the four main types of malignancy of the thyroid: such as papillary, follicular, anaplastic and medullary. Benign thyroid nodules do offen develop, and it is clinically difficult to distinguish these bening nodules from malignant thyroid lesions. The various different thyroid tumors are caused by different cancerogen infective viruses and their genetic basis of predisposition can also differ. A genetic predisposition without a viral infection does not cause any thyroid cancer or metaplasia. Only 5% of the thyroid nodules are malignant. Thyroid carcinomas originate from 2 cell types present in the thyroid gland.
The endodermally derived follicular cell gives rise to papillary, follicular, and probably, anaplastic carcinomas.
The neuroendocrine-derived calcitonin-producing C cell gives rise to medullary carcinoma of thyroid (MTC).
Thyroid lymphomas originate from intrathyroid lymphoid tissue, while sarcomas from connective tissue in the thyroid gland
A radiation exposure significantly increases the risk for thyroid malignancies, particularly for papillary thyroid carcinoma. Low-dose radiation exposures by imaging studies do not have a tumorigenic effect. A radiation targeting the thyroid gland (f.i. iodine-131 ablation of the thyroid) or a high-dose external-beam radiation therapy does not appear to increase the risk of papillary thyroid carcinoma. Populations with low dietary iodine intake have a high risk for follicular and anaplastic carcinomas of thyroid.
The activation of receptor tyrosine kinases (RET/PTC, TRK, MET) by rearrangement or gene amplification, acts specific on the transformation of thyroid follicular cells into papillary thyroid carcinomas. These rearrangements produce chimeric proteins with tyrosine kinase activities that contribute to the development of the malignant phenotype.

About half of the patients with sporadic papillary carcinoma have RET gene rearrangement.
The somatic point mutation in BRAF gene is the most common mutation present in papillary thyroid cancers. This gene encodes a serine/threonine kinase, which acts on the RAS-RAF-MEK-MAPK signaling pathway. RAS point mutations play also significant role in etiology.
The significantly increased risk for thyroid cancer of a person whose relatives have already had thyroid cancers suggests a genetic susceptibility to these tumors. There exists a significant correlation between papillary thyroid carcinomas and HLA-DR7 alleles.

Papillary carcinoma

This illness is the most common thyroid malignancy, represents about 80% of patients suffering from thyroid carcinoma and affects women 3 times more frequently. Papillary carcinoma and follicular carcinoma are well-differentiated thyroid carcinomas. The affected persons are mostly about 40 years old. This illness can be familial, either alone or in association with familial adenomatous polyposis (Gardner syndrome). Radiation exposure, especially in childhood, is associated with its development. Their latency period is about 10-20 years.
Papillary carcinoma is a slow-growing tumor arising from the thyroxine (T4)- and thyroglobulin-producing follicular cells of the thyroid. The cells are TSH sensitive, take up iodine and produce thyroglobulin.
Tumors grow directly through the thyroid capsule and can invade the surrounding structures, f.i. the trachea, leading to hemoptysis and airway obstruction. The cancerous process can involve the recurrent laryngeal nerves causing a hoarse, breathy voice and sometimes dysphagia as well. About 10% of these papillary carcinomas develop distant metastases, affecting typically the lungs and bones. Its prognosis is usually favorable.

Follicular carcinoma

Follicular carcinoma is the second most common thyroid malignancy representing about 10% of all thyroid cancers. These tumors arise also from the follicular cells of the thyroid, its cancer cells are TSH sensitive as well, taking up iodine and producing thyroglobulin. They can be differentiated from benign follicular adenomas by their tumor capsule and vascular invasion. The differentiating follicular adenomas from follicular carcinomas by cytology and frozen section analysis is extremely difficult. The rate of distant metastases is higher than that of papillary carcinoma. Metastases affect mostly the lungs and the bones.

Anaplastic cancer

Anaplastic cancer accounts for less than 10% of thyroid cancers and affects mostly elderly women. This cancer grows very quickly, and is an invasive tumor. Focal necrosis and hemorrhages are characteristically present in the tumor, which often extends through the capsule of the thyroid gland itself. Anaplastic thyroid carcinoma is believed to arise from a preexisting, well-differentiated thyroid carcinoma.

Medullary cancer

In case of medullary cancer excessive amounts of calcitonin and other hormones are produced, causing unusual symptoms. This cancer often spread through the lymphatic system to the lymph nodes and via the blood to the liver, lungs and bones. Medullary cancer can develop along with other types of endocrine cancers causing thus multiple endocrine neoplasia syndromes.
Patients usually belong to families with known familiar medullary thyroid cancer, though new germline mutations can also occur. Patients with new germline mutations are at risk for passing on the familial form of this cancerous syndrome.
The FMTC syndromes consist of MEN 2A, MEN 2B, and FMTC. They are inherited in an autosomal dominant fashion. Children inheriting an FMTC syndrome suffer 100% risk for developing MTC [redacted]

MEN 2A (Sipple syndrome) consists of MTC, pheochromocytoma (in the 50% of the patients), and hyperparathyroidism (in the 10-20% of the patients).
MEN 2B consists of MTC, pheochromocytoma (in the 50% of the patients), marfanoid habitus and ganglioneuromatosis.
The familial medullar thyroid cancer consists of medullar thyroid cancer alone, developing usually in adulthood. This familial medullar thyroid cancer is biologically most aggressive in MEN 2B syndrome, in which situation the thyroid cancer develops affecting children about 10 years old, grows rapidly and metastasizes quickly.

Primary thyroid lymphoma

Primary lymphomas of the thyroid gland represent approximately 2-5% of all thyroid malignancies. Most thyroid lymphomas are **non-Hodgkin B-cell tumors**. The next most common histologic type is the low-grade malignant lymphoma of mucosa-associated lymphoid tissue (MALT), though Hodgkin lymphoma, Burkitt's lymphoma, and T-cell lymphoma have also been reported.
This lymphoma is highly associated with chronic lymphocytic thyroiditis (Hashimoto thyroiditis). Its local extension into the aerodigestive tract or into the surrounding tissues may cause dysphagia, dyspnea, or symptoms of pressure in the neck. It can cause vocal fold paralysis, hoarseness and regional and distant lymphadenopathies as well.

Sarcoma of the thyroid gland

Sarcomas that arise in the thyroid gland are uncommon. These aggressive tumors arise from stromal or vascular tissues in the gland. Their treatment can be a total thyroidectomy. being unresponsive to chemotherapy. Its recurrence is common, and the patient's prognosis is sad.
Diagnosis: by biochemical examinations, hormon product examinations, ultrasound, CT, MRI, PETscan and by biopsy with histological analysis.
Treatment: By surgical excision in case of papillary and follicular carcinoma. By lobectomy, isthmectomy and total thyroidectomy depending on the type and the stage of the tumor and other influencing factors.
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequent resonances of thyroid cancer are: 316-321, 363, 370-374, 402-412, 416-420, 426-438, 439-448, 451, 489-493, 517-521, 525-527, 554-555 kHz

26.13. Double Parathyroid Adenoma

Double parathyroid adenoma can cause primary hyperparathyroidism (HPT) characterized by an inappropriate excess of parathyroid hormone (PTH) secretion. The elevated PTH level causes hypercalcemia and hypophosphatemia.
The primary function of the parathyroid glands is to regulate calcium homeostasis by producing PTH in response to hypocalcemia. PTH stimulates the bone resorption, which in turn leads to calcium release. In case of primary HPT the caused hypercalcemia is believed to be responsible for the clinical manifestations of the disease.
The clinical symptoms and disorders, wich are believed to be the result of bone resorption and the high serum concentration of calcium, are nephrolithiasis, bone pain, arthralgia, muscle pain, peptic ulcer disease, pancreatitis, fatigue, depression, anxiety, calcification in several different tissues, calcium deposit in the central nervous system and mental disturbances.
Genetic predisposition, gen abnormalities and viral infections can be involved in the pathogenesis of these adenomas and multiple adenomas.
Diagnosis: by noninvasive biochemical examinations of PTH levels, scintigraphy, ultrasonography, CT and MRI. By biopsy with histological analysis.
Treatment: surgically.
RFR method can detect and may eliminate the pathogens.
The most frequent resonances are: 402-410, 442-451, 493-495, 530-536 kHz

26.14. Gastric Cancers

More than 90 percent of stomach cancers are adenocarcinomas, other stomach cancers may be leiomyosarcomas, carcinoids, lymphomas and sarcomas.
Gastric carcinoma is the most common cancer in the world following lung cancer and is a major cause of morbidity and mortality. The cancerogen process of this tumor involves transformations f.i. first from gastritis to gastric atrophy, then to metaplasia, to dysplasia, and, finally, to cancer.
Helicobacter pylori can cause antral gastritis and if the patient has a *HPV* infection in his stomach as well, this bacterium can be a cofactor in the development of a gastric cancer. Patients with *H pylori* gastritis are 3-6 times more likely to develop gastric cancer than persons without this infection. There can exist several other different precancerous gastric conditions including chronic atrophic gastritis, pernicious anemia, Ménétrier syndrome, gastric dysplasia, adenomatous polyps and hereditary genetic predisposing factors as well. Hypochlorhydria occuring in case of gastric atrophy, promotes the bacterial colonization of the stomach. Although without a *HPV* infection no gastric cancer can develop, several different microorganisms with immune suppressing effect also play an important role in this carcinomatous process.
Gastric adenocarcinomas can be sorted into 2 types, such as the:
Intestinal type (type 1), which is characterized by well-formed glandular structures: This type involves usually the distal part of the stomach, occurs among patients with atrophic gastritis and is in a strong association with environmental factors, and the
diffuse type (type 2), which is characterized by poorly cohesive cells that tend to infiltrate the gastric wall: This type of tumors may involve any part of the stomach, especially the cardia, and are more malignant, than the intestinal type.
The early gastric cancers are confined to the mucosa or submucosa of the stomach, while the advanced ones invade the muscularis propria of the stomach. They spread and give often metastases to the regional lymph nodes or to other local or distant structures. Most patients being asymptomatic in the early stages often develop advanced tumors.
Symptoms often present are early satiety, nausea, vomiting, epigastric pain, dysphagia, anorexia, loss of weight, bloating, hematemesis, melena, iron deficiency caused anemia, and patients show positive results of fecal occult blood tests.
Diagnosis: symptomatically. In case of chronic gastric ulcers by endoscopy and biopsy in order to rule out malignancies with histological analysis. In case of early gastric cancers by a double-contrast barium upper gastrointestinal x-ray examination. Advanced gastric carcinomas can occasionally be seen even on plain abdominal radiographs. By CT, MRI, combined PET-CT scanning, etc. Mucin-producing carcinomas may show areas of punctate calcifications.
Differential diagnosis: by distinguishing it from chronic peptic ulcers and gastritis.
Treatment: by surgery, if the tumor is confined to the stomach. Chemotherapy and radiation therapy may relieve symptoms. (Chemotherapy and radiation therapies can be effective in case of gastric lymphomas). Symptomatically.
RFR method: detects and may eliminate the pathogen microorganisms.
The most typical resonant frequencies of gastric carcinoma types are: 314-319, 343-348, 350, 355-362, 370-373, 377-379, 392-393, 426-438, 442-452, 552-555 kHz

26.15. Carcinoid Syndrome

Carcinoid is a cancer usually occuring in the gastrointestinal tract, and can produce excessive amounts of several neuropeptides and biogene amines having hormonlike effects. These biologically active agents may be bradykinin, serotonin, histamine,

prostaglandins and ACTH which cause the symptoms of the carcinoid syndrome. Histologically, the tumors resemble adenocarcinomas but with no an aggressive behavior.
Gastrointestinal carcinoid is the most common primary tumor of the small bowel and appendix. These tumors are yellow submucosal nodules arising from enterochromaffin cells (Kulchitsky cells), which are neural crest cells present at the base of the crypts of Lieberkühn. Neural crest cells take part in the amine precursor uptake and decarboxylation system and are programmed for endocrine functioning. The mesenteric invasion of this cancer provokes an intense fibrotic reaction and if the tumors become large, they can seem to be like intraluminal polypoid masses, getting occasionally ulcerated.
These tumors elaborate serotonin and other histamine-like substances normally transported to and metabolized in the liver. Most tumors are clinically silent, though they may cause pain or intestinal obstruction, loss of weight, a palpable mass, or, rarely, a bowel perforation. Carcinoid syndrome occurs when the humoral load exceeds the capacity of monoamine oxidase (MAO) of the liver and the lung to metabolize serotonin. Most patients with carcinoid syndrome have liver metastases from a bowel carcinoid, although in rare cases, the humoral load from a primary tumor may overwhelm the metabolizing capacity mentioned above. Carcinoid syndrome may develop but in very rare cases of patients with noncarcinoid malignant tumors and dermatomyositis.
A carcinoid tumor spread into the liver may cause episodes of flushing, a bluish skin, abdominal cramps, diarrhea, heart damages and other symptoms constituting the carcinoid syndrome. Hepatic carcinoid is frequently involved as a metastatic disease originating from a gastrointestinal carcinoid.
Carcinoids arising in the stomach are usually associated with hypochlorhydria or achlorhydria. This condition but rarely becomes malignant, does never cause metastases; though, sometimes, it may produce histamine. Gastric carcinoids are rare tumors, multiple gastric carcinoid tumors are associated with enterochromaffin-like cell-hyperplasia, chronic atrophic gastritis and pernicious anemia. Solitary gastric carcinoid tumors and/or carcinoid tumors associated with multiple endocrine neoplasia type 1 (MEN-1) and Zollinger-Ellison Syndrome, have a higher potential for causing metastases. Most osseous metastases derive from carcinoids of the stomach.
Carcinoid tumors arising in the lung generally produce serotonin, gastrin, ACTH and histamine.
Carcinoids developing primarily outside the appendix are often malignant, while tumors developing in the appendix are usually benign if smaller than 2 cm in diameter. Appendiceal carcinoids are found mostly in younger patients, usually aged 20-40 years, while other carcinoids affect patients older than 50 years.
Rectal carcinoid tumors often produce polypeptides, polypeptide Y, neuropeptide Y, and other peptides, causing no symptoms. More over, these patients have no hormone-related symptoms even if they have liver metastases, too. Being asymptomatic, they are but incidentally discovered by proctoscopy and/or sigmoidoscopy.
In case of carcinoid syndrome, deposition of fibrous tissue may be found in the **cardiac** valves and the endocardium, resulting in tricuspid and pulmonary valve stenosis. **Esophageal and pancreatic carcinoids** are rare, with sad prognosis.
The most significant cause of carcinoid syndrome is a combined viral and bacterial infection. In all cases of carcinoid syndromes an infection with *one or more HPVsubtypes* can be experienced, always together with one or more immunosuppressive infections caused f.i. by *EBV, CMV, HTLV*, as well as a *mycoplasmal* infection caused typically by *Mycoplasma fermentans,* and with bacteria, producing colonia stimulating factors, f.i. *Escherichia coli* or/and other bacteria with similar stimulating effects. About 4% of malignant carcinoid syndrome cases are inherited and associated with the deficiency of a protecting protein.

The most common and often the earliest symptom of carcinoid syndrome is an uncomfortable flushing, typically of the head and neck (caused by excess histamine and bradykinin, which dilate the blood vessels). The skin can change color dramatically, from pale to red to a blue hue similar to cyanosis. Periorbital edema, excessive lacrimation and salivation, hypotension or hypertension, tachycardia, anxiety, and tremulousness, nausea, vomiting, explosive diarrhea and bronchoconstriction may also be experienced. Patients with carcinoid tumors may suffer also from symptoms similar to those of other intestinal cancers, such as cramping pain and changes in bowel movements caused by obstruction.
Diagnosis: by tests measuring the over production of 5-hydroxyindoles as well as of the serotonin in the blood, the increased excretion of 5- hydroxyindoleacetic acid in the urine. By x-ray, CT, MRI, (by somatostatin receptor scintigraphy). By biopsy and histological analysis (All carcinoids react positively with antichromogranin A antibodies)
Treatment: by surgery, by interferon therapy (Alpha, beta and gamma interferons given topically, systemically and intralesionally). Symptomatically (by administering histamine blockers, serotonin antagonists, corticosteroids. By administering f.i. octreotide, doxorubicin, streptozotocin, fluorouracil, etc.
RFR method: detects and may eliminate all pathogen viruses and bacteria.
The most frequent resonances found are: 307, 319, 332, 340, 353-362, 365, 371-383, 426-438, 442-454, 471-488, 493-495, 513, 518-529, 534, 544 kHz
RFR method accompanied by alpha, beta and gamma interferon therapy proves to be a good combination of the treatment. Patients need a very long monitoring time. Only the RFR method is able to eliminate all its causative pathogens as yet! The combined treatment with RFR method concerning carcinoid syndromes can assure a prolonged life for many patients.

26.16. Gastrinoma

Gastrinoma is a pancreatic tumor producing hormone gastrin, which stimulates the stomach to secrete acids and enzymes, causing thus pectic ulcers.
Zollinger-Ellison Syndrome (ZES) is a clinical syndrome causing severe peptic ulcer disease and diarrhea, due to enormous gastric acid hypersecretion, secondary to a neuroendocrine tumor, that secretes excessive amounts of hormone gastrin (gastrinoma). Gastrinomas occur in familial and sporadic form. Patients with gastrinoma in the familial form, belong to Multiple Endocrine Neoplasia type 1 (MEN-1). The malignant potential of gastrinoma is depending on its size, if greater than 2 cm, it must be removed.
Most patients with this condition have several tumors clustered in or near the pancreas.
The cause of ZES is a genetic predisposition and a combined infection with *HPV*.
Symptoms: are mild to severe abdominal pain and diarrhea. Perforation, bleeding and obstruction of the intestines can occur and are life threating.
Diagnosis: by CT, ultrasound, arteriography, MRI, PETscan, the locating of tumors may be difficult as being small and many of them.
Treatment: by total gastrectomy, (chemotherapy and radiotherapy have but a small effect on the tumor).
RFR method: detects and may eliminate the pathogen microorganisms.
The most frequent resonances are: 355-362, 426-438, 442-451, 471-480 kHz

26.17. Insulinoma

Insulinoma is a rare type of pancreatic tumors, secreting insulin, a hormone lowering the levels of glucose in the blood. Insulinoma may get cancerous. The most important symptom of insulinoma is the decrease of the blood glucose levels. The caused symptoms may mimic a variety of psychiatric and neurological disorders, causing headache, confusion, anxiety and panic, faintness, vision abnormites, muscle weakness,

unsteadyness, nervousness, marked changes in personality, convulsions and coma. Very low glucose levels and high insulin levels indicate the presence of insulinoma.
The cause of this disease is based on genetical predisposition and a *viral infection* caused by one of the *HPV* group.
Diagnosis: by biochemical laboratory examinations and symptomatically.
Treatment: by surgical removal.
RFR method: detects and may eliminate the HPV virus and Mycoplasma fermentans.
The most frequent resonances are: 314-319, 343-347, 372-383, 426-438, 442-451 kHz

26.18. Polyps and Cancers of the Colon

26.18.1. Polyps of the Colon

Polyp is a benign structure arising from the mucosal surface and projecting into the lumen of the bowel.
The most common adenomatous polyps may be solitary or multiple, sessile or pedunculated. The clinical significance of adenomatous polyps is their possible premalignancy. The contention that polyps are premalignant is based on the observations that foci of atypia or carcinoma in situ are occasionally found in polips and patients with multiple congenital polyposis of the colon have a significantly increased incidence of colon cancer, although areas of malignancy are but rarely found in adenomatous polips, a true progression from adenomatous polyp to carcinoma has rarely been demonstrated and carcinoma of the colon would be much more frequent if even a small percentage of adenomas underwent malignant changes. Treatment of polyps affecting adults is influenced by the debate concerning their possible transformation into cancer. Polyps in the sigmoid colon or rectum should be removed via the sigmoidoscope whenever possible.
Villous adenomas are polypoid lesions of the colon with a definite malignant potential.
Multiple congenital polyposis of the colon is transmitted as an autosomal dominant disease and is thus present in many members of an affected family. These family members transmit an inherited special *polyp virus*.
Gardener's syndrome refers to the coexistence of multiple colonic adenomatous polyps and various bening tumors, such as lipomas, fibromas, sebaceous cysts elsewhere in the body and osteomas, particularly in the jaw and skull bones. In case of this syndrome the colonic polyps develop at a later age than in that of multiple congenital polyposis, but the potency of its getting malignant is the same, so that colectomy is likewise recommended.
An entity easily confused with multiple colonic polyposis is the **juvenile polyposis** in which case inflammation of *polyps of viral* origin occur in the colon or in the large and small bowels. Chronic candida overgrowth in the intestines may influence the development of polyps.
The designation polypoid carcinoma refers to malignant lesions whose gross appearance resembles a polyp.
The most frequent resonances are: 296-312, 392-393, 408-411, 426-437, 440-452, 453-454, 459-464, 476-479, 550-556 kHz

26.18.2. Cancers of the Colon

Cancers of the large bowel accounts for about 25 percent of all cases of death caused by malignant diseases. The cause of colon cancers are several viruses, which are present in the polyps, too. While it is just possible, that adenomatous polyps become malignant, villous adenomas are definitely associated with cancer. A colon bearing a polyp may develop a cancer elsewhere. Congenital multiple polyposis of the colon has an astonishingly high malignant potential, ulcerative colitis also seems to potentiate and stimulate the

development of carcinoma in the diseased bowel and it is merely a question of time; when and in which part the carcinous process will develop.
Adenocarcinoma is the most common type of colon cancers. The degree of its differentiation varies widely and is not always correlated with the degree of its invasiveness or rapidity of its growth. All these tumors tend to invade the regional lymph nodes and to spread through the lymphatics and the portal vein to the liver. Their spreading directly into the paravertebral venous plexus does occasionally also occur.
Symptoms of colon carcinoma are usually vague and nonspecific at its beginning. Loss of weight and malaise are common. Cancers of the cecum and the ascending colon are usually flat or polypoid and clinically often silent as they do not obstruct the lumen of the bowel nor cause any visible melena. A sigmoid carcinoma may cause pain, just like an acute bowel obstruction or an acute perforation with peritonitis. Anemia, anorexia and malaise may also come about.
Diagnosis: by digital rectal examinations, by measuring levels of alkaline phosphatase, bromsulphalein retention and carcinoembryonic antigen in the blood, by colonoscopy, etc.
Differential diagnosis: by distinguishing it from colitis, diverticular diseases, tuberculosis, endometriosis, lymphogranuloma venereum, carcinoid tumors, metastatic cancers, etc.
Treatment: by surgery, by special chemotherapy.
The most frequent resonant frequencies found in case of polyps are: 296-312, 318-319, 323, 332-340, 344-356, 367, 372, 409, 454, 460, 468, 513, 534, 544, 554-555 kHz
The most frequent resonant frequencies found in case of adenocarcinoma are: 312, 314-318, 332-348, 356, 367-368, 392-393, 402-414, 426-438, 442-454, 460-464, 524-525, 534-545, 555-557 kHz

26.19. Primary Liver Cancers

Liver tumors may be benign at their beginning or be malignant at once. These cancerous tumors may originate in the liver, or may be metastasized to the liver from other parts of the body. Primary liver cancers originate in the liver; a cancer originating elsewhere in the body is named metastatic cancer.
Hepatocellular carcinoma (HCC, named also Hepatoma) is a cancer originating in the liver cells. Hepatomas are the most common primary liver cancers. Chronic infection with Hepatitis B and/or Hepatitis C virus increases significantly the risk of hepatomas. The association of Hepatitis D with carcinogenesis is possible, that of Hepatitis E is questionable. Alcoholic cirrhosis and fungal aflatoxin substances are risk factors of hepatomas. Other types of cirrhosis (f.i. PBC) are also risk factors of HCC. **Fibrolamellar carcinoma**) affecting relatively young adults is a rare type of liver tumors.
The first symptoms of a hepatoma are usually abdominal pain, loss of weight, and a large mass felt in the upper, right part of the abdomen. A person who has cirrhosis for a long time may unexpectedly become seriously ill. Occasionally, the first symptoms are acute abdominal pain and shock, caused by the rupture or bleeding of the tumor.
Cholangiocarcinoma is a cancer that originates in the lining of the bile channels in the liver or the bile ducts. In the orient, infestation with liver flukes may be partly responsible for this cancer. People suffering from long lasting ulcerative colitis and sclerosing cholangitis occasionally develop cholangiocarcinoma.
Hepatoblastoma is one of the more common cancers affecting infants. Occasionally, it occurs also among older children and may produce gonadotropin hormones resulting in early puberty.
Angiosarcoma is a rare cancer originating in the blood vessels of the liver and is caused by *virus*, and chemical carcinogen drugs such as vinyl chloride or other similar solvents.
Symptoms of primary liver cancers are abdominal pain, loss of weight, jaundice, icteric urinary, itching, fatigue, loss of appetite, vomiting, ascites, fever and nausea.

Diagnosis: by CT, MRI, ultrasound, x-ray, by antibody detection of the Hepatitis B, C, and D viruses, by physical examinations, etc.
Treatment: a small tumor might be surgically removed. Symptomatically
RFR method: detects and may eliminate the pathogen viruses!
The resonant frequencies of Hepatitis B virus are: 293, 340, 384, 392-398, 414-420, 444-448, 454, 488 kHz
The resonant frequencies of Hepatitis C virus are: 324-339, 350-352, 370-374, 396, 400-402, 450-456, 475-482, 540-541, 559-563 kHz
The resonant frequencies of Hepatitis D virus are: 348, 375, 386, 410, 432, 450, 468, 471, 490, 532, 535-548, 550-563, 580 kHz
The resonant frequencies of Liver flukes are: 280, 292, 346, 390, 420-430, 484 kHz
The resonant frequencies found in case of Primary liver cancers are: 343-347, 375-377, 390, 400-403, 408-409, 420-438, 442-451, 490-493, 513, 530-535, 548, 550-558 kHz
In case of primary liver cancers a lot of viruses can be found, such as Hepatitis B, C and D viruses, EBV, CMV, HPVs, and other unidentified wart viruses, but I don't known which one is important for the carcinogenesis.
Liver fluke can be found only in special cases of cholangiocarcinoma.

26.20. Adenocarcinoma of the Pancreas

Adenocarcinoma of the pancreas is a cancerous tumor originating in the cells lining the pancreatic duct.
Symptoms of this carcinoma are loss of weight, pain and unremitting jaundice. Digestive disorders, including anorexia, nausea, constipation are also prevalent. The course and intensity of jaundice depend on the degree of the caused biliary obstruction. Itching, an associated symptoms of jaundice may antedate the clinical icterus.
Pancreatic cancer can lead to death caused by inanition, biliary obstruction, local extension, or distant metastases. By its direct extension, the cancer may invade the liver, spleen, stomach, duodenum, colon, the portal venous system and the peritoneum. Metastases can develop in the regional lymph nodes, the liver, lungs, the mediastinal and cervical lymph nodes and the bones.
The cause of this tumor is a *virus*. The prognosis is very poor, radiotherapy and chemotherapy are usually ineffective. The only hope of the cure is surgery, to be performed on patients whose cancer has not spread, though the *virus* could develop a tumor in an other locus of the pancreas.
Diagnosis: to diagnose it at an early stage is very difficult. If an adenocarcinoma of the pancreas is suspected, the most commonly used diagnostic tests are ultrasound scanning, CT and endoscopic retrograde pancreatography. In order to confirm the diagnosis by biopsy for microscopic examinations.
Differential diagnosis: by distinguishing it from liver cancer, and metastases from an other cancer.
Treatment: by surgery. Symptomatically
RFR method: detects and may eliminate the pathogen virus or viruses.
The most frequent resonances found in case of adenocarcinoma of the pancreas are: 313-318, 368, 427-438, 442-451, 533, 544, 553-558, 568 kHz
The most frequent resonances found in case of ductal carcinoma of the pancreas are: 313-319, 368, 424-426, 442-451 kHz

26.21. Tumors of the Kidney

Benign renal tumors present but rarely any clinical problem, as they are usually asymptomatic. Benign renal tumors and other associated disorders include papilloma of the

kidney, adenoma, fibrolipomyoma, neuroblastoma, hydronephrosis, kidney cysts and multicystic kidney dysplasia.
The most frequent malignant tumor of children is the **nephroblastoma or Wilms's tumor,** probably arising from embryonic nephrogenic tissues and may contain epithelial and connective tissue elements.
Hypernephroma or carcinoma of the kidney (Grawitz's tumor) is the most common neoplasma of the kidney affecting adults.
Symptoms: Obscure fever together with moderate leucocytosis, is a fairly common symptom. One or more of the classic triad of hematuria, flank pain, and abdominal mass is present in about half of the cases, though the first symptoms are often caused by metastases to the lungs, bone, liver, or the brain. Calcification within the tumor mass can occasionally be seen on the plain x-ray of the abdomen. In rare instances polycythemia can be observed caused by and an erythropoietic substance produced by the tumor. In rare instances a pressor substance produced by the tumor may cause hypertension. Renin-secreting tumors of the juxtaglomerular cells can cause severe hypertension, which may be relieved by nephrectomy. An extension of carcinoma into the renal veins may cause acute varicoamyloidosis complicating the carcinoma of the kidney, and involving the liver, spleen and myocardium, too. A paraneoplastic syndrome, such as polyneuritis and myopathy found in association also with other forms of malignant diseases can occasionally be experienced in case of carcinoma of the kidneys and the bladder.
This tumor is relatively radiotherapy-resistant
Diagnosis: by intravenous urography, by renal angiography, ultrasound scanning, x-ray, CT, MRI and PETscan,
Differencial diagnosis: by distinguishing it from other tumors and metastases.
Treatment: by chemotherapy (Vinblastin), radiotherapy and surgery (nephrectomy).
RFR method: detects and may eliminate the pathogen viruses and bacteria!
The resonant frequencies of the kidney papilloma virus are: 303-307, 324-327, 370-371, 389, 392-399, 427-434, 436-439, 450-454, 469, 540-546, 564-567 kHz
The most frequent resonant frequencies found in case of renal cell carcinoma are: 324, 343-345, 368, 389-392, 402-409, 426-438, 440-452, 475, 493, 513, 540, 552-558, 568 kHz

26.22. Prostate Cancer

Prostate cancer is one of the most common cancers and the leading cause of cancer related death among males.
Prostate cancer is a disease, in which a cancer is developing in the prostate, a gland in the male reproductive system. It occurs if the cells of the prostate mutate and begin to multiply out of control. These cells often spread (metastasize) from the prostate to other parts of the body, especially to the bones and the lymph nodes.
Prostate cancer is an adenocarcinoma, a glandular cancer, beginning when normal semen-secreting prostate gland cells transforme into cancer cells. The most common location of this transformation to adenocarcinoma is the peripheral zone of the prostate gland. Initially, small clumps of cancer cells remain confined to otherwise normal prostate glands, which condition is known as carcinoma in situ or prostatic intraepithelial neoplasia (PIN). Though there is no proof that a PIN is a cancer precursor, it is closely associated with cancer. Over time, these cancer cells begin to multiply and spread to the surrounding prostate tissue (the stroma) to form a tumor. Eventually, the tumor can get enlarged, invade the nearby organs f.i. the seminal vesicles, or the rectum, and sometimes the tumor cells develop the ability to metastasize via the bloodstream and the lymphatic system.
Prostatic intraepithelial neoplasia is the putatively precancerous end of the morphologic continuum of the carcinomatous cellular proliferations within the prostatic ducts, ductules and acini. PIN can be low grade and high grade. The high-grade one is considered a

precursor to invasive carcinoma and may even coexist with a cancer of the same gland. Th transforming process of high-grade PIN into an early invasive cancer is characterized by the disruption of the basal cell layer and the basement membrane, by the progressive loss of secretory markers of the differentiation, by increasing nuclear and nucleolar abnormalities f.i. more and more variations in the content of DNA, and a fast increasing proliferative potential. This cancerous process affects men frequently, cancer can be found in about 60 percent of men (more than 70 years old), suffering from prostate hyperplasia.
The *Human Papilloma Virus* (its resonance frequencies being 315-319, and/or 404-405 kHz) causes the **ductile carcinoma of the prostate**, while an other *HPV* species (its resonance frequencies being 412-410 or 427-438 kHz) causes the **acinar prostate adenocarcinoma** together with a present infection caused by *Mycoplasma genitalium* (its resonance frequencies being 307-308, 342-350 kHz). Additional frequent viral coinfections used to be also present, caused by *Cytomegalovirus, Epstein-Barr Virus,* other *Herpes viruses* and bacterial coinfections caused by various species of the *Proteus group*, or seldom by *Ureaplasma* (its resonance frequencies being 384-388 kHz) as well as by *Pseudomonas.*
Symptoms: At the beginning of a prostate cancer, patients may suffer from symptoms similar to those of a benign prostatic hypertrophy, i.e. difficulty in urinating (starting and maintaining a steady stream of urine), frequent urination even at night, pain and erectile dysfunctions. In advanced cases blood in the urine, painful urination and ejaculation, as well as sudden urinary retention can also come to pass. Metastatic cancers can cause pain in the bones (mostly in the vertebrae, the pelvis and the ribs). Prostate cancer can also spread to the brain, causing seizures, confusion, and rarely other mental or neurological symptoms as well.
Diagnosis: by physical examinations (annually performing a digital rectal examination of the prostate) and by screening blood tests, i.e. (prostate specific antigen) PSA test, symptomatically, by biopsy and histological examinations, by ultrasound scanning, By x-ray and bone scans, in order to detect metastases.
Treatment: by surgery, radiation therapy, hormonal therapy, seldom chemotherapy, and their combinations and symptomatically.
RFR method: detects and may eliminate all the causative pathogen microorganisms!
Frequently found resonant frequencies in case of prostatitis, capable to develop a hyperplasia or tumor are: 318, 340, 348, 353, 372-373, 396-397, 408, 410, 418, 454, 470, 476, 513, 520-526, 534, 544, 555, 570-578 kHz
Frequently found resonant frequencies in case of Benign prostatic hyperplasia (BPH) are: 292, 318, 323, 340-343, 353, 372-381, 384-388, 392-397, 402-408, 418-426, 454, 470, 544, 555 kHz
Frequently found resonant frequencies in case of Prostate cancer are: 314-319, 343, 352-353, 372-383, 392, 402-410, 427-437, 434-444, 442-451, 452-453, 470, 506, 513, 524, 539-547 kHz
Making a comparison between these three groups one can experience a lot of identical frequency presenting just the same different pathogens. There are certain important differences in the case of cancer, affecting these frequencies: **402-410, 437, 539-547 kHz**, which are the resonance frequencies of the human papilloma and other wart viruses.
(**372-383 kHz** may be the resonance frequencies of EBV, which can be found in all three groups.)
555 kHz may be the resonance frequency of the Herpes virus group, which can be found in all three groups.
376-381 kHz may be the resonance frequency of Staphylococcus, which can be found in all three groups.
408-416 kHz may be the resonance frequencies of Proteus, which can be found in all three groups.)

The other frequencies have not been determined as yet. Sexually transmitted diseases are infections often present in case of chronic prostatitis and prostate cancers lasting for years after having been acutely infected f.i. by *Hemophilus ducreyi, Chlamydia trachomatis, Calymmatobacterium granulomatis, Shigella, Campylobacter, Salmonella, Trichomonas vaginalis, a few species of amebas, Ureaplasma urealyticum, Candida albicans. Genital herpes group such as HSV2, HSV1, CMV, EBV, and genital warts viruses such as Condyloma acuminatum virus, and HPVsubtypes 16-18.* The latter types as well as *other papilloma viruses* can cause prostate neoplasms and cervical intraepithelial neoplasms or other cancers of the genitourinal tract.
According to my opinion, one of these pathogens produces the prostate specific antigen (PSA).

26.23. Bladder Cancer

There are different types of cells forming the bladder, among which the cells lining the inside of the bladder-wall are most likely developing cancer. Anyone of these various cell types can become cancerous. The cancers are named after the cell types they originate from.
The most common type of bladder cancers is the **urothelial carcinoma or Transitional Cell Carcinoma** (TCC). The urothelium is present in the whole urinary tract, including the renal pelvis, ureter, bladder and urethra. In case of TCC, the normal lining cells undergo changes that lead to uncontrolled cell growth characteristic of cancer.
Squamous Cell Carcinoma: This type of cancers originates in thin, flat cells and is typically the result of a chronic bladder inflammation and irritation lasting for months or years.
Adenocarcinoma: This type of cancers originates from cells that make up glands. Glands are specialized structures producing and releasing fluids such as mucus.
The clinical course of bladder cancer owns a broad variety of aggressiveness and risk. Low-grade, superficial bladder cancers have a minimal risk of leading to death; while the high-grade muscle-invasive cancers are often lethal.
Bladder cancers are classified as being low grade (grade 1 and 2) and high grade (grade 3) tumors. These tumors can also be classified by their pattern of growth: papillary (70%), sessile or mixed (20%) and nodular (10%). Carcinoma in situ (CIS) is a flat, noninvasive, high-grade urothelial carcinoma. The most significant prognostic factors for bladder cancer are its grade, depth of invasion, and the presence of CIS.
Spreading: Bladder cancers are most likely to spread to their neighboring organs and lymph nodes prior to spreading through the blood stream to the lungs, liver, bones, or other organs. Bladder cancers can be sorted into superficial and invasive diseases. A superficial bladder cancer is limited to the innermost linings of the bladder mucosa and the lamina propria. An invasive bladder cancer penetrates at least the muscular layer of the bladder wall. Nearly all adenocarcinomas and squamous cell carcinomas are invasive. Urothelial cell carcinomas are usually not invasive, they therefore go no deeper than the superficial layer (mucosa) of the bladder.
Benign, noncancerous papillary tumors or papillomas grow as projections into the hollow part of the bladder. They can be easily removed, but sometimes they grow again.
Genetical predisposition: several genetic mutations are identified concerning bladder cancers. Mutations of the tumor suppressor gene for p53, found on chromosome 17 are associated with high-grade bladder cancer and CIS. Mutations of the tumor suppressor gene for *p15* and *p16*, found on chromosome 9 are associated with low-grade and superficial tumors. Retinoblastoma tumor suppressor gene mutations are also noted. Bladder cancers are associated with an increased expression of the epidermal growth factor gene and the *erb*-b2 oncogene, and with mutations of the oncogenes p21 *ras*, c-*myc* and c-*jun*.

Infections often present in case of bladder carcinoma are: mycoplasmal infections caused by *M. fermentans, M. penetrans, M. genitalium* (and rarely by *M. pneumoniae)*; infections caused by *HPVs, Human Lymphotrop Viruses, CMV, EBV; Herpes simplex 1 and 2 viruses, Coxsackie viruses, Adenoviruses, Proteus group bacteria, Candida species, E. coli, Gardnerella, Shigella, Nanobacteria, Ureaplasma* and in tropical countries by *Schistosoma parasites.*

Diagnosis: by ultrasound, CT, intravenous pyelogram, cystoscopy, biopsy and histological analysis.

Treatment: with intravesical immunotherapy, intravesical chemotherapy (f.i. with Methotrexate, Vinblastine, Adriamycin and Cisplatin in combination), prior to a radical cystectomy or external beam radiotherapy, radical cystoprostatectomy (in case of men) and anterior pelvic exenteration (in case of women).

RFR method: detects and may eliminate all the pathogen microorganisms.

The resonant frequencies of a HPV infection present in Urothelial carcinoma are: 343-347, 402-410, 418-426, 459-464, 517-521, 525-527 kHz

The resonant frequencies of a HPV infection present in Squamous cell carcinoma are: 538, 541-545 kHz ,

The resonant frequencies of a HPV infection present in Adenocarcinoma are: 426-438 kHz

The most frequent resonances of Mycoplasmal infections are: 307-308, 321-324, 342-350, 440, 442-451, 493-495 kHz

The most frequent resonances of HTLV are: 297-299, 307, 311-315, 320-340, 354, 359, 365-367, 370-376, 382-383, 397-400, 406, 416, 428-439, 453-455, 474-476, 480-482, 484, 487-490, 493-504, 523-530, 540-545, 570-578 kHz

The most frequent resonances of Proteus vulgaris are: 327-329, 333-339, 408-416, 426, 522-529, 535 kHz

As to the frequencies of the other pathogen microorganisms see in their special Chapters.

26.24. Uterine Fibroids (Myoma uteri)

Uterine fibroids are mesenchymal benign tumors growing from the muscle layers of the uterus. Uterine fibroids have two types, i.e. leiomyoma, which is the common form of uterine fibroids though it can grow in the skin and the gut as well, and rhabdomyoma, which is in childhood a rare tumor of the muscles, becoming often malignant.

Leiomyoma arises as an overgrowth of the smooth muscle and the connective tissue of the uterus mostly in case of a genetic predisposition. A monoclonal proliferation of the smooth muscle cells can histologically be experienced. The hormonal dependency of this tumor is characterized by its estrogen and progestin receptors. Uterine leiomyoma may but rarely undergo malignant degeneration and become a sarcoma. The true incidence of this malignant transformation is difficult to determine, as leiomyomas are common, while malignant leiomyosarcomas are rarely occuring and can arise de novo as well.

Leiomyoma can develop in case of genetic predisposition caused by an existing special infection. The cause of its tumorous process is a determining infection caused by *certain tumor viruses* combined with infections caused by some *immunesuppressant pathogens* (440-452 kHz and other) and secondary co-infections with various other bacteria as well.

Symptoms: Most women with uterine fibroids are asymptomatic. Symptoms may include abdominal cramping, pain, usually felt in the days of menstruation, bleeding (menorrhagia), rarely even a massive intraperitoneal bleeding, causing severe anemia, frequent, urgent urination, and incontinency resulting from the pressure of the fibroid on the bladder. Constipation, difficult defecation and rectal pain owing to the pressure on the colon come but rarely about. Generalized pelvic and/or lower abdominal discomfort, and fibroid calcification (caused by *nanobacteria*) can sometimes also be experienced.

Complications during pregnancy can include spontaneous abortion, intrauterine growth retardation, premature and preterm labor, uterine dyskinesia, obstruction of the birth canal, postpartum hemorrhages and hydronephrosis.
Diagnosis: by physical examination, palpation, symptomatically, by ultrasound, CT and MRI.
Treatment: depending on the symptoms of fibroids, conservatively, by selective myomectomy, uterine artery or fibroid embolization, hysterectomy etc.
RFR method: detects and may eliminate all the causative pathogen microorganisms.
The most frequent resonances found in case of leiomyoma are: 425-428, 462, 516 kHz
The most frequent resonances found in case of rhabdomyoma are: 340, 353-355, 396, 402-410, 425-432, 442-451, 461-463, 476, 514-519, 544-545 kHz
The most frequent resonances found in case of leiomyosarcoma are: 445-448 kHz
The most frequent resonances found in case of rhabdomyosarcoma are: 385, 401, 408, 442-451, 512-517, 524-527, 535-537, 544-549, 559, 567 kHz
The most frequent resonances found in case of embryonic rhabdomyosarcoma are: 331, 350, 420-423, 513-520, 524, 569 kHz

26.25. Uterine Cancers

Endometrial carcinoma begins in the uterine endometrium. It is the most frequently occurring female genital cancer. There are nowadays no screening tests for cancer of the uterine recommendable for asymptomatic women. No evidence suggests that routine endometrial samplings or transvaginal examinations by sonography done in order to evaluate the endometrial condition of asymptomatic women (i.e. screening) has a role in early detection of uterine cancers, nor even among women who are administered tamoxifen having had breast cancer. Endometrial adenocarcinoma occurs usually in the reproductive and menopausal years. Most patients with this malignancy are about 50-60 years old. About 5% of women younger than 40 years have uterine adenocarcinoma and only about 20% of women are diagnosed as cancerous before their menopause. Most endometrial carcinomas are **endometrioid adenocarcinomas**.
Adenoacanthomas (with benign squamous components) and **adenosquamous carcinoma** (with malignant squamous components), **clear cell and papillary serous adenocarcinomas** represent about 10% of all endometrial cancers. Data suggest that irrespective of whether a squamous component is present (either benign or malignant), or not, the prognosis is related to the grade of the adeno component, though if a malignant squamous component is present, there probably will be a more poorly differentiated adeno component experienced. These endometrial cancers may originate within an endometrial polyp or in a diffuse multifocal way. Early tumor growth is characterized by an exophytic and spreading mode as well.
Symptoms: are characterized by spontaneous abnormal bleeding from the uterus, even at the early stages occuring due to the friability of this carcinoma. Later on myometrial invasion and growth toward the cervix will come about. The spreading of this carcinoma occurs mostly by a direct/local spread as well as by a lymphatic spread to the pelvic, the para-aortic, and, rarely, to the inguinal lymph nodes. Its hematologic spreading causes metastases in the lungs, liver, bone, and sometimes in the brain. Transtubal intraperitoneal metastatic implants can but rarely develop.
The diagnosis **of uterine sarcomas**, especially that of **Leiomyosarcomas** versus leiomyoma is based on histological findings characterized by cellular atypia, high mitotic activity and uncertain malignant metastatic potential of the sarcoma.
Carcinosarcomas or homologous mixed müllerian tumors (MMT) are typically a higher grade undifferentiated spindle cell sarcoma combined with an endometrioid carcinoma. These MMTs are characterized by an early extrauterine spreading and lymph node

metastases. New data suggest the effect of substantial expressions of c-kit receptors of MMTs on the character of this tumor.
Family history of endometrial cancer appears to be at increased risk. Other identified risk factors concerning patients suffering from adenocarcinoma of the endometrium are obesity and a significantly enhanced estrogen level of the patient (either administered as replacement therapy or produced endogenously f.i. by a granulosa cell tumor or a polycystic ovarian disease). Some data indicate, that smoking and the administation of combination oral contraceptives (OCs) decrease the risk of developing endometrial cancer. Breast, colon, and ovarian cancers are frequently associated with endometrial cancer.
Causative agents of these malignant cancers are many different *HPVs* (their resonance frequencies being mostly 402-410 kHz), various *HTLV*, *HBLV* and *Mycoplasma genitalium and fermentans, Herpes genitalis, rarely sarcoma virus* (carcinosarcomas) as well.
Diagnosis: symptomatically, by vaginal ultrasonography, endometrial biopsy, hydroultrasonography, hysteroscopically directed biopsy, PAP test, CT, MRI, and PETscan.
Treatment: must be determined according to the stage of the tumor. By surgery, radiation or/and chemoterapy (f.i. cisplatin) in order to eradicate the carcinoma, to reduce morbidity, and to prevent complications.
Prevention: by vaccination. The most available vaccine contains solely one HPV group (the resonance frequency of which is 404.5 kHz), polyvalens vaccines contain HPVs (their resonance frequencies being 402-410 kHz).
RFR method: detects and may eliminate all pathogen microorganisms.
The most frequently found resonances are: 307-308, 314, 342-350, 352-363, 365-366, 370-375, 402-410, 425-435, 442-451, 453-455, 480-485, 487-490, 493-495, 517-521, 525-527, 536 kHz
Rarely found resonancies are: 316-319, 459-464, 470-476, 510-515, 542-545 kHz

26.26. Cancer of the Uterine Cervix

Cancer of the uterine cervix is the second most common malignant tumor affecting women, most common among younger women. It is characterized by a gradually progressing from precancerous lesions to recognizable stages before developing into an invasive disease. The cancerous process is almost certainly curable if it is identified before its invasive cancer stage. *Human Papilloma Virus* **(its most frequent resonant frequency being 404.5 kHz)** is recognized as the most important causative agent in cervical carcinogenesis. Cancer of the cervix typically originates from a dysplastic or premalignant lesion at the squamocolumnar junction of the cervix. The transformation from a mild dysplastic form into an invasive carcinoma **(the frequency resonances of which are 402-410, 427-438 kHz)** occurs generally slowly within several years.
Carcinoma in situ is known to precede the invasive cervical cancer in most cases. The progression to invasive carcinoma becomes established and is considered to be irreversible if the malignant process invades the basement membrane and the invasion of the cervical stroma already occured. The forms of this cancer can be: exophytic, nodular, infiltrative and ulcerative.
The most frequent **exophytic form** is mostly polypoid or papillary, erising from the exocervix. It may become an enlarged, friable, bulky mass involving the superficial part of the cervix with a tendency to bleed excessively.
The **nodular form** arises in the endocervix, invades the cervical stroma forming confluent, firm masses either involving the endocervical region or extending into the endocervical canal causing a „barrel-shaped cervix".
The **infiltrative form** leads to a stone-hard cervix, invades the vaginal fornices, the upper part of the vagina, the corpus and the lateral parametrium.

The ulcerative form of the tumor gets necrotic and usually complicated by secundary infections causing a seropurulent discharge.
An invasive cervical cancer often spread into the regional pelvic lymph nodes sometimes into the retroperitoneal, inguinal, or thoracic lymph nodes as well.
The lymphatic spreading of the tumor occurs first involving the regional paracervical and parametrial lymph nodes and then the internal and external iliac lymph nodes. The most common sites of hematogenous metastases are the lungs, bones, urinary tract, rectal regions and the liver.
Cervix cancers are histopathologically in most cases Squamous Cell Carcinomas, but if erising from the endocervical-type cells they will develop various forms of adenocarcinomas **(the resonant frequencies of which are 427-438 kHz)** constituting about 10% of all cervical malignancies. Some miscellaneous uncommon cancers of the cervix do also exist.
Symptoms include abnormal vaginal bleeding (f.i. postmenopausal bleeding, irregular menses, heavy menstrual flow, painless metrorrhagia, or postcoital bleeding). Abnormal vaginal discharge (watery, purulent, or mucoid) can also be associated with the illness. In advanced cases pelvic and abdominal pain, urinary or rectal symptoms can also be experienced.
Cervical cancer occur mostly among young women. This cancer is caused by a Human Papilloma Virus (resonance: 404.5 kHz) which may be transmitted during sexual intercourse. Other risk factors of getting cervical cancer can be to have several sexual partners, as well as infections caused by HSV2 and a recurrent and chronic candidal vulvovaginitis.
Prevention:
By vaccination. The most easily available vaccine contains only one HPV (404.5 kHz), the polyvalens vaccine contains HPVs (402-410 kHz).
By the screening of women older than 30 with Pap test and HPV-DNA test, every 3 years. Women with certain risk factors (f.i. infections with HIV, HTLV, HTLB, Genital herpes, Mycoplasma genitalium, Mycoplasma fermentans infection, prenatal diethylstilbestrol exposure, etc), should be screened annually, though if using Pap smear in the detection of cervical dysplasia there can likely be more false-negative results be found.
Diagnosis: by palpation, inspection, colposcopy, Pap smear, biopsy, endocervical curettage, hysteroscopy, cystoscopy, proctoscopy, IVU, and radiographic evaluation of the lungs and skeleton.
Treatment: depending on the stage of the cancer, surgically, by radiation therapy and/or chemotherapy.
RFR method: detects the pathogen microorganisms and eliminates them.
The resonance frequencies found depend on the stage of the cancer. In an earlier stage there are less pathogen frequencies present, at the end stage there are many different microorganisms and resonances to be found.
The most frequent resonances are: 307-308, 314, 342-350, 352-363, 365-366, 402-410, 427-438, 442-451, 453-455, 480-485, 487-490, 493-495, 517-521, 525-527 kHz

26.27. Basal Cell Carcinoma

Basal Cell Carcinoma (BCC) is the most common skin cancer. This tumor typically develops on sun-exposed skin, is slowly growing and but rarely metastasizes. If neglected, it can cause a significant local destruction and even disfigurement. BCC is a malignant neoplasm, which rarely metastasizes, but if it does, the most common loci of its metastases are the lymph nodes, the lungs and the bones., Basal cell tumors tend to be locally destructive. Periorbital tumors can invade the orbit, lead to blindness, if its diagnosis and

treatment are delayed. Perineural invasion may also occur, causing damages of the functioning of the nerves.
Its various forms include:
The nodular form, characterized by a flesh-colored papule with telangiectasis; which can ulcerate becoming a „rodent ulcer" (ulcus rodens), an ulcerating nodule with a pearly border.
The cystic form: occurs but seldom and is difficult to distinguish from the nodular form, It has a central cavity filled with fluid.
The pigmented form: is a variant of the nodular form that may be confused with melanoma.
The sclerosing/cicatrising form: is a scar-like, colourless lesion.
The superficial form: is a red scaling patch.
These BCCs are believed to arise from pluripotent cells (having the capacity to form hair), as well as from sebaceous glands and apocrine glands. They usually arise from the epidermis or the outer root sheath of a hair follicle. Immunosuppression can modestly increase the risk of BCC, so that recipients of organ or stem cell transplants have a higher risk of developing BCC. Persons diagnosed to have a nonmelanoma skin cancer are at increased risk of developing additional tumors in the future.
Xeroderma pigmentosum is an autosomal recessive disease caused by an inherited deficient ability to repair UV-induced damages of DNA. Pigmentary changes are early present and are followed by the development of BCCs, Squamous Cell Carcinomas, and sometimes by malignant melanoma as well. Other symptoms include corneal opacities, eventual blindness and neurological deficits.
Nevoid BCC Syndrome (Basal Cell Nevus Syndrome, Gorlin-Goltz syndrome) is an autosomal dominant disorder characterized by the early formation of multiple odontogenic keratocysts, palmoplantar pitting, intracranial calcification and rib anomalies. Various tumors such as medulloblastoma, meningioma, fetal rhabdomyoma, and ameloblastoma can also develop. The patched/hedgehog intracellular signaling pathway plays a role in the development of sporadic BCCs and the nevoid BCC syndrome (Gorlin-Goltz syndrome) as well. This pathway influences the differentiation of a variety of tissues during the person's fetal development. After embryogenesis, it continues to function in the regulation of cell growth and differentiation. The loss of the inhibition of this pathway is associated with human malignancy, including BCC.
Rombo syndrome is an autosomal dominant condition characterized by BCCs, atrophoderma vermiculatum, trichoepitheliomas, hypotrichosis milia and peripheral vasodilation with cyanosis.
Bazex syndrome: There exist two different entities named Bazex, one of them being a genetic detemined syndrome in case of which follicular atrophoderma named also „ice pick" marks, especially dorsally on the hands, multiple BCCs, hypotrichosis and local anhidrosis (decreased or absent sweating) are characteristic.
Histopathologically basal cell carcinomas are malignant epithelial tumors arising only in the skin, from the basal layer of the epidermis or the pilosebaceous adnexa. Tumor cells resemble normal basal cells (small, monomorphous) and are settled in palisade form at the periphery of the tumor nests, are spindle-shaped and irregular in the middle. The tumor clusters are separated by a reduced stroma with an inflammatory infiltrate.
BCC is caused by certain viral infections (caused probably by *different viruses*) and immunosuppressant factors and agents such as UV light (sunlight), *HTLV, Mycoplasma species, EBV* and *CMV* and carcinogenic drugs and substances.
Symptoms: a nonhealing sore of varying duration, visible typically on the face, ears, scalp, neck and the upper trunk. Mild trauma, such as face washing or drying with a towel, may cause bleeding. A history of chronic recreational or occupational sun exposure is characteristic. Intense sun light often exposed to in childhood and young adulthood in the

patients history. Nodular BCCs occur mostly on the head, neck, and upper back. Symptoms include crusting and telangiectases over its surface as well.
Diagnosis: symptomatically, by skin biopsy and histologic examinations
Treatment: by surgery, ionizing radiation, superficial x-ray, cryotherapy, drug therapy with 5-Fluorouracil, local therapy with chemotherapeutic and immune-modulating agents (Imiquimod 5% cream), etc.
RFR method: detects and may eliminate the pathogen agents (After the surgical course the basalioma virus can survive. RFR method can detect and eliminate it.)
The most frequent resonances found in case of BCC are: 291-292, 313-314, 389, 524, 541, 540-545, 557-558, 582-585 kHz
RFR method can prevent the development of metastases and a recidive lesion.
Often accompanied immunosuppressive infections are mostly caused by *HTLV, Mycoplasma species, EBV* and *CMV.* (See their special Chapters.)

26.28. Malignant Melanoma

Malignant melanoma is a cancer originating in the pigment producing melanocytes of the skin and can metastasize into many organs. This malignant tumor is caused by a *primitive retrovirus* combined with infections caused by *other viruses and bacteria.*
Melanoma can begin as a new, small, pigmented skin growth on normal skin, most often on sun exposed areas, but nearly half of the cases develop from existing pigmented moles. Melanoma readily spreads to distant parts of the body, where it continues its fast growing and the destroying of tissues. The less the melanoma is grown into the skin, the greater the chance of healing it. If a melanoma is grown deep into the skin, it can more likely spread via the lymph and blood vessels, causing death of the patient within a few months or years. Melanoma can develop metastases in the heart, lung, liver and the CNS. The course of the disease varies greatly and depends on the strength of the body's immune defenses. Some people survive in apparent good health for many years despite the spreading of the melanoma. If the existence of a melanoma is suspected, the surgent must remove the entire tumor in order to examine it microscopically.
If the melanoma is already metastasized, the cure rate is bad, however, anyone who had a melanoma is at risk of developing an other one. Sunburn is a very dangerous risk factor for predisposed persons.
Treatment: by surgery, chemotherapy, immunotherapy with IL-2, IFN, TNF, and other natural or synthetic immunostimulants and antibody therapy may be useful.
RFR method: detects and eliminates the pathogen virus and all other associated microorganisms!
The most often found resonant frequencies are: 294-300, 322-328, 342-356, 440-456, 465-473, 480-489, 490-495, 500-507, 533-543, 554-563, kHz

26.29. Moles

Gestational trophoblastic neoplasms (GTNs) represent a spectrum of premalignant and malignant disorders occuring after abnormal fertilizations. GTNs include complete hydatidiform moles, partial hydatidiform moles, invasive moles, choriocarcinoma and placental-site trophoblastic tumors. Atypical moles and dysplastic nevi are acquired melanocytic lesions of the skin the clinical and histologic definitions of which are controversial and as yet not exactly established. Quite a numerous definitions and criteria have been proposed, including the use of the term atypical moles for clinically abnormal nevi, dysplastic nevi and histologically abnormal nevi. Clinically abnormal nevi are evaluated histologically, some studies have found a lack of concordance, some clinically abnormal nevi having no dysplastic features and some nevi appearing to be normal having some dysplastic features. Atypical moles differ in several respects from the commonly

acquired melanocytic nevi, including diameter and lack of pigment uniformity, though some atypical moles can not be clinically distinguished from melanoma. The clinical and histologic appearances of atypical moles occurring in a familial setting appear to overlap with sporadically occurring atypical moles. In atypical moles the virus of the melanoma is usually present.

Genetical predisposition: 3 genes, i.e. CDK2NA and CDK4, mapped to 9p21 and 12q14, and CMM1 gene, mapped to 1p are, though not proved as yet, supposed to be in association with a subset of hereditary melanomas and with the familial atypical mole and melanoma (FAMM) syndrome. Somatic mutations in PTEN, BRAF, and MCR1 (melanocortin-1 receptor) may be associated with melanoma.

Ultraviolet light (UV-A and UV-B) is supposed to initiater and promote the transformation of melanocytes into atypical melanocytes or melanoma. UV light exposure may be required for the full expression of FAMM syndrome. Patients with FAMM syndrome are at an increased risk of developing melanoma, although the individual risk varies.

Diagnosis: by histological analysis.

Treatment: by surgery.

RFR method: detects and can eliminate the infective agents after surgery.

The most frequent resonances of moles are: 307, 319-320, 332, 335-340, 370-374, 401-403, 442-451, 474 kHz

The most frequent resonances of melanoma are: 370-374, 442-451, 501-507, 533-543, 556-562 kHz

26.30. Rhabdomyosarcoma

A rhabdomyosarcoma is a type of sarcoma, in which the sarcoma cells are thought to arise from skeletal muscle progenitors. It can be found attached to muscle tissue, wrapped around the intestines and anywhere in the body excepting the bones. Rhabdomyosarcoma is a malignant tumor of striated muscle origin, derived from primitive mesenchyme that retained its capacity for skeletal muscle differentiation. Rhabdomyosarcoma of the head and neck is primarily a disease of children under 10 years, and is the most frequent soft tissue sarcoma in childhood. Head and neck are the most frequent loci of its origin, less frequently affected loci are the genitourinary tract, extremities, trunk, retroperitoneum, intrathoracic region, GI tract, perianal and anal regions. The most frequent locations of the head are the parameningeal and orbital places. Several genetic syndromes and environmental factors are associated with an increased prevalence of RMS.

Genetic syndromes include Neurofibromatosis, Li-Fraumeni syndrome (a germline mutation of the tumor suppressor gene TP53), Rubinstein-Taybi syndrome, Basal Cell Nevus Syndrome, Beckwith-Wiedemann syndrome and Costello syndrome.

Rhabdomyosarcoma is divided into 5 major histologic categories: embryonal, alveolar, botryoid embryonal, spindle cell embryonal, and anaplastic.

Embryonal rhabdomyosarcoma is the most common subtype observed among children. The tumors are most commonly observed in the genitourinary region or the head and neck. By histological examination, they have a high cytological variability, which represents several stages of the skeletal muscle morphogenesis. Embryonal rhabdomyosarcoma cells show a loss of specific genome material from the short arm of chromosome 11. This consistent loss of the material from the 11p15 region may suggest the presence of a tumor suppressor gene.

Alveolar rhabdomyosarcoma

Alveolar rhabdomyosarcoma is most frequently observed among adolescents and involves mostly the extremities, the trunk and the perianal and/or the perirectal region. Individuals with the PAX7 translocation are younger and may have longer event-free survival than those with the PAX3 translocation. Unlike embryonal rhabdomyosarcoma, alveolar

rhabdomyosarcoma demonstrates gene amplification, its DNA content is typically tetraploidy.

Botryoid rhabdomyosarcoma

This subtype characteristically arises under the mucosal surfaces of body orifices; and can therefore be mostly observed in areas of the vagina, bladder and nares.

Spindle cell rhabdomyosarcoma

This subtype occurs predominantly in the paratesticular region but rarely in the head and neck.

Anaplastic rhabdomyosarcoma

Anaplastic rhabdomyosarcoma is the least frequent of all subtypes, affecting patients between 30-50 years.

Diagnosis: by surgical biopsy, chest CT, and technetium diphosphonate bone scanning. By lumbar puncture for cerebrospinal fluid cytology.

The **prognosis** for a child with rhabdomyosarcoma depends on the site of tumor origin, tumor size, nodal involvement, histology, and its cellular DNA content. Biologic factors may also influence the prognosis.

Treatment: by surgical resection followed by chemotherapy, ending with a standard course of radiation.

RFR method: detects and may eliminate all pathogen microorganisms.

Its most frequent resonances are: 331, 372, 385-389, 401, 408-411, 442-454, 512-517, 520-527, 533, 535-537, 544-546, 557-559, 567-569 kHz

26.31. Ewing's Sarcoma

Tumors of the Ewing's sarcoma (ES) family include Ewing's sarcoma, peripheral primitive neuroectodermal tumor, neuroepithelioma, atypical Ewing's sarcoma and Askin tumor (tumor of the chest wall). Tumors of this family are thought to derive from cells of the neural crest, possibly of the postganglionic cholinergic neurons.

According to current researches of tumors of the Ewing's sarcoma family a translocation joins the ES gene EWS on chromosome 22 to a gene of the ETS family, friend leukemia insertion (*FLI1*) on chromosome 11. The EWS-FLI1 fusion protein acts as an aberrant transcription factor, therefore, the EWS-FLI1 fusion *viral protein* is implicated in the pathogenesis of tumors of the Ewing's sarcoma family.

The survival of patients with these tumors depends on the initial manifestation of the disease. Most patients suffer a localized disease, and about 20% have metastatic diseases, mostly metastases in the bone, the bone marrow and the lungs.

Ewing's sarcoma (ES) is a highly malignant primary bone tumor derived from the red bone marrow and is histologically related to reticulum cell sarcoma.

Symptoms depend mostly on the location of the tumor and its metastases. The tumor is frequently located in the metaphysis or diaphysis of the long bones of the extremities though it may also occur in the pelvic area, the ribs and scapulae. Fever and loss of weight often indicate a metastatic disease. Pathologic fractures of the long bones, pain in the back, indicating a paraspinal, retroperitoneal, or deep pelvic tumor, are characteristic. Patients with lung metastases can have pleural signs and asymmetric breath sounds, while those with significant bone marrow metastases have petechiae or purpurae on the skin, due to thrombocytopenia.

The cause of Ewing's sarcoma is an inherited predisposition and a combined infection caused by a *sarcoma virus and HTLV* and most frequently by *Mycoplasma fermentans*. Many other various secondary infections are often present, too.

Diagnosis: symptomatically, by blood tests (blood culturing, CRP, CBC count, etc.) in order to distinguish it from other diseases. By biopsy and immunohistochemical analysis. By radiology, CT, MRI, PETscan, FDG-PETscan.

Treatment: by administering cancer chemotherapy, by radiation therapy and specific surgery, depending on the growth, location and other characteristics of the tumor.
RFR method: detects and may eliminate the infective microorganisms.
The most frequent resonances are: 318, 343, 348-354, 370-372, 395-406, 436-438, 442-451, 459-464, 470-473, 488, 491-493, 496, 511-519, 523-527, 530-540 kHz
Consecutive treatment is most effective.

26.32. Fibrosarcoma

Fibrosarcoma (FS) is a malignant tumor derived from the fibrous connective tissue and is characterized by immature proliferating fibroblasts or undifferentiated anaplastic spindle cells. It can be a soft-tissue mass, or a primary or secondary bone tumor.
A Primary fibrosarcoma of bone is a fibroblastic malignancy, producing variable amounts of collagen. It is either centrally arising within the medullary canal, or peripherally from the periosteum.
The Secondary fibrosarcoma of bone arises from a preexisting lesion, or following a radiotherapy to an area of bone, or soft tissue. It is an aggressive tumor with sad prognosis. These tumors may be low grade differentiated, intermediately malignant and high malignant, i.e. anaplastic.
Symptoms: Sarcomas involving the bone cause, but only after their long duration, pain and swelling. They grow to be large enough to threaten the structural integrity of the bone and can cause at first pathologic fractures.
Soft-tissue sarcomas are usually painless masses. Their symptoms are usually nonspecific, being a fixed, firm mass, the affected area may be tender. These tumors arise often deep in the muscular fascia, and can become large tumors, before detected.
Most lesions develop around the knee, proximally in the femur, or in the arm and in the hip region. In advanced cases neurological or vascular changes can also come about.
Diagnosis: by CT, MRI, PETscan and by biopsy and histology with special staining methods.
Differential diagnosis: by distinguishing it from osteosarcoma, Paget sarcoma, malignant fibrous histiocytoma, fibrous dysplasia, fibrous histiocytoma etc.
Treatment: by radiation treatment and chemotherapy in order to improve the local control and to lessen the risk of metastasizing. By surgery, combined with radiation therapy or/and chemotherapy. Symptomatically.
RFR method: detects and may eliminate the causative virus.
The most frequent resonances of this virus are: 441-451, 513-516, 533 kHz

26.33. Lipoma and Liposarcoma

26.33.1. Lipomas

Lipomas are benign tumors composed of mature fat cells. They are the most common benign mesenchymal tumors. Lipomas are found in the subcutaneous tissues and, less commonly, in the internal organs as well. These slow-growing, benign fatty tumors form soft, lobulated masses enclosed by thin, fibrous capsules. Although it has been hypothesized that lipomas may rarely undergo a sarcomatous change, this has never been convincingly documented.
Lipomas differ biochemically from the normal fat by showing increased levels of lipoprotein lipase and by the presence of an enhanced number of precursor cells.
In the gastrointestinal tract, lipomas are submucosal fatty tumors. Their most common locations include the esophagus, stomach and the small intestine. Their symptoms are caused by luminal obstruction and bleeding. Duodenal lipomas are mostly small but may

become pedunculated obstructing the lumen. Lipoma may also be found near to the endocrine organs, f.i. the thyroid, the adrenal glands, pancreas and the parathyroid glands. Maxillofacial lipomas do also occur. In rare instances, intraosseous, intra-articular and mediastinal involvement can also come about. Gynecologic lipomas may occur in the uterus, ovaries and the broad ligament, too.
There are other fatty tumors, f.i. lipoblastomas, hibernomas and atypical lipomatous tumors as well.
Lipomas are classified into the following categories:
Solitary lipomas are the most common ones. Most solitary lipomas are superficial and small. Solitary lipomas may develop with weight gainbut but usually do not shrink after weight loss.
Diffuse congenital lipomatosis can be characterized by poorly demarcated lipomas localized mostly on the trunk.
Benign symmetric lipomatosis (Madelung disease).
Lipomas of the head, neck, shoulders and the proximal part of the upper extremities characterize this disorder. These patients are often alcoholists or suffer from diabetes. Malignant tumors of the upper airways, hyperuricemia, obesity, renal tubular acidosis, peripheral neuropathy and liver disease are often associated with this disease.
Familial multiple lipomatosis
This clinical entity is characterized by few-to-many, small, well-demarcated, encapsulated lipomas that commonly involve the extremities. This form typically appears during or soon after adolescence. The neck and shoulders are spared.
A family history of multiple lipomas does usually exist. This disease is in an autosomal dominant way inheritable.
Dercum disease (adiposis dolorosa) As to this disorder [redacted]
Angiolipomas are a rare form of lipomas. These tender, soft, subcutaneous, frequently multilobulated nodules are present typically in adolescence. The associated pain is vague and may develop spontaneously or caused by pressure.
Hibernomas are located in the interscapular region, axillae, neck and mediastinum. Histologically they are composed of embryonic brown lipoblasts termed mulberry cells.
Genetic predisposition of lipomas: Though an exact etiology of lipomas is uncertain as yet, an association with gene rearrangements of chromosome 12 was established in cases of solitary lipomas, as having an abnormality in the HMGA2-LPP fusion gene.

26.33.2. Liposarcomas

Liposarcoma is a malignant tumor of fat cells. In adults, it is the most common soft tissue sarcoma. Liposarcoma normally appears as a slowly enlarging, painless, nonulcerating submucosal mass in a middle-aged person, though some lesions grow rapidly and become ulcerated early. The commonly affected loci include the thigh, the gluteal region, retroperitoneum, leg and the shoulder area. Liposarcomas rarely arise from preexisting lipomas.
Liposarcoma occurs in three main clinic forms:
(1) well-differentiated liposarcoma;
(2) myxoid and/or round cell liposarcoma and
(3) pleomorphic liposarcoma.
There can be still an other form, the mixed-type liposarcoma which is a combination of all these morphologic types.
Genetical predisposition of liposarcomas: an abnormality of band 12q13 can be associated with the development of liposarcomas. The most common chromosomal translocation form is the FUS-CHOP fusion gene, which encodes a transcription factor necessary for the differentiation of adipocytes.

Liposarcoma develops caused by a viral and mycoplasmal coinfection. The viral agents are *Sarcoma viruses* and *HTLVs*, the most frequently occuring mycoplasmal agent of this disease is the *M. fermentans*. Liposarcoma is a systemic infectious disease, not localized to one region or organ.
The role of trauma has been emphasized in several papers as a causative factor of liposarcoma, this causal relationship is, however, difficult to assess as minor traumas do frequently occur. The interval between trauma and the development of a liposarcoma is quoted to be 6-16 months. I think, that no liposarcoma does develop without any viral and mycoplasmal infection.
Liposarcoma is a tumor affecting adults. It is a tumor of large connective tissue spaces in which the cells have retained their ability for lipogenesis. Their principal loci of involvement are the intermuscular gliding spaces and the perivascular and subcoelomic regions. These tumors occur mostly in the lower limbs, particularly in the popliteal fossa, with a special preference for the adductor canal, the medial thigh, the shoulder area and the retroperitoneal, perirenal, and mesenteric regions.
Its most common intra-abdominal location is the posterior perirenal area, where it tends to displace the kidney medially and anteriorly. Though a renal invasion is usually not experienced, the tumor does often compress the renal pelvis and sometimes the ureter, and invades the anterior or lateral abdominal wall.
Its mortality and morbidity rate depends on the locus and the histologic type of the liposarcoma.
Diagnosis: the well-differentiated tumors are easily characterized as fat-containing masses, although the differentiation between a malignant tumor and a benign one is clinically difficult. Poorly differentiated tumors can not even with MRI be differentiated from other mesothelial tumors, lymphomas, metastases and inflammatory masses.
Treatment: by surgery
RFR method can detect and eliminate the pathogen microorganisms.
The most frequent resonances of lipomas are: 308-312, 360-364, 442-451 kHz
The most frequent resonances of liposarcomas are: 308, 360-365, 370-374, 382, 442-451, 470-473, 488-496, 513-534 kHz

26.34.3. Myelofibrosis

Myelofibrosis is a myeloproliferative disease in case of which the proliferation of an abnormal type of bone marrow stem cell causes fibrosis, i.e. the replacement of the bone marrow with collagenous connective tissue fibers. The hallmark of myelofibrosis is an increased reticulin staining. The fibrous network observed in myelofibrosis is collagenous and contains fibronectin; the reticulin stain reacts with a protein which is intimately associated with type III collagen and is generally considered to be a form of procollagen. The fibrosis of the bone marrow presumably reflects the overgrowth of the normal marrow matrix.
The cause of a primary or idiopathic myelofibrosis is a genetic predisposition and a combined infection of the patient.
Genetic predisposition: Chromosomal imbalances, such as the gain of 9p, 13q, 2q, 3p and 12q are the most commonly seen abnormalities. All other abnormalities confer an independent adverse effect on the survival and are also associated with higher JAK2V617F mutational frequency. The gain-of-function V617F mutation in the JAK2 gene (on chromosome 9p) can be seen in many adult patients suffering from idiopathic myelofibrosis. The presence of this mutation correlates with a shift from thrombopoiesis toward an increased erythropoiesis and may also predict a progression to massive splenomegaly and to leukemic transformation.
Infections caused by *HPVs, HTLVs (mostly HTLV-1 and HTLV-3) and Mycoplasma fermentans* play also an important role in the development of this illness.

Symptoms: myelofibrosis patients typically show a fairly insidious onset of pallor and fatigue with or without fever, bruising, pain of the bones, and the left upper quadrant of the abdomen. The bone marrow will be replaced by collagen fibrosis, impairing the patient's ability to produce new blood cells, and resulting thus a progressive pancytopenia. An extramedullary haematopoeisis can occur if the haemopoetic cells migrate away from the bone marrow to the liver and spleen. Patients often have hepatosplenomegaly and poikilocytosis.

Diagnosis: by histological analysis of the blood and of the bone marrow smear. Expected findings are anemia and/or thrombocytopenia with or without leukocytosis with left shift. The peripheral blood smear may have an erythroblastic appearance. The bone marrow biopsies show marked fibrosis with pockets of fibroblasts and atypical megakaryoblasts. Less commonly, a hyperplasia with the predominance of the megakaryocytic and erythroid precursors can be observed.

Differential diagnosis: By distinguishing it from Acute myelocytic leukemia, myelodysplasia, histoplasmosis, lymphohistiocytosis, hyperparathyroidism, osteopetrosis and Gray platelet syndrome.

Treatment: by administering folic acid, allopurinol, dexamethasone, alpha-interferon, hydroxycarbamide, Lenalidomide and Thalidomide. By blood transfusions and stem cell transplantation.

RFR method: can detect and eliminate the pathogen microorganisms.

The most frequently present resonances in case of myelofibrosis are: 340, 343-347, 353, 370-374, 396, 410, 418-426, 430-433, 442-451, 493-495, 514, 525-527, 543-546 kHz

26.34. Myeloproliferative Diseases and Myelodysplastic Syndromes

Myeloproliferative Diseases (MPDs), named also myeloproliferative neoplasms, are illnesses of the bone marrow in which case excess amounts of cells are being produced. Chronic myeloproliferative diseases involve: Chronic Myelogenous Leukemia (CML); Polycythemia Vera (PV), Essential Thrombocythemia (ET), Agnogenic Myeloid Metaplasia (AMM), Chronic Neutrophilic Leukemia (CNL) and Chronic Eosinophilic Leukemia (CEL)/Hypereosinophilic Syndrome (HES), which later is also known as Myelofibrosis (MF). MPDs are related to and may evolve into myelodysplastic syndromes and into acute myeloid leukemia, though myeloproliferative diseases generally have a far better prognosis than these latter illnesses. In the recently established WHO Classification of Hematologic malignancies this group of diseases was renamed from myeloproliferative diseases into myeloproliferative neoplasms. This nomenclature reflects the underlying clonal genetic changes which are the salient characteristics of these illnesses.

Genetic predisposition: in case of **chronic myelogenous leukemia** the tyrosine kinase activity of the *bcr-abl* hybrid gene is increased. In case of **polycythemia vera** and **essential thrombocythemia** the prevalent genetic lesion appears to be the substitution of valine to phenylalanine at the amino acid position 617 (V617F) within the Janus kinase 2 *(JAK2)* gene. This lesion produces hypersensitivity to erythropoietin. (In case of myelofibrosis the leukemic transformation is probably not related to the JAK-2 (V617F) mutation state). **Systemic mastocytosis** is linked with the D816 mutation of the *KIT* gene. The *FIP1L1-PDGFR* mutation has been identified in a subgroup of people suffering from systemic mastocytosis with eosinophilia.

Chronic myelogenous leukemia (CML) is Philadelphia Chromosome positive.

Polycythemia Vera (PV), Essential Thrombocytosis (ET) and Myelofibrosis (MF) are Philadelphia Chromosome negative.

Myelodysplastic Syndromes (MDSs) is a collection of hematologic disorders with an ineffective production/or dysplasia of myeloid blood cells with the risk of getting

transformated into Acute Myelogenous Leukemia (AML). A severe anemia requiring repeated blood transfusions is frequently present. Myelodysplastic syndromes are bone marrow stem cell disorders characterized by a damaged and ineffective hematopoiesis caused by irreversible quantitative and qualitative defects of the hematopoietic cells. The course of this disease is usually chronic, when a gradually worsening cytopenia will develop due to the progressive bone marrow failure. A significant increase of the apoptotic cell death can be experienced as well as an impaired differentiation of the blood precursor cells. The clonal expansion of the abnormal cells results in the production of cells unable to differentiate. The progression of MDS to AML is a good example of the multi-step theory of carcinogenesis provoked by infective agents, in which case in an initially normal cell a series of mutations occurs transforming it into a cancer cell.

Genetic predisposition in case of MDSs: the loss of the long arm of chromosome 5 has been associated with dysplastic abnormalities of the hematopoietic stem cells in the 5q-syndrome. The MDS is thought to arise due to mutations in the multi-potent bone marrow stem cells, though the specific defects responsible for these diseases are as yet poorly defined.

Infections are transformic factors in case of MPDs and MDSs, the infective agents cause series of mutations occuring in an initially normal cell, transforming it thus into a cancer cell. These infections are combined chronic viral and mycoplasmal aggressive attacks.

The viral components are usually *Human T-cell Lymphotropic Viruses (HTLVs), Human B-cell Lymphotropic Viruses (HBLVs), Human Myeloid Leukemia Viruses (HMLVs)* and *Human Papilloma Viruses (HPVs).*

The Mycoplasmal components are most frequently *M. fermentans* and *M. penetrans* and rarely *other Mycoplasma species* as well.

Symptoms: Some patients complain of a gradual onset of fatigue and weakness, dyspnea and pallor, but at least half of all the patients are asymptomatic and their MDS is discovered only incidentally by routine blood examinations. Previous chemotherapy and radiation exposure are important medical historical data. Fever and weight loss should point to a myeloproliferative rather than to a myelodysplastic process.

The singals of a good outcome concerning MDS are: Younger age, normal or moderately reduced neutrophil and platelet counts, low blast counts in the bone marrow(<20%) and no blasts in the blood, no Auer rods, ringed sideroblasts, normal karyotypes or mixed karyotypes without complex chromosome abnormalities.

The signals of a bad outcome concerning MDS are: Advanced age; Severe neutropenia and thrombocytopenia, high blast count in the bone marrow (20-29%) and blasts in the blood; Auer rods, the absence of ringed sideroblasts, abnormal localization and immature granulocyte precursors in the bone marrow section, mostly abnormal karyotypes or complex marrow chromosome abnormalities and the leukemic growth pattern of the in vitro bone marrow cultures.

Diagnosis: depending on the symptoms of the myeloproliferative disorder diagnostic tests can include red cell mass determination (for polycythemia), bone marrow aspirate and trephine biopsy, arterial oxygen saturation and carboxyhaemoglobin level examinations, neutrophil alkaline phosphatase level tests, vitamin B12 (or B12 binding capacity) tests and serum urate tests and gene mutation tests (BCR-ABL translocation, thrombopoietin receptor mutation, JAK2 mutation)

Children with Down syndrome are susceptible to MDS, and the family history may indicate a hereditary form of sideroblastic anemia or Fanconi anemia.

Treatment: the goals of the therapy are to control the symptoms, improve the quality of life, improve the overall survival, and decrease the progression to an acute myelogenous leukemia. By giving erythropoetin, cytostatic drugs, such as Azacytidine, Decitabine and Lenalidomine.

These treatments do not stop the chromosome abberation and the viral infections.
RFR-method: detects and can eliminate the pathogen agents.
The most frequent resonances of the Mycoplasmal infection are: 440-451 kHz
The most frequent resonances of the different viral infections are: 313-315 *(HTLV)*; 329-324 *(HTLV)*; 339-340 *(HTLV)*; 353-354 *(HTLV)*; 370-374 *(HTLV)*; 402-403 *(HPV)*; 418-426 *(HPV)*; 432-433 *(HTLV)*; 470-473 *(Sarcoma)*; 476-479 *(HPV)*; 517-521 *(HPV)*; 543-546 *(HPV)*; 567 *(HTLV)* kHz

26.34.1. Myeloid Leukemia

Acute myeloid leukemia (myelotic, myelogenous, myeloblastic, myelomonocytic) is a life threatening illness, in which myelocytes become cancerous and replace, often very rapidly, the normal cells in the bone marrow. Chronic myeloid leukemia is a disease in which a cell in the bone marrow becomes cancerous and produces a large number of abnormal granulocytes.
This disease may affect people of any age and of either sex, but is uncommon in children under 10 years old. The leukemia cells accumulate in the bone marrow, destroying and replacing cells that produce normal blood cells. They are released into the bloodstream and transported to other organs, where they continue to grow and divide. They can form small tumors in, or just under the skin, and can cause meningitis, anemia, liver and kidney failure and other organ damages as well. Most leukemic granulocytes are produced in the bone marrow, but some are produced by metastases in the spleen, liver and other organs. Leukemic granulocytes tend to crowd out normal cells in the bone marrow, often leading to the formation of large amounts of fibrous tissues that replace the normal bone morrow. During the course of the disease, more and more immature granulocytes enter the bloodstream and bone marrow. During this phase, anemia and thrombocytopenia will develop, and the proportion of immature white blood cells and blastcells will increase dramatically. Eventually, after a long time, the further dedifferentiation of myeloid leukemia cells will lead to its development into a reticulosarcoma.
Sometimes the leukemic granulocytes undergo more changes, and the disease progresses to blast crisis. In case of blast crisis, the cancerous stem cells begin to produce only immature granulocytes, a sign that the disease has become much worse. At this time, chloromas can grow in the skin, bones, brain and lymph nodes.
Symptoms include weakness, shortness of breath, infection, fever and bleeding. Other symptoms may include headache, vomiting, irritability, fatigue, bone and joint pain.
In its early stages, chronic myelocytic leukemia may produce no symptoms. However, some people get fatigue and weak, lose their appetite and their weight, develop fever and sweating at night. Lymph nodes and spleen may get enlarged. Over time, the patients become very ill as the number of their red blood cells and platelets decreases, leading to paleness, bruising and bleeding. Fever, lymph node enlargements, and formation of skin nodules filled with leukemic granulocytes and chloromas are also experienced.
Diagnosis: by blood examinations, bone marrow biopsy, by x-ray, CT, MRI and ultrasound.
Treatment: by chemotherapy (Hydroxyurea, Busulfan, etc.), by radiation therapy of the spleen.
RFR method: detects and may eliminate the pathogen resonance frequencies!
The most frequent resonant frequencies are: 311, 314, 330-340, 353, 372, 402-410, 420-437, 446, 450-452, 461-469, 496, 513-515, 540-544, 558-559, 567-573 kHz
This list may be incomplete.

26.34.2. Polycythemia Vera

Polycythemia vera is a stem cell disorder, characterized as a panhyperplastic, malignant, and neoplastic bone marrow disorder. The most prominent feature of this disease is an elevated absolute red blood cell mass caused by an uncontrolled red blood cell production. This is accompanied by increased white blood cell (myeloid) and platelet (megakaryocytic) production, due to an abnormal clone of the hematopoietic stem cells characterized by an increased sensitivity regarding the different growth factors of maturation.

Pathophysiology: Normal stem cells are present in the bone marrow of patients with PV. There are also abnormal clonal stem cells present, that interfere with or suppress the normal stem cell growth and maturation. The etiology of panmyelosis is an unregulated neoplastic proliferation. The cause of this stem cell transformation is an infection with *retroviruses* and several, different other infections such as *Mycoplasma, EBV and Cytomegalovirus*. Retrovirus infections have the potency to command the host's cells to divide.

The progenitor blood cells of these patients display abnormal responses to growth factors. JAK2 is the most likely affected gene involved in the pathogenesis of PV which is directly involved in the intracellular signaling following an exposure to cytokines concerning which these PV progenitor cells display hypersensitivity. A unique, recurrent, acquired clonal mutation in JAK2 has been recently found among most PV patients and those suffering from other Myeloproliferative Diseases (MPDs) including essential thrombocythemia and idiopathic myelofibrosis.

Thrombosis and bleeding, caused by disrupted hemostatic mechanisms owing to an elevated RBC ad platelet counts, do frequently occur among persons with PV and MPD. As concerns clotting, tissue factors, polymorphonuclear leukocytes, the platelet surface as a contributor to phospholipid-dependent coagulation reactions and the entity of microparticles play all an additional role.

Symptoms are related to hyperviscosity and thrombosis, leading to a poor oxygen delivery, the signs of which include headache, dizziness, vertigo, tinnitus, visual disturbances, angina pectoris and intermittent claudications. Splenomegaly and hepatomegaly can often be experienced. Erythrosis of the face, palms, nailbeds, mucosa and conjunctiva are characteristic. Among patients with PV hypertension is common. The red blood cell mass should differentiate PV from the Gaisbock syndrome, meaning hypertension and pseudopolycythemia. Bleeding complications. including epistaxis, gum bleeding and ecchymoses come also about.

Thrombotic complications include venous thrombosis, thromboembolism and an increased prevalence of getting stroke and some other arterial thromboses.

Abdominal pain can be caused by increased histamine levels and gastric acidity causing ulcer, or by Budd-Chiari syndrome (hepatic portal vein thrombosis) and mesenteric vein thrombosis. Splenomegaly or splenic infarction can also come to pass. Pruritus develops from increased histamine levels released from increased numbers of basophil and mast cells and can be exacerbated by warm bathing and showering.

Diagnosis: symptomatically, by PCR-based methods in order to detect JAK2 V617F mutation, which may become the first molecular diagnostic marker of PV, similar to BCR/ABL of CML. By serum Epo assay, by cytogenetic karyotyping of the bone marrow cells, by clonal assays, by studying bone marrow morphology and histology, by CT scan and ultrasound, etc.

Treatment: symptomatically, as there is no specific therapy regarding PV.

The risk of developing secondary leukemia depends on the type of the therapy (f.i. phlebotomy, radioactive phosphorus P 32, chlorambucil) or on the type of the myelosuppressive agents (f.i., hydroxyurea, anagrelide, interferon alfa) and on the duration of the therapy. By splenectomy (in case of a painful splenomegaly or in case of repeated episodes of thrombosis causing splenic infarction).

These medications do not solve the problem of the infection triggering this illness.

RFR method: detects and eliminates the causative pathogens!
Patients suffering from polycythemia vera are usually infected by several different pathogen microorganisms.
The most frequent resonances are: 287-301, 313-319, 343-345, 355-362, 372-383, 408-410, 442-451, 449-452, 530-544, 560-568 kHz

26.35. Lymphoid Leukemia

Lymphoid leukemia are cancers of the lymphatic system. Cancerous lymphocytes can be localized in a sole lymph node or can spread throughout the body. This type of leukaemia consists of two types of lymphoma, i.e. the Hodgkin's lymphoma, and the group of non Hodgkin's lymphomas.

26.35.1. Hodgkin's Disease

Hodgkin's disease is a lymphoma, characterized by Reed-Sternberg cells which are large cancerous lymphocytes.
Symptoms: The first noticeable symptoms are enlarged lymph nodes in a single area, f.i. on the neck, or the groin, or throughout the body. The lymph nodes and the spleen get enlarged slowly and are usually painless. Enlarged lymph nodes in the tonsils cause occasionally difficulty in swallowing. Enlarged lymph nodes deep within the chest or abdomen may press against various organs, causing difficulty in breathing, loss of appetite, severe constipation, abdominal pain, or the progressive swelling of the legs. These enlarged lymph nodes deep within the chest are sometimes found unexpectedly by a rutin chest x-ray examination. Fever, sweating at night, anemia, fatigue and loss of weight are often present.
The cause of Hodgkin's lymphoma is probably an infection caused by an unidentified *virus* and by some *species of the Chlamydia family.*
Diagnosis: by blood and lymph node examinations with microscope. By CT.
Treatment: by combination chemotherapy according to the protocol (f.i. MOPP, ABVD etc.)
RFR method: detects the resonance frequencies! RFR method is not suitable for diagnosing!
The most frequent resonant frequencies present in Hodgkin's disease are: 317-319, 379-383, 389, 429, 440, 444, 456, 480-483, 566 kHz
Advised alternative therapy: together with a clinical laboratory control: eliminate the pathogens on their resonance frequencies! If there is no good effect within three weeks RFR method will be ineffective.

26.35.2. Non-Hodgkin's Lymphoma

Non-Hodgkin's lymphomas are a group of related cancers originating in the lymphatic system and usually spreading throughout the body. Non-Hodgkin's disease is more common than Hodgkin's disease. The cause of non-Hodgkin's lymphoma may be an unidentified *virus,* though the disease does not appear to be contagious. A rare type of progressive non-Hodgkin's lymphoma is related to an infection caused by *HTLV-1*, a retrovirus similar in function to the human immunodeficiency virus causing AIDS. Non-Hodgkin's lymphoma can also be a complication of AIDS.
Symptoms: include infiltrations of lymphoma cells into the bone marrow, spleen, blood, skin, intestine, brain and spinal cord. These infiltrations cause anemia, rashes, neurological symptoms, such as weakness and abnormal sensations. Blocked lymph vessels in the chest cause fluid accumulations around the lungs. The susceptibility to severe bacterial infections and the invasion of lymphoma cells into the bone marrow and lymph nodes, is caused by a decreased normal antibody production. Destruction of red blood cells occurs in

the enlarged and overactive spleen. The destruction of red blood cells by abnormal antibodies causes hemolytic anemia. The invasion by lymphoma cells leads to the destruction of the bone marrow.
According to classification systems, cell types of lymphoma determine the prognosis of the disease. Lymphomas can be categorized into low grade, intermediate grade and high grade types, the latter having the most unfavourable prognosis.
Diagnosis: by biopsy of the lymph node and microscopic examinations. By determining the types of cells.
Treatment: by combination chemotherapy according to the protocol (CVP, CHOP, C-MOPP etc):
RFR method: detects the resonance frequencies! But does not diagnose!
This group of lymphomas is extremely variable, the cytogenic and morphologic evidence suggests the involvement of all elements, derived from the stem cell, while the tumor process is able to dedifferentiate the cells from lymphoma to reticulumsarcoma.
The resonant frequencies of Human T-cell Lymphotropic Virus-1 are: 311-314, 330-331, 370-376, 406, 432-435, 496-504 kHz
The most frequent other frequencies are: 340, 353, 402-410, 420, 426, 442-452, 513, 536, 544-545 kHz
This lymphoma may get transformed into lymphosarcoma or reticulumsarcoma.
The resonant frequencies of lymphosarcoma are: 493-500 kHz
The resonant frequencies of reticulumsarcoma are: 496-514 kHz
Advised alternative therapy: together with a clinical laboratory control: eliminate the pathogens on their resonant frequencies! If there is no good effect within three weeks RFR method will be ineffective.

26.35.3. Mycosis Fungoides

Mycosis fungoides is a rare, persistent, slow-growing type of non-Hodgkin's lymphomas that originates from a mature T-lymphocyte and affects the skin; it may affect the lymph nodes and the internal organs as well.
Its **symptoms** are long lasting itchy rashes, later developing nodules, spreading slowly. Some patients develop a Sezary syndrome, characterized by abnormal lymphocytes present in the blood.
Diagnosis: by blood examinations and lymph node biopsy examinations.
Treatment: by chemotherapy, PUVA.
Its resonant frequencies are: 397-400, 434-440, 442-451, 570-580 kHz
Advised alternative therapy: together with a clinical laboratory control: eliminate the pathogens on their resonant frequencies! If there is no good effect within two weeks RFR method will be ineffective.

26.35.4. Burkitt's Lymphoma

Burkitt's lymphoma is a very high grade non-Hodgkin's lymphoma, originating from a B-lymphocyte, and tends to spread to areas outside the lymphatic system, such as the bone marrow, blood, central nervous system and the spinal fluid. This lymphoma often develops in people suffering from AIDS. It is caused by *EBV*, which is also the cause of human infectious mononucleosis. Burkitt's lymphoma progresses rapidly and is fatal.
Treatment: by combination chemotherapy according to the protocol (CMVDCy).
RFR method: detects the resonance frequencies! But it is not suitable for diagnosing.
The most frequent resonances are: 337, 339-347, 352, 372-382, 397-398, 403-410, 422, 424, 476, 491, 516, 518, 528, 560 kHz

Advised alternative therapy: together with a clinical laboratory control: eliminate the pathogens on their resonant frequencies! If there is no good effect within three weeks RFR method will be ineffective.

26.35.5. Chronic Lymphocytic Leukemia

Chronic lymphocytic leukemia is characterized by a large number of mature cancerous lymphocytes and by enlarged lymph nodes. B-cell leukemia is the most common type. The other type is hairy cell leukemia, a rare type of leukemia.
Treatment: according to protocols, usually, by combination chemotherapy.
RFR method: detects the resonance frequencies! But does not diagnose!
The most frequent resonant frequencies of Hairy cell leukemia are: 318, 399, 440-452, 477, 496-498 kHz
This list is not complete yet.
Advised alternative therapy: together with a clinical laboratory control: eliminate the pathogens on their resonant frequencies! If there is no good effect within two weeks RFR method will be ineffective.

26.35.6. Plasmacell Dyscrasias

The group of plasmacell dyscrasias consists of several disorders generally characterized by an uncontrolled proliferation of cells normally involved in antibody synthesis. These disorders are classified depending on the antibodies:
Multiple myeloma (IgG, A, D and E),
Macroglobulinemia (IgM),
Heavy chain diseases (gamma chain).
Plasmacytoma is a tumor of neoplastic monoclonal plasma cells either in the bone or extramedullary, where a clone of abnormal plasma cells multiplies, forms tumors in the bone marrow, and produces a large quantity of abnormal antibodies, accumulating in the blood, other tissues and the urine. Its types include extramedullary plasmacytoma, solitary bone plasmacytoma, myeloma, multiple myeloma, plasmablastic sarcoma.
The skeletal plasmacytoma type frequently disseminates and develops into multiple myeloma over the course of 5-10 years. The soft tissue plasmacytoma type occurs mostly in the respiratory tract, rarely disseminates and can be cured by resection.
The dynamic of the development of the plasma cell tumors: there develops a precancerous cell from a plasma cell, then the soft-tissue or nonosseous extramedullary plasmacytoma (EMP), or the solitary bone plasmacytoma (SBP), from this stages these cells can develop into multiple myeloma cells, and finally, they may develop into plasmablastic sarcoma cells. Plasmacytoma can arise in any part of the body. A solitary bone plasmacytoma (SBP) arises from the plasma cells located in the bone marrow, whereas extramedullary plasmacytoma (EMP) is thought to arise from plasma cells located in mucosal surfaces.

26.35.6.1. Plasmacytoma

Plasmocytoma is a well-defined neoplastic proliferation of abnormal plasmacells arising from the bone marrow, or other soft tissue sites, in the absence of a generalized plasma cell disease. It is most often an isolated tumor of bone with osteolytic potency, or a visceral or soft tissue mass. Solitary bone plasmacytoma is an uncommon form of plasmacytoma and is localized to the involved bone, it develops mostly in the pelvic bones, spine, ribs and skull, but it develops in other areas as well, particularly in the lungs, the proximal airways and the reproductive organs. Pulmonary plasmocytomas can be observed by x-ray as a nodule, or a lobulated mass, the rarely developing tracheal plasmocytoma is usually a focal polypoid tumor, detectable only by CT scanning. The abnormal plasma cell almost always

produces a large quantity of abnormal antibodies, and decreases thus the amount of normal antibodies.
Plasmocytomas are caused by an infection of the B lymphocyte with the *Human B-cell Lymphotropic Virus (HBLV)*. Pieces and fragments of these abnormal antibodies frequently end up in the kidneys, damaging them and causing sometimes kidney failures. Deposits of antibody fragments in the kidneys or other organs can lead to amyloidosis. This illness is the result of a damaged and inadequate immune response. Also anemia results when the abnormal plasma cells crowd out the normal cells producing red blood cells in the bone marrow.
Symptoms: patients with a solitary plasmocytoma can be asymptomatic or can feel pain, resulted from bony erosions. The other symptoms depend on the site of the lesion, f.i. a solitary plasmacytoma of the skull may cause cranial nerve palsy. Endobronchial plasmocytomas can cause atelectasis and obstructive pneumonitis with fever and anemia. Though the first symptoms are usually anemia and pain in the bone, especially in the spine or ribs, and the weakening of the bone, causing fracture. Neurological symptoms can include confusion, headache and visual problems.
If not treated effectively, most patients progress to multiple myeloma over years. Malformation into plasmoblastic sarcoma or other similar malignancies can also develop.
Diagnosis: by complete blood analysis, immune electrophoresis, Bence Jones protein examinations, by x-ray, CT and MRI. Symptomatically.
Treatment: Symptomatically, by administering strong analgesics. By radiation therapy. By surgical excision, by chemotherapy.
RFR method: detects and eliminates the Human B-cell Lymphotropic Virus, or the plasma cell infiltrating virus.
The most frequent resonances of plasmacytoma are: 427-432, 442-451, 476-479, 485-490, 525-527 537-539, 545-549 kHz
The most frequent resonances of plasmablastic sarcoma are: 408-421, 442-451, 470-473, 476-479, 485-496, 513, 525-549 kHz

26.35.6.2. Multiple Myeloma

Multiple myeloma is the most common form of plasma cell dyscrasias.
This disease is a plasma cell cancer in which a clone of abnormal plasma cells multiplies, forms tumors in the bone marrow, and produces a large quantity of abnormal antibodies accumulating in the blood and the urine system. In advanced stage the proliferating plasma cells cause diffuse osteoporosis, characteristic punched-out bony lesions often involving the skull, pathologic fractures most frequently of the vertebrae and ribs. The osteclast stimulating effect of the invading plasma cells causes the dissolution of the bone, leading to hypercalcemia. The abnormal plasma cells produce continously a large quantity of abnormal antibodies while the production of normal antibodies will be reduced. Multiple myeloma patients are therefore especially susceptible to infections.
Symptoms of advanced cases osteoporosis, pathological fracture of the bone, anemia, frequent and recurrent infections, mostly bacterial pneumonias, are characteristic. The abnormal antibodies ending up in the kidneys damage them, causing kidney failures. Deposits of these antibody fragments in the kidney and other organs can lead to amyloidosis. In case of amyloid deposits in the kidneys, nephrosis can develop. Recurrent pyelonephritis, hyperuricemia due to rapid cellular turnover can also come to pass. The reabsorption of large amounts of Bence Jones proteins filtered by the glomeruli cause tubular damages, destroying the nephrons, causing thus proteinuria, nephrotic syndroma and uremia. This state of the adult patient is named Fanconi syndrome, showing all the clinical symptoms of renal tubular acidosis.
Due to the hyperviscosity of the blood, neurologic symptoms, such as confusion, visual problems and headache can also come to pass.

Plasmacytomas are caused by infection of *Human B-cell Lymphotropic Virus*. *Human B-cell Lymphotropic Virus particles* have been demonstrated repeatedly in a patient suffering from multiple myeloma. Plasmacytoma may transform into lymphosarcoma and then reticulosarcoma. By these processes the frequency of the found viral resonance increases. I think, that it does not mean a new virus appearing in the body of the patient, but rather a typical tumorous virus transformation of the same *HBLV virus*.

Diagnosis: by laboratory examinations of Bence Jones protein (serum protein and urine electrophoresis and immunoelectrophoresis), by anemia, calcium levels, x-ray, biopsies, CT, MRI. Symptomatically.

Treatment: by administering analgesics, hydratation and corticosteroids. by radiation therapy, combination chemotherapy according to the protocol.

RFR method: detects and eliminates the pathogen microorganisms!

The most frequent resonances of Human B-cell Lymphotropic Virus-1 in case of Multiple myeloma are: 243, 485-490 kHz

The most frequent resonances of "Human Lymphoblast causing virus" in case of Lymphosarcoma are: 245-247, 492-497 kHz

The most frequent resonances of "Human Reticulosarcoma causing virus" are: 248-257, 502-514 kHz

Some other pathogen participants, such as *Mycoplasma species*, *other Lymphotropic Viruses* and *different bacteria* are also to be found in case of this tumorous process.

The most frequent other resonances are: 304, 327-331, 340, 377-380, 421, 428-432, 438, 486, 510, 514, 538-549 kHz

26.36. Multiple Endocrine Neoplasia

The term multiple endocrine neoplasia (MEN) consists of several distinct syndromes featuring tumors of endocrine glands, each according to its own characteristic pattern. In some cases, the tumors are malignant, in others, benign. Benign or malignant tumors of endocrine tissues are components of some of these tumor syndromes.

MEN syndromes are inherited as autosomal dominant disorders. According to the current classification they are sorted into type 1 MEN and type 2 MEN, with subcategories type 2A MEN (Sipple syndrome) and type 2B MEN.

MENIN gene is a tumor suppressor gen influencing the production of a protein named menin. The loss of this protein leads to tumorgenesis. Alternatively, ret protein produced by the RET gene, which is a proto-oncogene, can be continuously activated, causing abnormal cell proliferation (See also chapter 24.11)

Type 1 MEN is defined by hyperfunctioning tumors in all 4 parathyroid glands, pancreatic islets (i.e., gastrinoma, insulinoma, glucagonoma, vasoactive intestinal peptide tumor pancreatic polypeptide–producing tumor), and the anterior pituitary gland (f.i., prolactinoma, somatotropinoma, corticotropinoma, nonfunctioning tumors). Other associated tumors include lipomas, angiofibromas, or those located in the adrenal gland cortex (rarely, in the adrenal medulla).

The MENIN gene responsible for type 1 MEN is located on chromosome 11 producing a tumor suppressor protein called menin. The MENIN gene is ubiquitously expressed and is localized to the nucleus of cells. Patients with type 1 MEN have a germline mutation in the MENIN gene and develop tumors if inactivation of the wild-type allele occurs.

Most tumors arise in the pituitary gland and pancreatic islet cells. Hyperparathyroidism cases are sporadic.

Type 2A MEN is defined by medullary thyroid carcinoma (MTC), pheochromocytoma (in about 50% of cases), and hyperparathyroidism caused by parathyroid gland hyperplasia (in about 20% of cases).

The gene responsible for type 2 MEN is a proto-oncogene called RET. In contrast to MENIN of type 1 MEN, RET is specifically expressed in neural crest–derived cells, such

as the C cells in the thyroid gland and the chromaffin cells in the adrenal gland. Whether RET is also expressed in the parathyroid glands or not, remains unknown, especially if considering the low rate of hyperparathyroidism in patients with type 2A MEN and the lack of hyperparathyroidism in type 2B MEN. RET encodes the tyrosine kinase RET protein subunit of a cell surface receptor. The activation of RET leads to hyperplasia of the target cells.

Familial MTC [redacted] is also recognized. Familial MTC is hereditary MTC without other associated endocrinopathies, although adrenomedullary hyperplasia secondary to a germline RET mutation may still be present remaining undiagnosed. Type 2B MEN is defined by medullary thyroid cancer and pheochromocytoma. Associated abnormalities include mucosal neuromas, medullated corneal nerve fibers, and a marfanoid habitus.

Subsequent secondary events can lead to tumor formation.

The tumor does not develop in all person with inherited predisposition, but only in persons who have *a viral infection*. The syndrom can only develop, if this inherited predisposition and the *viral infection* are together present, and these families are carriers of this viral agent. If they would not have this viral infection, the syndrome could not have been manifested.

Symptoms: The clinical picture depends on the glands involved and the hormones secreted. Hyperparathyroidism occurs with mild hypercalcemia and bone abnormalities. Gastrinoma causes diarrhea, abdominal pain due to peptic ulcer disease and esophagitis. Insulinoma causes hypoglycemia.

Glucagonoma can cause hyperglycemia. Rare cases of type 1 MEN are associated with erythema, anemia, diarrhea, or venous thrombosis. Pituitary tumors may cause headache, visual field defects, and other effects, depending on hormone production.

Pheochromocytomas cause hypertension, sweating, palpitations and tachycardia, headache, emotional lability, nausea, vomiting, polyuria and polydipsia.

Marfanoid phenotype develops in all patients affected. Phenotypic characteristics include a slender body build; long and thin extremities, abnormal laxity of joints.

The facies is characterized by enlarged thick lips as a result of embedded mucosal neuromas. Neuromas may be found on the surface of the lips, tongue, eyelids and cornea. Ganglioneuromas may occur at any level of the GI tract, causing constipation or diarrhea due to an abnormal control of intestinal motility.

Diagnosis: each of these tumors needs different laboratory studies, by biochemical evaluations, by genetic defect examinations. By biopsy with immune histological examinations. By examinations of the parathyroid glands, the pancreas, duodenum, stomach, the pituitary gland, etc. By CT, MRI, PETscan.

Treatment: by surgery, radiation, chemotherapy, symptomatically, etc.

Medical therapy concerning the specific endocrine syndromes.

RFR method: detects and may eliminate the pathogen microorganisms.

RFR method is a complementary treatment should only be used together with the medical therapy.

The most frequent resonances in case of MEN syndromes are: 307-308, 314-319, 343-347, 360-366, 372, 402-410, 412-413, 418-426, 427-438, 447-451, 452-453, 459-464, 469, 476-479, 493-495, 517-521, 525-527, 538, 543-545 kHz

There are different tumors developing in the multiple endocrine neoplasia group and also the *Human Papilloma Viruses,* present in these tumors differ respectively. Sometimes there are many different *human papilloma viral* infections present in certain tumors. Patients should be monitored concerning their *HPV* resonances in order to control the recurrence of the disease. After an initial follow-up visit, patients may be evaluated every 6 months, and later on yearly, only if they are asymptomatic. In these evaluations, patients should undergo physical examinations, biochemical examinations, 24-hour urine catecholamine,

metanephrine and vanillylmandelic acid, CEA level, calcitonin, and serum calcium testing. The treatment of patients with MEN syndromes has to be done in an Endocrinology center where a team of experts of clinical genetics, endocrinology and oncology work together.

26.37. Double Pituitary Adenoma

Double pituitary adenoma (DPA) may cause the overproduction of multiple hormones. Clinically nonfunctioning, PRL-producing adenomas are the most common types experienced at the autopsy. Although the pathogenesis of double adenomas is unclear, a genetic predisposition with abnormalities in DNA, *HPV, CMV* and *mycoplasmal* infections might be involved. If the preoperative MRI suggests double adenomas to be present, a careful surgical exploration is necessary in order to avoid hurting the other adenoma, especially concerning patients with functioning adenomas. Double adenomas may be caused also by an incidental occurrence of two monoclonal expansions of transformed anterior pituitary cells, or that of an other monoclonal proliferation within an adenoma originally of one cell type.

Symptoms: DPA patients have acromegalic symptoms caused by an enhanced GH-production, and have sometimes hyperprolactinemia and familial pituitary adenomas unrelated to multiple endocrine neoplasia type I (MEN-1).

Diagnosis: by MRI, PETscan.

Treatment: by surgery

RFR method can detect the infective agents.

Do not use RFR method as there might develop an edema around the necrotized adenoma.

The most frequent resonances are: 408-410 ***(CMV)*****; 442-444** ***(HPV)*****; 442-451** ***(Mycoplasma fermentans)*****; 530-536** ***(CMV)*** **kHz**

27. SOME IMPORTANT CONNATAL INFECTIONS

A prenatal infection is an infection caused by bacteria, viruses, fungi or less commonly by parasites that is passed from a mother to her baby during her pregnancy or childbirth. The embryo and fetus has either no immune function, or an immature one. The health of the fetus depends on its mother's immune function and on the absence of its mother's infection. It can get infected in two way, i.e. in a transplacental or a transcervical way. Several infective agents are capable of crossing the placenta and of causing infection in the embryo or fetus, i.e. of causing a connatal infection. Microorganisms producing minor illnesses of the mother are nevertheless often very dangerous for the development of the embryo or fetus. These infections can result in spontaneous abortion or in some major developmental disorders. In case of many an infection the fetus is more at risk at certain stages of pregnancy. Problems related to connatal infections are not always directly noticeable.

Infants can become infected also via the vagina of their mother during their birth. Some infective agents may be transmitted to the embryo or fetus in the uterus, during their birth or even shortly after it. Distinction is important because when transmission occurs primarily during or after birth, an intervention directly after birth may prevent the infant getting infected. During childbirth, the infant is exposed to maternal blood and body fluids and to the maternal genital tract, there being no placental barrier intervention anymore. Due to this, microorganisms transmitted by blood (f.i. *Hepatitis B Virus, HIV*) and other organisms, associated with sexually transmitted diseases (f.i. *Neisseria gonorrhoeae, Chlamydia trachomatis*, etc) are the most common ones present in infections of newborns. The most frequent and severe connatal infections are caused by *HIV and other Human T or B lymphocyte viruses, CMV, EBV, Measles, Rubella virus, Herpes Simplex virus, Toxoplasma gondii, Treponema pallidum, Borrelia B.sensu lato, Candida* and different parasites such as *Schistosoma*.

27.1. Connatal HIV Infection

The Human immunodeficiency virus is a lentivirus (a member of the retrovirus family) that can lead to acquired immunodeficiency syndromes (AIDS), a condition present in humans, in case of which, the immune system weakens more and more, leading to life-threatening opportunistic infections. Formerly this virus was named Human T-cell Lymphotropic Virus-3 (HTLV-3), lymphadenopathy-associated virus (LAV) and AIDS-associated retrovirus (ARV). The four major routes of transmission are the unprotected sexual intercourse, contaminated needles, breast milk and the transmission from an infected mother to her baby at its birth (vertical transmission). The transmission of the virus from the mother to child can occur in utero during the mother's pregnancy and during the child's birth. If not treated, the transmission rate between the mother and her baby is about 25 percent. However, if there does occur a combination of antiretroviral drug-treatment and Cesarian section, the risk can be reduced to one percent. Breast feeding also presents a risk of infection for the baby. HIV differs from many viruses in that it has a very high genetic variability and that there is no vaccine or healing cure for HIV or AIDS. Usually the treatment for HIV infection consists of administering a highly active antiretroviral therapy, or protease inhibitor-based HAART. Entry Inhibitors provide treatment options for patients infected with viruses being already resistant to common therapies. As the progress of AIDS in children, particularly in young infants, is more rapid and less predictable than in case of adults, a more aggressive treatment is necessary. HIV infection in young children arises

most commonly as a result of vertical transmission, i.e. of mother to child transmission (MTCT). The following factors have proved to increase the risk of MTCT: chronic chorioamnionitis, early rupture of membranes before delivery and pre-term birth.
Female babies are more likely to be early infected (transplacental/perinatal routes).
A co-existent *malarial* or *mycoplasmal* infection may increase the rate of HIV transmission, though this statement is not firmly established with study results. HIV-malaria and HIV-mycoplasma co-infected mothers are more prone to complications such as anaemia and have higher malaria parasitaemia and HIV viral-load counts. As some of HIV-positive mothers might slip through the checking system, one must take care and suspect a possible HIV-infection in children feeling unwell, as an undetected early infection might be transmitted transplacentally, perinatally, or later on through breastfeeding. Some cases can be symptomless even in later childhood, despite of being infected either in the fetal, or in the perinatal or infant states.
Symptoms: an impairment of cellular immune defences (characteristic to HIV infections) should be suspected in case of children who recurrently have severe bacterial infections, particularly invasive infections like meningitis, septicaemia and pneumonia, otitis media, urinary tract infections, sinusitis, and some rare infections like *Mycobacterium avium complex* pneumonia and *Pneumocystis carinii* pneumonia, recurrent *fungal* diseases such as thrush failing to respond to standard therapy, severe viral infections, caused f.i. by *HSV, VZV, shingles,* or have *CMV* retinitis or *mycoplasmal* pneumonia, splenomegaly and hepatomegaly, suffer from developmental delay, without any nutritional, metabolic, endocrine or other cause. Older children may show subtle diminution in intellectual skills such as concentration and memory, or schooling problems due to HIV-encephalopathy. Motor delay, abnormalities of muscle tone, spastic diplegia and oral motor dysfunction can be signs of HIV infection of the CNS. An opportunistic CNS infection can also be present in various forms.
Dermatological symptoms can be erythematous, papular rashes caused by HIV, Candidal dermatitis with marked erythema and/or purpura and bruising.
Sometimes HIV-induced thrombocytopenia, parotid enlargement, larged tonsils, oral aphthous ulcers, oral/pharyngeal plaques due to thrush and leukoplakia can develop.
Prevention is far more important concerning babies and infants, as the outcome of their healing is significantly worse compared to that of those infected in later life, despite the enormous advances done concerning the prophylaxis and treatment of-opportunistic-infections. Their mean survival rate counted from the diagnosis is about 10 years. About 15% of children suffer rapidly progressive and fatal diseases. In developing countries, the tragedy of HIV infection is underlined by the fact that failing a treatment 20% of babies born with HIV infection will die before their 4th birthday, while 50% will not live longer than about 9 years. The prevention of primary HIV infection concerning women and the prevention of unintended pregnancies among those living with HIV is most important, and so is the routine HIV-testing of pregnant women and the adequate drug therapy of those pregnant with established HIV and those who are found positive by screening.
Diagnosis: by HIV tests, f.i. using ELISA, Western blot and IFA methods
Treatment: in HIV-AIDS centers, f.i. with protease inhibitor-based HAART, nucleoside analogue reverse transcriptase inhibitors (NARTIs or NRTIs), plus either a protease inhibitor or a non-nucleoside reverse transcriptase inhibitor (NNRTI), though the latter has no healing effect on connatal HIV infections.
RFR method: can detect and eliminate HIV infection previous to the pregnancy of women. Examine the newborn baby after its birth if its mother was infected and eliminate all infectious agents!
The most frequent resonances of HIV virus-1 are: 312, 318-319, 324, 340, 364-366, 372, 384, 396-399, 401-403, 428, 450-455, 476, 487, 496, 508, 526, 556 kHz

The most frequent resonances of HIV virus-2 are: 290-291, 318-319, 349-350, 365, 372, 384, 396-397, 402-403-406, 428, 444-445, 450-454, 461-462, 509 kHz
See HTLV 1-6 in its special Chapter.
The frequency of the most frequent Mycoplasma fermentans species are: 440-451 kHz, as to the other Mycoplasma groups, see their special Chapter.
As to the frequencies of other pathogen microorganisms occuring, see their special Chapters.

27.2. Connatal Rubella Viral Infection

Sequelae of rubella viral infections of newborns include three distinct neurological syndromes, that is: the postinfectious encephalitis after an acute infection, certain neurological manifestations after connatal infections causing neurodegenerative disorders and the progressive rubella panencephalitis, that can follow either a connatal or a postnatal infection. Rubella viral infection during early pregnancy can lead to severe birth defects known as connatal rubella syndrome (CRS). The CRS can develop after an intrauterine infection caused by Rubella viruses and comprises cardiac, cerebral, ophthalmic and auditoral defects. It can also cause a low birth weight, neonatal thrombocytopenia, anaemia and hepatitis. The risk of major defects affecting the organogenesis is high in case of getting infected in the first trimester. CRS was the main reason for developing a vaccine to prevent rubella infections. Many mothers who contract rubella within their first critical trimester suffer either a miscarriage or get a still-born baby. If the baby survives the infection, it may be born with severe heart disorders, blindness, deafness, or other life threatening organ-disorders. Skin manifestations of CRS are named blueberry muffin lesions.
Prevention: by vaccination before pregnancy.
Treatment: by administering gammaglobulin antibodies.
RFR method: can detect the infection of the newborn baby if its mother was infected and can eliminate rubella and all the other infectious agents.
The most frequent resonances of rubella are: 332-334, 364, 372, 381, 387, 390, 402, 440, 450-451, 468, 520-530 kHz

27.3. Connatal Toxoplasmal Infection

In case of connatal toxoplasmosis the unborn fetus gets infected via the placenta. A toxoplasmal infection during pregnancy can cause a connatal infection of the child leading to its mental retardation and blindness. Hydrocephalus, intracranial calcification and retinochorioiditis are the most common manifestations of connatal toxoplasmosis. The effect of a prenatal treatment concerning these diseases is doubtful and the best method for preventing and controlling connatal toxoplasmosis is still unknown. The incidence of a transplacental infection during the first trimester of pregnancy is low (about 15%) but the developing disease of neonates is most severe. The incidence of a transplacental infection during the third trimester of pregnancy is high (about 65%), the infants are nevertheless usually asymptomatic at their birth. *Toxoplasma parasites* may also trigger the development of *schizophrenia, bipolar disorders, Parkinson's Disease, Tourette's syndrome and of attention deficit disorders*. Toxoplasma produces an enzyme named tyrosine hydroxylase, which, due to its effect on the dopamine production, may contribute to the development of these psychological disorders. These changes in the chemistry of the brain can significantly modify the patient's behavior. The role of dopamine played on the mood, the sociability, attention, motivation and on the patterns of sleeping is well known. Since already a long time schizophrenia is known to be associated with dopamine. Connatal toxoplasmosis can occasionally be also fatal.

A positive IgG antibody titer to Toxoplasma gondii indicates a previous exposure and immunity largely ensuring the safety of the unborn baby. The result of the test for toxoplasmosis got at the doctor's first prenatal visit can determine whether the woman was previously infected or not. If a woman gets infected with toxoplasma during her pregnancy, her baby will be particularly at risk of getting ill. A woman with no previous exposure should avoid handling raw meat, gardening and to get in contact with cats and their feces. Pregnant women with negative Toxoplasma antibody titer have to be tested monthly as the treatment of those first exposed to T. gondii during their pregnancy decreases dramatically the risk of passing the parasite to the fetus. As an invasive prenatal testing incurs some risk to the fetus, the postnatal or neonatal screening is preferable, excepting cases with noted fetal abnormalities.

Diagnosis: by antibody and PCR examinations.

Treatment: by administering spiramycin or other effective drugs. Since a baby's immune system does not fully develop at its first year of life, and the resilient cysts that form throughout the body are very difficult to eradicate with anti-protozoans, this infection can be very serious concerning babies.

RFR method: can detect toxoplasmal infections got before pregnacy.

RFR method: can detect the toxoplasmal infection of the baby.

The most frequent resonances of toxoplasmosis are: 394-397, 440-445, 460-463 kHz

27.4. Connatal CMV Infection

It is well known that Cytomegalovirus (CMV) is a member of Betaherpesvirinae of the subfamily Herpesviridae. Other Betaherpesvirinae species include f.i. the Human Herpes Virus-6 (HHV6) and HHV7, which share some common clinical characteristics with CMV. CMV is a cytolytic virus causing a cytopathic effect in vitro and in vivo. The pathologic hallmark of CMV infection is an enlarged cell with viral inclusion bodies. The cell-mediated immunity is considered to be the most important factor in the controlling of CMV infection. Patients deficient in cell-mediated immunity are at a greatest risk for getting a CMV disease. CMV-specific $CD4^+$ and $CD8^+$ lymphocytes play an important role in the immune protection acting after a primary infection or after the reactivation of a latent disease. Recent investigations into the molecular biology of CMV have revealed the presence of many viral gene products, which appear to modulate the host's inflammatory and immune responses. Several CMV genes interfere with the host's normal antigen processing and the generation of cell-mediated immune responses.

Most people get infected with CMV at some time of their life. CMV mononucleosis is an acute or subacute febrile illness with relative or absolute lymphocytosis and many atypical lymphocytes. CMV patients often complain of headache, back and abdominal pain, sore throat and rubelle-like rasches. Like other herpes viruses, CMV also establishes a chronic latent infection in the host. In case of immunosuppression secondary to drugs, or an intercurrent infection (f.i. HIV), CMV may get reactivated. There does frequently develop a clinically significant CMV disease among patients immunocompromised by *HIV and other HTLVs*, in case of *mycoplasmal* infection, solid-organ transplantation, bone-marrow transplantation, chemotherapy and/or radiation therapy. The symptomatic disease of immunocompromised individuals can affect almost every organ of their body, resulting in fever of unknown origin, interstitialis pneumonia, hepatitis, encephalitis, myelitis, colitis, uveitis, retinitis and neuropathy.

The intrauterine transmission of the disease from a mother with acute CMV infection during pregnancy is a significant cause of connatal neurological abnormalities and deafness of their newborns. The CMV-related immune state of the woman is important in determining the risk of placental infection and that of a subsequent symptomatic disease of her fetus or newborn. A connatal symptomatic CMV disease does less likely to develop among pregnant women with a pre-existing immune response to CMV, than in case of

CMV-naive women. One among ten cases of acute CMV infection during pregnancy is estimated to result in a connatal CMV disease.
A risk factor of CMV mononucleosis is the transfusion of multiple units of blood, the clinical sign of this way of infection can be a posttransfusion fever.
The connatal infection with CMV can be an important cause of hearing, cognitive and motor impairments of newborns. Connatal CMV infection can cause the symptoms of hepatosplenomegaly with hepatitis and cirrhosis, pupura and encephalitis with microcephaly and microgyria. Despite of the common occurrence of viruria and despite of bearing cells in the kidney, no progressive renal disease does develop.
Connatal CMV infection is one of the TORCH infections (Toxoplasmosis, Other infections (including syphilis and Mycoplasma fermentans), Rubella, CMV and Herpes Simplex Virus), which all carry the risk of a significant symptomatic disease and some developmental defects in newborns. The clinical signs of a connatal cytomegalic inclusion disease includes jaundice, splenomegaly, thrombocytopenia, intrauterine growth retardation, microcephaly and retinitis.
Connatal CMV infection connected with coinfections: Concerning children with connatal *HIV* infection, the co-infection with CMV appears to accelerate the progression of HIV disease and of HIV-associated neurological diseases. Accumulating evidence suggests that a CMV infection in connection with a *mycoplasmal* infection can be a cofactor in the pathogenesis of atherosclerosis. Moreover, the phenomena of posttransplant vascular sclerosis and postangioplasty restenosis appear to be CMV-induced lesions.
Vaccination: the experience got with rubella stresses the necessity of vaccination in order to prevent connatal CMV infections, espite the fact, that vaccines can cause local reactions including pain, erythema, induration and warmth, as well as systemic reactions, such as chills, arthralgia and myalgia.
Diagnosis: by antibody examinations or/and CMV-DNA examinations with PCR methods concerning all the different cell lineages and organ systems in the body.
Traditionally, CMV antibody tests were performed using complement fixation and showed peak viral titers 4-7 weeks after infection. Multiple tests for CMV antibody are now available. Some tests are sensitive enough to detect anti-CMV IgM antibody early in the course of the illness and during CMV reactivation. Reactivation of the virus is not uncommon, sometimes occurring with viremia and a positive IgM in the presence of IgG antibody. This is usually observed during intercurrent infections or in case of stress. The clinical significance, time course and natural history of reactivation among immunocompetent patients is in respect to viruses unknown.
Treatment: administering oral ganciclovir may be used as prophylaxis of CMV retinitis or other local processes. It should not be used for treatment. In case of severe inflammations, the treatment with corticosteroids may be necessary, though corticosteroids can increase the number and spreading of the viruses.
Prevention: by vaccination.
RFR method: can detect and may eliminate CMVs prior to the pregnancy of the woman. Examine the newborn after its birth, if its mother is/had been infected!
The most frequent resonances are: 304-306, 349-350, 408-411, 533-535, 547-550 kHz

27.5. Connatal Borreliosis

Both, syphilis and lyme borreliosis are caused by spirochetes leading to multisystem diseases worldwide. The connatal borreliosis symptoms seem to be somehow similar to, but usually milder than the connatal syndromes experienced in case of syphilis, leptospirosis and relapsing fever.
Its symptoms develop mostly if the mother got infected during her pregnancy. The symptoms can be low birth weight (due to a chronic placental insufficiency), hypotonia, macrocephaly (hydrocephaly) or microcephaly, supraventricular tachycardia, chronic

cardiologic damages, hyperbilirubinemia, recurrent rashes and lymphadenopathy of the affected infants. Connatal neurological disorders caused by borreliosis may also develop. From available evidence, it seems reasonable to conclude that no distinct pattern of teratogenicity of a connatal borrelia infection has been as yet described, and that there does not exist any connatal borreliosis syndrome, analogous to connatal syphilis. The connatal neurological symptoms can sometimes be caused by various peripherial and central neural damages.

Connatal borrelosis can form two groups:

1. Being only a borrelial infection,
2. Borrelial infection with coinfections.

The most frequently occuring severe coinfections in case of connatal borreliosis are caused by *Mycoplasma fermentans, Mycoplasma penetrans, Mycoplasma pneumoniae, Mycoplasma genitalium and other mycoplasmas*, as well as by *EBV, CMV* and *Human T-cell and B-cell Lymphotropic Viruses*. The infections belonging to this second group can cause very serious and dangerous processes and are, moreover, mainly seronegative borrelial diseases. Miscarriage, stillbirth, neonatal death (rarely) and connatal borrelia infections have all been described in the medical literature. Further researches are still necessary to find the possible teratogenic effects that might occur if the spirochetes reach the fetus during the period of its organogenesis.

Gestationally acquired Lyme borreliosis can cause fetal death, hydrocephalus, cardiovascular anomalies, neonatal respiratory distress syndrome, hyperbilirubinemia, intrauterine growth retardation, cortical blindness, Sudden Infant's Death Syndrome, spongiform encephalopathy and maternal toxemia of pregnancy.

Diagnosis: by the history of erythema migrans and other specific clinical symptoms of the mother, positive serology (ELISA, Immunoblot, etc.) and PCR examinations of the mother. The serological (immunoblot) testing for borreliosis of the infant may initially be negative, but can, later on, become positive. PCR examinations are more specific but less sensitive.

Treatment: by administering effective antibiotics in adequate doses and duration to mother and infant.

RFR method: can detect and may eliminate the borrelial infection previous to the woman's pregnancy.

Examine the newborn baby after its birth if its mother was infected!

The most frequent resonances of Borreliosis are: 302, 378-387 kHz

Other frequencies of Lyme disease can be: 301, 309-319, 327, 341-344, 352, 371-376, 387, 401-407, 412, 420-422, 429, 441-442, 452, 496, 510-511, 544-548, 556, 565 kHz

The most frequent coinfective agents are Mycoplasmas: 307-308, 321-324, 342-350, 442-451, 493-495 kHz

The resonant frequencies of EBV are: 372-383, 518-519 kHz

The resonant frequencies of Cytomegalovirus are: 408-411 kHz

The resonant frequencies of HTLV are: 297-299, 307, 311-315, 320-340, 354, 359, 365-367, 370-376, 382-383, 397-400, 406, 416, 428-439, 453-455, 474-476, 480-482, 484, 487-490, 493-504, 523-530, 540-545, 570-578 kHz

27.6. Connatal Herpes Simplex Infection

The intrauterine herpes simplex infections 1 and 2 are rather rare, though several different coinfections caused f.i. by *mycoplasma, HTLV, malaria* may promote them, and, later on, when passing through the birth canal, birth-acquired herpes is the most common herpes infection of newborns.

Intrauterine herpes infections can cause inflammation of the retina, i.e. chorioretinitis, brain damages i.e. encephalitis, skin lesions and jaundice of the fetus.

The birth-acquired herpes can cause **localized** (i.e. only in one locus of the body) or, in case of certain coinfections, **systemic** diseases as well. Localized skin infections of infants

consist of small fluid-filled blisters that rupture, crust over and finally heal, leaving sometimes a mild scar. Birth-acquired herpes infection can lead to encephalitis, an inflammation of the brain which can result in seizures and later on even in brain and nervous system (neurologic) disorders. If untreated, it may lead to death.

Immune suppressed patients (coinfected with *mycoplasma, HIV or other HTLVs*) may suffer from its most dangerous type, the disseminated herpes infection. In case of these combined type of infections, herpes virus can affect different internal organs, including the liver, the lungs, kidneys and the brain. There may or may not develop vesicles on the skin. This type of infection is often fatal.

The symptoms of a disseminated herpes infection can be bleeding, breathing difficulties with cyanosis, tachypnea, short periods without breathing (apneic episodes), which may end in coma, enlarged liver and spleen, jaundice, different kidney failures, hypotermia, poor feeding, letargy, seizures, respiratory distress, anxiety and shock. Concerning infants the outcome of systemic herpes infections or of herpes encephalitis is often rather bad, despite of antiviral medications even if given in time.

The symptoms in case of its coinfection with malaria are hepatomegaly, icterus, and edema, often observed especially in case of *Malaria falciparum* infections. This type of infection is frequently present in tropical countries.

The symptoms in case of its coinfection with mycoplasmas or HTLVs are those of encephalitis and permanent central neurological damages leading to coma or prenatal death.

Diagnosis: as babies with connatal herpes may not have the characteristic blisters of the disease, it is difficult to diagnose. More over, many symptoms of herpes infections resemble other diseases or disorders. Mother and baby must usually be tested simultaneously if herpes infection is suspected: making urine test, blood test, CT scan or MRI scan of the head of the baby.

Treatment: infants with connatal herpes are usually treated with antiviral medications given intravenously over a period of several weeks. The most commonly used treatment for connatal herpes is i.v. acyclovir. Other treatment may still be necessary for the various symptoms of herpes. Herpes infection with coinfections must be treated in special medical centres.

Prevention: safer sexual practices can help to prevent the mother from getting genital herpes. Mothers who are not infected with herpes cannot pass the herpes virus to the fetus during delivery. Patients with "cold sores" (herpes labialis) should avoid contacting newborn infants. If the person with a cold sore is a caregiver, force them to wear a surgical mask and to wash their hands carefully before coming into contact with the infant.

RFR method should be used previous of getting pregnant in order to detect and eliminate infections caused by Herpes Simplex Viruses, HIV and other HTLVs and Malaria plasmodium.

Examine the newborn baby after its birth in case its mother was infected! Eliminate all infectious agents!

The resonant frequencies of Herpes Simplex Virus-1 are: 290-294, 307-309, 328, 336-339, 344-346, 350, 398-401, 413, 420, 458, 483-486, 490, 533 kHz

The resonant frequencies of Herpes Simplex Virus-2 are: 318, 352-365, 372, 383, 396, 402, 450, 463, 476 kHz

The most frequent resonances of Mycoplasma fermentans are: 442-451, 493-495 kHz

The most frequent resonances of HIV-1 are: 312, 318-319, 324, 340, 364-366, 372, 384, 396-399, 401-403, 428, 450-455, 476, 487, 496, 508, 526, 556 kHz

The most frequent resonances of HIV-2 are: 290-291, 318-319, 349-350, 365, 372, 384, 396-397, 402-403-406, 428, 444-445, 450-454, 461-462, 509 kHz

See the frequencies of the other HTLVs and Malaria in their special Chapters.

27.7. Connatal American Trypanosomiasis

The American trypanosomiasis, also known as Chagas disease, is caused by infection with the protozoan parasite *Trypanosoma cruzi*, a member of the Trypanosomatidae family, belonging to a special section called Stercoraria. The infective forms of *T. cruzi* are contained in the feces of their insect vectors and gain entry into their mammalian hosts through contamination. The spraying of insecticides against cone-nosed bugs (Triatoma infestans) and the screening of blood donors decreased the human incidence of *Trypanosoma cruzi* infections in Argentina, Bolivia, Brazil, Chile, Paraguay and Uruguay, nevertheless a relative emergency of vertical transmission is still threatening. The transmission of this parasite can occur transplacentally, or via blood transfusion and also by organ transplantation. The rate of the transplacental transmission (from mothers, suffering chronic *T cruzi* infections to their newborns) is about 2-10%. Intrauterine infections can cause spontaneous abortion and premature delivery. In its acute form, connatal Chagas disease often resembles an otherwise acquired type. It has its begin usually at the child-birth or a few months later. Infected infants often have a low birth weight, hepatosplenomegaly, jaundice, anemia, fever, edema, meningoencephalitis with convulsions, hypotonia, hyporeflexia, lympadenopathy and tremors. Some develop metastatic hemorrhagic chagomas in the skin or the mucous membranes. Intracranial calcifications and ocular lesions have also been described. Cardiac involvement is rare, in case of which the heart becomes dilated with a thin muscular wall, especially in the right atrium. An aneurysm can be present at the sometimes rupturing apex of the left ventricle. Megaesophagus causes dysphagia and even aspiration. Denervation and fibrosis occurs usually in the submucosal (Meissner) and myenteric (Auerbach) plexuses. Dysfunction of peristalsis may lead to the hindrance of transit, extreme dilatation and hypertrophy.If the histiocytes and other inflammatory host cells ingest the parasites, they get transformed into amastigotes. These forms of parasites can multiply within the cells of virtually every organ and tissue.

Following their local multiplication, the pathogens assume a trypomastigote form, invade the bloodstream, carrying the infection to all parts of the body. The cells of the reticuloendothelial system; the cardiac, skeletal and smooth muscles and the neural cells are preferentially infected. The inflammatory reaction of the host is characterized by a local accumulation of polymorphonuclear leukocytes, lymphocytes and plasma cells and can cause marked cellular destructions. *T. cruzi* infections can be present throughout the whole life.

Neurotrypanosomiasis develops more commonly in case of intrauterin infections, in young infants and among immunosuppressed patients. Clinical manifestations include nuchal rigidity, convulsions, paralysis and coma. Encephalitis develops in immunosuppressed patients. Death comes in about 50% of these neurological cases.

Connatal neurotrypanosomiasis leads usually to death within the first few weeks of life. Those surviving have severe neurologic sequelae, mental deficiency and disability to learn. Meningoencephalitis can also occur.

Prevention: by screening of pregnant women and newborns in T. cruzi-endemic countries.

Diagnosis: by serologic tests (IFA, indirect hemagglutination, Radioimmunoprecipitation assay and ELISA)

Criteria of diagnosing connatal cases are:

1) the baby must be born from a mother with positive serology for *T. cruzi,*
2) parasites have to be identified at birth,
3) parasites or specific antibodies of not maternal origin are detected later after birth, providing that previous blood transfusion and vectorial contamination can be discarded.

Symptomatic cases (i.e. connatal Chagas disease cases) are frequently premature infants, displaying low birth-weight and hepato-splenomegaly. Meningoencephalitis and

myocarditis can be observed in cases of co-infection with *HIV* or other *HTLVs*. There is no specific clinical marker of connatal Chagas disease.
Treatment: by prospectively administering effective, nontoxic drugs to pregnant women. Once the disease is diagnosed, the appropriate course of treatment has to be Benznidazole. Once the disease has proceeded to a chronic form, antibiotics are effective in treatment. For this disease there is no vaccine available. The Chagas disease treatment is as difficult as that of borreliosis.
RFR method can detect Trypanosoma cruzi infection before pregnancy.
RFR method can detect Trypanosoma cruzi infection of the baby.
The most frequent resonances of Trypanosomiasis are: 460-466 kHz, though it has several different resonances as well.
The most effective therapy is a combinated treatment, administering antibiotics and using RFR-method.

27.8. Connatal Syphilis

Connatal syphilis is a multisystem infection caused by *Treponema pallidum pallidum* transmitted to the fetus via the placenta.
Symptoms: Its *early signs* are characteristic skin lesions, lymphadenopathy, hepatosplenomegaly, failure to thrive, blood-stained nasal discharge, perioral fissures, meningitis, chorioiditis, hydrocephalus, seizures, mental retardation, osteochondritis and pseudoparalysis.
Its *later signs* are gummatous ulcers, periosteal lesions, paresis, tabes, optic atrophy, interstitial keratitis, sensorineural deafness and dental deformities. Untreated syphilis results in a high risk of a bad outcome of pregnancy, including Mulberry molars in the fetus. Syphilis can cause miscarriage, premature birth, stillbirth and the death of newborn babies. Some infants with connatal syphilis have symptoms already at their birth, but most of them develop the symptoms later. Untreated babies can have deformities, delay in development, seizures and many other problems such as rash, fever, swollen liver and spleen, anemia and jaundice. The sores of infected babies are infectious. The illness sometimes develops imperceptibly in infants, but later on, the **late-stage syphilis symptoms** will develop, including the damages of their bones, teeth, eyes, ears and brain. Connatal syphilis usually does not show up until after 2 years of the child's life and causes gummatous ulcers involving the nose, the septum, and the hard palate and causes periosteal lesions resulting in saber shins and bossing of the frontal and parietal bones.
Connatal neurosyphilis is usually asymptomatic, though juvenile paresis and tabes can develop. Optic atrophy, sometimes leading to blindness, may occur. Interstitial keratitis, the most common eye lesion, frequently recurs, often resulting in corneal scarring. Sensorineural deafness, which is often progressive, may appear at any age. Hutchinson's incisors, mulberry molars and the maldevelopment of the maxilla resulting in "bulldog" face are characteristic infrequent sequelae.
Serious coinfections can occur caused by *HIV, other HTLVs* and *Mycoplasma fermentans*.
Diagnosis: is usually suspected and based on maternal serologic testing, routinely performed early in pregnancy, in the 3rd trimester of pregnancy and at delivery. Neonates of mothers showing positive tests should undergo a thorough examination, darkfield microscopy of any skin and mucosal lesions and a quantitative nontreponemal serum test. The placenta and the umbilical cord should be analyzed using darkfield microscopy or fluorescent antibody staining. Infants with clinical signs of illness or suggestive serologic test results should suffer lumbar puncture with cerebrospinal fluid analysis for cell count, VDRL and proteins, CBC, liver function tests and long-bone x-rays. Diagnosis can be confirmed by microscopic visualization of spirochetes in samples from the neonate or the placenta. Hutchinson's triad of interstitial keratitis, Hutchinson's incisors, and 8th cranial nerve deafness is diagnostic. The standard STS and T. pallidum immobilization tests can

be negative, but the fluorescent treponemal antibody absorption test is usually positive. The diagnosis should be considered in cases of unexplained deafness, progressive intellectual deterioration and keratitis.

Treatment: in case of a new infection by administering penicillin G. For the later stages of syphilis or neurosyphilis, the appropriate regimen for nonpregnant patients should be followed. After a therapy like this, a severe Jarisch-Herxheimer reaction does occur occasionally, leading to spontaneous abortion. A treatment administered at a later state of pregnancy can eliminate the infection of the fetus, but not certain signs of syphilis appearing at birth.

Prevention: pregnant women should be routinely tested for syphilis and retested if they acquire other sexually transmitted diseases during pregnancy.

RFR method can detect this bacterial infection before pregnancy.

RFR method can detect this bacterial infection of the baby.

The most frequent resonances of Treponema pallidum pallidum are: 337-340, 345-350, 458-452 kHz

An effective RFR method can cause a strong Jarisch-Herxheimer reaction.

The most effective treatment is a medical therapy together with RFR method.

27.9. Connatal Hepatitis B and Hepatitis C Infections

Hepatitis B virus (HBV) is a double-stranded DNA virus of the Hepadnaviridae family. The most important mode of transmission of *HBV* from mother to child is the vertical way. Connatal *HBV* infections are seldom recognized (as proved by the persistence of HBsAg) and the affected infants suffer more likely from chronic infections compared to adults contracting the infection. *HBV* can be found in highest concentrations in the blood, and in lower concentrations in semen, vaginal secretions, and in wound exudates. Less than 1% of pregnant women are HBsAg positive in Western countries compared to the about 25% in countries of Africa and Asia. About one half of acute *HBV* infections are symptomatic in adults with 1% of cases resulting in acute liver failure and death. Acutely infected individuals develop clinically apparent acute hepatitis with loss of appetite, nausea, vomiting, fever, abdominal pain and jaundice. 10-20% of women seropositive for HBsAg transmit the virus to their neonates due to the absence of immunoprophylaxis. In case of women who are seropositive for both HBsAg and HBeAg the chance of a vertical transmission is approximately 90%. A vertical transmission occurs in up to 10% of neonates if the infection occurs in the first trimester, while in about 80-90% of neonates if it occurs in the third trimester of their mothers pregnancy, ill with acute Hepatitis B viral infection. The transmission rate can be significantly reduced by active immunization with Hepatitis B vaccine combined with passive immunization using Hepatitis B immunoglobulin within 12 hours of child-birth.

HBV infection does not appear to be teratogenic. However, there is a higher incidence of low birth weight among infants born to mothers with acute HBV infection during pregnancy. A short study discussing acute maternal hepatitis cases (type B or nontype B) does not mention any effect on the incidence of connatal malformation, stillbirth, abortion and intrauterine malnutrition. However, acute hepatitis increases the incidence of premature child-birth.

Caused by *HIV, other HTLVs* and *Mycoplasma fermentans* **serious coinfections** are apt to occur.

If *Hepatitis C Viruses (HCV)* are detectable by PCR in the mother, a vertical transmission can occur in approximately 6% of cases. The risk increases to 23% if these women are *HIV* positive as well. Infected infants become viremic and are at risk of getting chronic hepatitis. Interferon therapy may be used postpartum. Elective caesarean section, formula

testing and administration of immune globulin do not reduce the risk of vertical transmission.

Diagnosis: in the absence of IgM anti-HBc a positive HBsAg is indicative of chronic infections. But if it is present, a perinatal immunization or HBV prevention program has to be followed to ensure a proper case management of the mother and an appropriate postexposure immunization of her infant being at-risk.

The **Treatment** of acute HBV infections is but supporting. Persons with chronic HBV infections should be referred to health-care professionals with great experience in the treatment of Hepatitis B. A course of HBV vaccine into her deltoid should be given in case of the pregnant woman's exposure to a person with acute HBV as a result of sexual contact within 14 days following the most recent sexual contact.

There are monovalent Hepatitis B vaccines available for preexposure immunization and postexposure prophylaxis. All sexual partners of HBsAg-positive women identified through prenatal screening should be vaccinated.

RFR method can detect HBV infection before pregnacy and eliminate it.

RFR method is not a method of diagnosing hepatitis. During her pregnancy the usage of RFR method concerning the mother is not advisable.

RFR method can detect HBV infection of the baby and eliminate it.

The most frequent resonances of Hepatitis B Virus are: 293, 341, 384, 392, 398, 414-420, 444-450, 454, 488 kHz

27.10. Connatal Listeriosis

Listeriosis is a bacterial infection caused by a gram-positive, motile bacterium, *Listeria monocytogenes*. Listeriosis does only occur relatively rarely and affects primarily pregnant women, their newborn infants, elderly and immunocompromised patients. It is one of the most virulent foodborne pathogens, 20 percent of the clinical infections result in death. Listeria monocytogenes has many subtypes with different antibiotic resistance. Listeria is a common veterinary pathogen as well, can cause abortion and encephalitis of sheep and cattle. It can be isolated from soil, water and decaying vegetation. People get listeriosis via infected food (salads contaminated with infected animal faeces, undercooked meats, unpasteurised milk, soft cheese and pates.)

Listeria has been isolated from prepared meat (f.i. hot dogs, deli meat), dairy products, unwashed raw vegetables and seafood. Soft cheese and unpasteurized milk are the most frequently incriminated dairy products.

The most common clinical manifestation of this illness is diarrhea. A mild fever, nausea, vomiting resembling a gastrointestinal illness can also come about. Its more serious manifestations, f.i. bacteremia and meningitis affect only high risk patients.

In case of pregnant women the ingestion of Listeria can cause nausea, vomiting, diarrhea, fever, malaise, back pain and headache. Pregnant women can carry Listeria in their gastrointestinal tract or vagina asymptomatically aswell. The manifestations of listeriosis in case of maternal diseases are septicemia, meningitis or meningoencephalitis, encephalitis, corneal ulcer, pneumonia and intrauterine or cervical infections of pregnant women, which two latter may result in spontaneous abortion (2nd/3rd trimester) or stillbirth. Listeria can get into the placenta and might, owing to the impaired cell-mediated immunity during pregnancy, infect it. Maternal infection with Listeria can cause chorioamnionitis, premature labor, spontaneous abortion, or stillbirth. Fetal infections can occur via transplacental transmission. A transplacental infection of the fetus is more usual than an ascending one. An **early infection** in pregnancy usually results in miscarriage, whereafter, still birth (20%) and prematurity are typical. A vertical transmission can also come about from mother to infant via the passage through an infected birth canal or when ascending through the ruptured amniotic membranes.

An early beginning neonatal listeriosis is usually associated with sepsis or meningitis. Among infants the infection presents itself as granulomatosis infantiseptica (baby and placenta are covered with miliary granulomata) or as pneumonia without granulomata. Meningitis can also be caused. The mortality of affected infants is above 30%. The surviving neonates suffering from fetomaternal Listeriosis may have granulomatosis infantiseptica and pyogenic granulomas distributed all over the body and this illness can cause physical and mental retardation of the babies. A late-beginning neonatal listeriosis frequently causes a purulent meningitis. Listeriosis often involves many organs with microabscesses or granulomas. A disseminated rash with small, pale, granulomatous nodules is histologically characteristic of granulomatosis infantisepticum. Infants past the neonatal period suffering from Listeria infection have an underlying immunodeficiency or are immunocompromised. Older children with Listeria infections frequently develop meningitis. A CNS infection may manifest itself also as a meningoencephalitis or as an abscess. Endocarditis can also come about. A localized infection may manifest itself as septic arthritis, osteomyelitis, and, rarely, as pneumonia or bronchopneumonia.
Influenza-like early symptoms, including persistent fever, usually precede the onset of the aforementioned disorders. Gastrointestinal symptoms, such as nausea, vomiting and diarrhea may precede the more serious forms of listeriosis or may remain the only symptoms.
Prevention: is the safe handling, cooking and consumption of food, including washing raw vegetables and cooking raw food thoroughly, as well as reheating leftover or ready-to-eat foods like hot dogs until steaming hot.
Another aspect of prevention is advising high-risk groups such as pregnant women and immunocompromised patients to avoid unpasteurised pâtés and soft cheese like feta, Camembert cheese and bleu cheese
Dangereous coinfections caused by *HIV* and *other HTLVs* or *Mycoplasmas* can come about.
Diagnosis: by detecting the Listeria monocytogenes bacterium in the blood, meconium and the cerebrospinal fluid cultures, and by placental examinations.
Treatment: by administering effective antibiotics.
RFR method can detect Listeria monocytogenes infection before pregnacy.
RFR method can detect Listeria monocytogenes infection of the baby as well.
The most frequent resonances of listeriosis are: 320-324, 365-370, 384, 396, 482, 502-504, 553 kHz

27.11. Connatal Parvovirus B19 Infection

The B19 virus is generally referred to as Parvovirus B19, or sometimes, Erythrovirus B19. Erythroviruses belong to the Parvoviridae family of small DNA viruses. These viruses primarily spread by infected respiratory droplets and via blood-borne transmission. B19 virus causes a childhood rash called Fifth disease, a very common viral infection of children, that, if symptomatic, is named Erythema infectiosum (Slapped cheek syndrome). This virus infects and lyses human erythroblasts. Only 5% of children under 5 years of age get infected, but if they are already about 5 years old, 20-40% of them can get infected. 50-75% of adults are seropositive. Seronegative pregant women can become infected and the infection can infect the fetus in utero. 60-70% of women of child-bearing age are susceptible to infection, but only about 1% of them get infected. Perinatal and intrapartum infections are very rare.
Infections in utero do but seldom result in fetal death, nonimmune fetal hydrops, birth defects of the eyes, different CNS lesions and premature child-birth. B19 viral infection may cause seronegative arthritis of pregnants, which can usually easily be controlled with analgesics. Possibly about 15% of all new cases of arthritis is caused by parvoviruses. This form of arthritis does not progress to other forms of arthritis. Its joint symptoms typically

last as long as 1-3 weeks, in 10-20% of affected persons they last for weeks and months. The most important sign of a parvoviral infection concerning pregnant women is associated with **hydrops fetalis** caused by severe **fetal anemia**, leading sometimes to miscarriage or stillbirth. The risk of fetal loss is about 10% if the infection occurs within the 14th-20th week of pregnancy. The risk to the fetus will be reduced by the correct diagnosis of the mother's anemia and by blood transfusions.

Most dangereous coinfections can occur with *HIV, other HTLV*s and *Mycoplasma fermentans*. In case of these infections a severe generalized pathologic process can develop.

Aplastic crisis: although in case of most patients the erythropoiesis gets hindered owing to a parvoviral infection, it is most dangerous in patients suffering from sickle cell anemia or hereditary spherocytosis, being therefore heavily dependent on erythropoiesis due to the reduced lifespan of the red bloodcells. This state leads to reticulocytopenia or aplastic crisis.

Diagnosis: by serology

Treatment: by blood transfusion.

RFR method can detect and eliminate B19 viral infection before pregnancy.

Examine the newborn baby after its birth if its mother was infected and eliminate all infectious agents, such as B19 virus as well.

The most frequent resonances of B19 virus are: 412-416 kHz

If the baby has a *HTLV* or *Mycoplasmal* infection as well, the first step to take is to eliminate these infections and then to eliminate the parvovirus.

27.12. Connatal Mycoplasma Fermentans Infection

Mycoplasma fermentans, a human pathogen Mycoplasma species is suspected to play a role as coinfection in the progression of autoimmune diseases, such as diabetes, rheumatoid arthritis, fibromyalgia, SLE, Multiple Sclerosis, ALS, Hashimoto's thyroiditis, and in several different cancer processes together with HPVs. Instead of causing an acute transformation, it gives rise to a multistage process promoting and enhancing a malignant cell transformation with a long latency period.

Mycoplasma fermentans and *Mycoplasma penetrans* infection can be found usually in AIDS patients. The Mycoplasma species owns a capacity to invade cells, tissues and the blood, causing systemic infections of the immunsystem and the CNS. Mycoplasma infection can trigger an inflammatory cytokine production, can decrease the normal immune function, and activate a pathological autoimmune response.

Mycoplasma species are capable of **crossing the placenta** and of causing infection in the embryo or fetus, i.e a connatal infection. Mycoplasma species, which often produce minor illnesses in the mother are very dangerous concerning a developing embryo or fetus. Mycoplasmal infections can result in spontaneous abortion and in major developmental disorders and/or can produce damages in numerous organs of newborns.

Diagnosis: by PCR or by immunehistochemical methods. See the special Chapters.

Treatment: If detected in time, diseases associated with invasive mycoplasmal infections are treatable with high-dose antibiotics, which do not damage the fetus. *Mycoplasma fermentans* gets easily relatively or absolutely antibiotic resistant.

RFR method: can detect *Mycoplasma fermentans* infection before pregnancy and can eliminate it.

RFR method: can detect *Mycoplasma fermentans* infection of the baby after its birth and can eliminate it.

The most frequent resonances of Mycoplasma fermentans are: 442-451, 493-495 kHz

28. BIOLOGICAL WEAPON AND THE RFR METHOD

The Convention on the Prohibition of the Development, Production and Stockpiling of microbiologic agents and toxin weapons and on their destruction (usually referred to as the Biological Weapons Convention, abbreviated: BWC, or Biological and Toxin Weapons Convention, abbreviated BTWC) was the first multilateral disarmament treaty banning the production of an entire category of weapons. Biological warfare (BW) means the use of pathogenic infectious microorganisms or other disease-causing agents as biological weapons or bioweapons. Subsequent Review Conferences have reaffirmed that relevant to the Convention, the general purpose criterion encompasses all future scientific and technological developments. It is not the objects themselves, but rather certain purposes for which they can be employed are prohibited. The Biological Weapons Convention (BWC) in 1972 is signed by more than 100 countries. The BWC remains in force and prohibits the storage, stockpiling and usage of these weapons. Biological weapons are intended to kill, incapacitate, or seriously impede individuals as well as entire cities and places. It may also be defined as a material or defense against such employment. The BW is a military technique that can be used by nation-states or non-national groups. In the latter case, or if a nation-state uses it in a clandestine way, it may also be considered as a bioterrorism.

Biological warfare agents can be bacteria, mycoplasma, viruses, rickettsiae and fungi. Biological weapons are distinguished by being living organisms, reproducing within their host victims, who then become contagious with a multiplier weakening, deadly effect. Toxins, in contrast, do not reproduce themselves in the victim and, needing but the briefest incubation period; kill within a few hours. Ideal characteristics of biological weapons targeting humans are high infectivity, high potency, non-availability of vaccines and their being delivered as an aerosol.

The biological agents used as biological weapons can often be created quickly and easily. The primary difficulty is not the production of the biological agent but their delivery in an effective form to a vulnerable target.

Anthrax, for example, is for several reasons considered as an effective agent. First of all, it forms hardy spores proved to be perfect for dispersing in aerosols. Secondly, lung infections of anthrax do not usually cause secondary infections in other people. Thus, the effect of the agent is usually confined to its target. A pneumonic anthrax infection starts with ordinary "cold" symptoms and quickly becomes lethal, with a fatality rate of 90% or even higher. Finally, the personnel can be protected with suitable antibiotics.

Diseases considered for weaponization, or known to be already used as weapons include *Anthrax, Ebola, Marburg virus infection, Plague, Cholera, Mycoplasmal infection, Tularemia, Brucellosis, Q fever, Machupo, Coccidioidomycosis, Glanders, Melioidosis, Shigellosis, Rocky Mountain spotted fever, Typhus, Psittacosis, Yellow fever, Japanese B encephalitis, Rift Valley fever, Smallpox* and might be even others. There could be developed antibiotics resistant bacterium species for weaponisation, and the virulence of these bacteria can be increased by genmanipulation.

All manipulated bacteria species have DNA stuctures, and, in this way, have resonant frequencies. These bacteria can be recognised by their frequencies and the RFR method is able to eliminate them.

Detect and eliminate these genmanipulated species!

29. GENETIC DISORDERS ASSOCIATED WITH INFECTIONS NOT MENTIONED AS YET

29.1. Albright Syndrome (McCune–Albright Syndrome)

Albright Syndrome is characterized by osteitis fibrosa disseminata, fibrous dysplasia, pigmented maculae and endocrine dysfunctions with precocious puberty. The lesions of fibrous dysplasia are mostly present in the craniofacial bones, the ribs and skull; the other features of the syndrome may usually be absent. The most common form of autonomous endocrine hyperfunction of this syndrome is the gonadotropin-independent precocious puberty, the affected persons can have hyperthyroidism, hypercortisolism, pituitary gigantism and acromegaly, too. Nonendocrine abnormalities developing in case of this disorder include hypophosphatemia, chronic liver disease, tachycardia, and, rarely, sudden death possibly caused by cardiac arrhythmias.

Precocious puberty, the most common endocrine disorder of Albright Syndrome, is the result of a gonadotropin independent autonomous ovarian or testicular function. Precocious puberty caused by this disease is far more common among girls than boys. Female infants with Albright Syndrome can have breast development and vaginal bleeding even if they are but as young as 4 months. There does a dominant ovarian cyst develop independent of being stimulated by gonadotropins. This cyst secretes estradiol, causing sexual precocity. ACTH–independent hypercortisolism generally results in growth failure and hypertension even in infancy. In this case the adrenal glands are bilaterally enlarged, containing multiple small nodules in the cortex.

Hyperthyroidism occurs typically later in the Albright patient's childhood, but it can occur even in the first year of its life. Just like in case of hypercortisolism and precocious puberty, hyperthyroidism associated with Albright Syndrome is a result of one or more autonomous hyperfunctioning nodules.

Hypophosphatemia is a result of a decreased reabsorption of phosphates in the renal tubules, similar to the phosphaturia experienced in case of hyperparathyroidism. The parathyroid hormone levels are not elevated, suggesting a parathyroid hormone-independent stimulation of phosphaturia. Growth hormone excess from somatotrop adenomas in the pituitary gland can occur at any age, resulting in gigantism and/or acromegaly.

A persistent tachycardia could be observed in addition to a mild-to-moderate cardiomegaly (due to *Coxsackie-viral* infection). Tachycardia resulting from severe hyperthyroidism may complicate or trigger a cardiac dysfunction. Gigantism or acromegaly occur with a risk of developing glucose intolerance, hypertriglyceridemia, hypertension and a mild myopathy.

(Symptoms of an intracranial process, such as abrupt vision changes, night-time headaches, or night-time emesis, are characteristic for hypothalamic lesions, can lead to gonadotropin dependent precocious puberty but are not consistent with Albright Syndrome.)

Genetic predisposition: Albright Syndrome is the result of a postzygotic somatic mutation in the gene that codes for Gsa. G proteins bind the cell surface receptors to intracellular proteins in order to activate or inactivate the signaling cascades. The stimulatory G protein becomes normally activated when a hormone or an other ligand binds itself to the cell surface receptor. The specific mutations causing these syndromes occur at a locus in the protein mediating the inactivation of the Gsa subunit. The specific phenotype of the patient depends on the cell type containing the mutation. The classic triad of the features present in

Albright Syndrome, i.e. polyostotic fibrous dysplasia, autonomous endocrine function and café au lait skin pigmentations, can all be explained by the activation of the Gsa subunit and by the increased intracellular cAMP.
Eumelanogenesis (the formation of brown/black pigments) is normally stimulated by melanocyte-stimulating hormones binding themselves to the MSH receptors, a classic G protein receptor coupled to Gsa. The constitutive activation of the Gsa subunit in melanocytes results in the increase of brown pigmentations characteristic of the café au lait spots seen in this syndrome.
There are different *viral and mycoplasmal infections* present in the Albright Syndrome.
Diagnosis: depends on finding at least 2 of the phenotypic features associated with activating Gsa mutations. The presence of 2 distinct physical findings consistent with autonomous function increases the likelihood that the single underlying cause is an activating Gsa mutation rather than activating mutations in genes specific to a tissue type. By molecular analysis of the affected tissue.
Treatment: symptomatically and by orthopedic operations.
RFR method can detect and may eliminate the pathogen microorganisms.
The most frequent resonances are: 285-301, 340-341, 343-347, 352-363, 370-383, 393, 402-410, 418, 428-430, 440-451, 472, 513 kHz

29.2. Alport Syndrome

Alport Syndrome encompasses a group of heterogeneous inherited disorders involving the basement membranes of the kidney and frequently involving also the cochlea and the eye. Alport Syndrome is a genetic disorder characterized by glomerulonephritis, endstage kidney disease, loss of hearing and eye problems. Hematuria is almost always found in this illness. Alport Syndrome is caused by genetic mutations in the collagen biosynthesis genes COL4A3, COL4A4, and COL4A5. Mutations in everyone of these genes prevent the proper production and assembly of collagen type IV, an important structural component of basement membranes in the kidney, inner ear and eye. (Basement membranes are thin, sheet-like structures that separate and support cells in many tissues.)
Criteria of the diagnosis of Alport syndrome:
1. Family history of hematuria, progressing mostly in males to an end-stage renal disease, and posterior polymorphous corneal dystrophy.
2. Thickening and splitting of the glomerular basement membrane detected by electron microscopy.
3. Progressive, high-frequency, sensorineural deafness in frequencies ranging from 2000-8000 Hertz (Hz).
4. Anterior lenticonus and perimacular flecks.
Treatment: by kidney transplantation (and gene therapy?).
RFR method detects the microorganisms. Alport Syndrome patients usually have several different pathogens, the most important among them being *Mycoplasma fermentans* and various kinds of *Çoxsackie viruses*. These pathogen microorganisms can be found in mother and baby.
The most frequently found pathogen microorganisms in case of Alport Syndrome are: 270-274, 280-302, 304-309, 319-321, 442-451, 493-495, 540-544 kHz
Questions to be answered are:
1. Do these pathogen microorganisms play a role in the development of Alport Syndrome?
If yes:
2. What is their role in the genetic mutation of the collagen biosynthesis genes?
3. Do they influence the further development of the disease?

29.3. Chediak-Higashi Syndrome

Chediak-Higashi Syndrome (CHS) is a rare, in an autosomal recessive way hereditary disorder which results in a pigmentary dilution of the skin and hair (silver hair), as well as in the presence of large clumps of pigment in the hair shafts and in the accumulation of melanosomes in the melanocytes. In case of its variant, hepatosplenomegaly, lymphohistiocytosis, a combined T- and B-cell immunodeficiency with neutropenia and skin infections such as cellulitis and abscesses can develop. The associated immunodeficiency often causes an impaired Natural Killer Cell activity, the damage of delayed-type hypersensitivity and a poor cell proliferation response to antigenic challenges. CHS patients with an abnormal NK-cell function suffer from several different infections (f.i. aphthae and gingivitis) and peripheral neuropathy as well.

Their primary infections are usually caused by *HTLV, HBLV* and various *Mycoplasma species.* (mostly by *M. fermentans).*

Their secondary infections usually occur due to different antibiotics-sensitive or resistant *Staphylococcus aureus* species, *Streptococcus pyogenes, E. coli, etc.*

The CHS gene regulates the synthesis and maintenance of the storage and the secretory granules of various cell types. The lysosomes of leukocytes and fibroblasts, the dense bodies of platelets, the azurophilic granules of neutrophils, and the melanosomes of melanocytes get generally larger in size as well as irregular in their morphology, indicating that a common pathway is affected in the synthesis of organelles responsible for the storage in patients with CHS.

Genetic predisposition: The CHS locus present on the human chromosome 1 encodes a lysosomal trafficking regulator, termed *CHS1. Mutations in one of its 3 genes can damage its function.* Two of these genes are located at band 15q21 and named RAB27A and MYO5A. Defects of these 2 genes can result in similar and distinct physical and pathologic findings as well. The third form of CHS, showing the characteristic hypopigmentation of the disease, results from the mutation in the gene encoding melanophilin (MLPH). An identical phenotype can result from the deletion of the MYO5A F-exon. The first genetic defect identified in CHS was that of the gene coding for myosin V-MYO5A. Later on, a second gene, the guanosine triphosphate (GTP)-binding protein RAB27A whose gene product is a reticular activating system–associated protein (RAS-associated protein), was cloned on a nearby locus. Mutations in RAB27A were found in all analyzed patients with CHS who had no mutated MYO5A.

Myosin Va (or Myosin 5a) is a member of the unconventional class myosin V family. A mutation in gene myosin 5a causes pigment granule transport defects in case of CHS. Slac2-a/melanophilin links the function of myosin 5a and of GTP-Rab27A present in the melanosome.

The gene products of MYO5A and RAB27A influence the movement of melanosomes, so that their defects cause pigmentary dilutions. The genes MYO5A and RAB27A are differently expressed in the body and cells. MYO5A is expressed in the brain, whereas RAB27A is not. Defects in MYO5A cause neurologic pathology, while defects in RAB27A do not.

The GTP-binding protein, which is the gene product of RAB27A appears to be involved in the control of the immune system, as all patients with RAB27A mutation develop a certain form of CHS, while those with MYO5A mutation do not. Rab27A-deficient T cells exhibit reduced cytotoxicity and cytolytic granule exocytosis, while MYO5A-defective T cells do not. Rab27A appears to be a key effector of cytotoxic granule exocytosis, a pathway essential for immune homeostasis. RAB27A-deficient T cells have a normal granule content in perforin and granzymes A and B, but show a defective granule release.

The onset of CHS (accelerated phase) seems to be associated with a viral infection (caused f.i. by *EBV, Hepatitis A Virus, HHV6)* and sometimes with a bacterial infection. Following their remission recurrent, accelerated phases of CHS with increasing severity can be experienced. Patients with RAB27A mutation have also neurologic problems related to

CHS as well as lymphohistiocytic infiltrations in the CNS. The CNS problems of patients with mutations in MYO5A do not wax and wane.
As mentioned above, an other gene termed MLPH located in humans at band 2q37 produces melanophilin, which is involved in melanosome movements and in the interaction of the gene products of RAB27A and MYO5A.
Neeft et al have found that Munc13-4 interacts with Rab27a. Rab27a and that Munc13-4 are intensely expressed in the cytolytic T lymphocytes and mast cells. Rab27a and Munc13-4 co-localize on secretory lysosomes. It was also found that the overexpression of Munc13-4 enhances the degranulation of the secretory lysosomes in mast cells. This finding demonstrates that Munc13-4 plays a positive regulatory role in the secretory lysosome fusion. Further on it was supposed that the Rab27a/Munc13-4 complex is an essential regulator of the secretory granule fusion with the plasma membrane in hematopoietic cells.
The diagnosis is based on the symptoms and genetical examinations.
Treatment: by administering antibiotics in order to cure primarily and secondary infections.
RFR method: detects and may eliminate the pathogens present.
The most frequent resonances of the primary infections are: 297-299, 315, 321-324, 330, 340-341, 354, 359, 365, 370-374, 382, 428, 432-433, 438-440, 442-451, 453-455, 459-464, 476-479, 482, 485-490, 493-495, 523, 526-530, 574 kHz
The most frequent resonances of the secondary infections are: 360-376, 377-382, 392-393 kHz
As to the frequencies of antibiotics resistant *Staphylococci* see the special Chapter of SARS.

29.4. DNA Repair Inhibition Syndrome

DNA Repair Inhibition Syndrome (DRIS) is characterized by signs of aging prematurally. Hutchinson-Gilford Progeria Syndrome (HGPS), Werner Syndrome (WS), Cockayne Syndrome (CS), Bloom Syndrome and Fanconi's anemia are inherited diseases, characterized by aging prematurally. In case of these syndromes the DNA repair does not function normally. DNA repair is in connection with regeneration. The fact, how long the cells will live, is determined by genes. If the cells of an organ die, the organ will begin to function badly and will, eventually, not maintain the biologic functions necessary to live. Programmed senescence determines the maximal age of an organism. By aging more and more damages can affect the cells, so that many a cell can no more function normally and will thus die, causing the death of the body. In human cells, and eukaryotic cells in general, DNA is found in two cellular locations: inside of the nucleus and inside of the mitochondria. Nuclear DNA (nDNA) exists during the non-replicative stages of the cell cycle as chromatin, and is, during the cell division, condensed into aggregate structures known as chromosomes. The DNA is in both state highly compacted and wound around by bead-like proteins named histones. Whenever a cell needs to express the genetic information encoded in its nDNA the required chromosomal region is unravelled, genes located therein are expressed, and then the region is condensed back to its resting conformation. The mitochondrial DNA (mtDNA), existing in multiple copies, is located inside the mitochondrial organelles, and is also tightly associated with a number of proteins in order to form a complex, known as nucleoid. Inside of the mitochondria, reactive oxygen species (ROS) and free radicals, both byproducts of the constant production of adenosine triphosphate (ATP) via oxidative phosphorylation, create a highly oxidative environment, able to damage the mtDNA. A critical enzyme, counteracting the toxicity of these byproducts is named superoxide dismutase, present in the mitochondria and in the cytoplasm of eukaryotic cells. DNA repair means a collection of processes by which a cell is identifying and correcting the damage done to the DNA molecules encoded in its

genome. In human cells, normal metabolic activities, free radical formations and environmental factors, i.e. UV light and radiation can cause DNA damages, causing about 1 million individual molecular lesions per cell and per day. Many of these are structural damages to the DNA molecule and can alter or eliminate the ability of the cell to transcribe the gene encoded by the affected DNA. Other lesions induce potentially harmful mutations in the cell's genome, which after undergoing mitosis, affect the survival of its daughter cells. Consequently, the DNA repair process is constantly active as it responds to the damage in the DNA structure.

The rate of the DNA repair depends on many a factor, including the extracellular environment, and the type and age of the cell. In contrast to DNA damages, mutation means a change in the base sequence of the DNA. A mutation, present in both of the DNA strands can not be recognized by enzymes, so that it can not be repaired. When the cell replicates, mutations will also be replicated. Mutations can cause alterations in the function of proteins and in the regulation of the affected cells. In a population of cells, mutant cells will either increase or decrease in frequency according to the effects of the mutation on the ability of the cell to survive and reproduce itself. DNA damages and mutations are related, though they distinctly differ from each other. DNA damages often cause errors of the DNA synthesis during their replication or repair, which errors may lead to mutations. If the DNA damage corrupts the integrity and accessibility of an essential information in the genome, the cell will not be able to function (though it will function superficially, if the so-called "non-essential" genes are missing or damaged). Depending on the type of the damage inflicted on the double helical structure of the DNA, a variety of repair strategies can be involved in the restoration of the lost information. Cells will, if possible, use the unmodified complementary strand of the DNA, or the sister chromatid as a template in order to recover losslessly the original information. Without finding an access to a template, cells, as a last resort, use an error-prone recovery mechanism known as translesion synthesis. A damage to DNA can alter the spatial configuration of the helix, which can be detected by the cell. Once a damage is localized, specific DNA repair molecules near the locus of the damage, inducing thus other molecules to bind and form a complex that enables the actual repair to take place. The type of the involved molecules as well as the mobilized mechanism of the repair depend on the type of the damage and the phase of the cell cycle in which the cell is in.

Base excision repair (BER) means the repair of damages done to a single nucleotide caused by oxidation, alkylation, hydrolysis, or deamination. The base will be removed by glycosylase and will ultimately be replaced by the repair synthesis done with DNA ligase.

Nucleotide excision repair (NER) means the repair of damages affecting longer strands of 2-30 bases. This process recognizes bulky, helix-distorting changes such as thymine dimers as well as single-strand breaks (repaired with enzymes such as UvrABC endonuclease). The specialized form of NER, known as Transcription-Coupled Repair (TCR) deploys NER enzymes of high-priority to genes being actively transcribed.

Mismatch repair, (MMR) means the correction of errors of DNA replication and recombination that result in mispaired (but normal, that is non- damaged) nucleotides. In case of aging syndromes and often also in case of genetically predisposed diseases and diseases associated with damages of genes, these DNA repair mechanisms are inhibited by *combined viral and mycoplasmal infections.*

The most frequent resonances: 370-374, 442-451, 534-544 kHz

29.4.1. Hutchinson-Gilford Progeria Syndrome

This syndrome is an extremely rare hereditary *(viral)* disease or an *acquired viral disease* that affects the skin, the musculoskeletal system and the vasculature. Hutchinson-Gilford Progeria Syndrome (HGPS) is characterized by signs of premature aging, as the repair process does not work. Its significant morbidity and mortality is caused by the accelerated

atherosclerosis of the carotid and coronary arteries, leading to premature death within the first or second decade of life. In contrast to the arteriosclerosis of the general population, the only lipid abnormality in case of this progeria is the decreased high-density lipoprotein cholesterol level and the development of diabetes.
Patients with HGPS can also develop other clinical signs of accelerated aging, including loss of subcutaneous fat and muscle, skin atrophy, diabetes, osteoporosis, arthritis, small growth and alopecia. It is interesting that patients with HGPS do not develop other disease processes associated with aging, such as increased tumor formation, cataract development or senility. In this sense, HGPS is considered to be a segmental progeroid syndrome, as it does not recapitulate every characteristic phenomena of aging.
Extensive lipofuscin deposition, a marker for aging, is extensively distributed in the body of patients with HGPS. Affected organs include the kidneys, brain, adrenal glands, liver, testes and the heart.
The syndrome is related to the aberrant processing of the nuclear envelope protein lamin A and to the accumulation of farnesylated prelamin A. Autosomal dominant mutations in the *LMNA* gene, located on band 1q21.1-1q21.3 are responsible for most of the HGPS cases. De novo mutations associated with the advanced paternal age are responsible for most cases, although a case of maternal transmission of a mutant *LMNA* gene from an asymptomatic mother with somatic and gonadal mosaicism has also been reported. In addition, autosomal recessive transmission is also thought to account for the reported development of HGPS in several sets of siblings born to unaffected parents. *LMNA* genes encode nuclear A-type lamins, which are type V intermediate filament proteins localized in the cell nucleus and form the nuclear lamina, a structure supporting the nuclear envelope. They are important in maintaining the nuclear stability and in organizing the nuclear chromatin. The nuclear lamins may also play a role in the regulation of gene expression, DNA synthesis and DNA repair. The most common *LMNA* mutation involves a C–>T transition at nucleotide 1824 (G608G), which substitution results in the production of a truncated lamin A protein, called progerin. The abnormal progerin protein acts in a dominant-negative manner to prevent the normal assembly of nuclear lamins into the nuclear lamina. As a result of the absence of lamin A in the nuclear lamina, the cell nuclei from HGPS patients display abnormal nuclear blebbing and aberrant nuclear shapes.
A transgenic mouse model for HGPS has been created by introducing a splicing defect into intron 9 of the mouse *LMNA* gene. Transgenic mice display many of the features of HGPS, including loss of subcutaneous fat, decreased bone density, growth failure, craniofacial deformities, skeletal abnormalities and early death.
Several genes that help to control mitosis are, as concerns progeria patients, down-regulated . Many of the genes that control cell division, DNA or RNA synthesis and processing were also found to be down-regulated in progeria patients; moreover many of these changes can also be experienced in case of normal aging. Some of these changes may lead to genetic instability and to a variety of disturbances in gene function.
Changes were also seen in the expression of many genes involved in collagen remodelling and in the formation of the extracellular matrix. In general, these changes favor the extracellular matrix deposition, leading to the characteristic changes seen in the skin and the vasculature of progeria patients. Expression of transforming growth factor-beta, a factor that regulates tissue homeostasis and the sustained expression of which is responsible for tissue fibrosis, is highly up-regulated in patients with progeria.
The expression of several transcription factors, involved in the musculoskeletal development, is also decreased in progeria patients. Expression of MEOX/GAX, a negative regulator of the cell proliferation in mesodermal tissues, is elevated almost 30-fold in patients with HGPS, suggesting a contributory role in the development of the musculoskeletal abnormalities seen in HGPS.

An increase in the excretion of hyaluronic acid is characteristic in persons with progeria. Besides persons with progeria, this increase can only be detected in case of those with Werner Syndrome, a disease characterized by a later onset of premature aging, occurring in the second decade of life. The production of hyaluronic acid and other glycosaminoglycans increases usually in the fifth to seventh decade of life. The increase in hyaluronic acid is possibly a normal feature of advancing age. The fibroblasts from patients with progeria show a 3-fold increase in their total glycosaminoglycan production, in particular, in their hyaluronic acid production, as compared to the age-matched control groups. This increase is owing to an abnormal degradation and not to an increased synthesis.
Data got from the embryonic development point to the importance of the changes in the level of hyaluronic acid for a person's morphological development. Experiments performed in chick embryos have demonstrated a correlation between cell differentiation and hyaluronic acid degradation. Hyaluronic acid is also necessary for the morphologic development of the blood vessels of chick embryos. In regions with high hyaluronic acid levels, the reduction or absence of the blood vessels can be experienced. The decreased density of vasculature, sclerodermatous changes in the skin, and the high prevalence of cardiovascular diseases present in persons with progeria may be induced by increased levels of hyaluronic acid. This increased level may also promote the calcification of blood vessels, contributing thus to arteriosclerosis.
Looking for the link between progeria and aging researchers investigated (among other topics) the role of the life span of fibroblasts. Cells taken from older donors exhibit a reduced number of cell divisions in comparison to cells of younger donors. The reducted life span of cultured fibroblasts derived from patients with progeria revealed inconsistent results. Its significant reduction had been claimed in some studies, but was questioned in later investigations. A recent thorough study indicates that the life span of fibroblasts in cultures does not depend on the donor's age.
The changes, observed in cultured fibroblasts from patients with progeria, are reduced mitotic activity, DNA-synthesis and cloning efficiency and a reduced capacity for DNA repair following gamma irradiation.
The most frequent resonances: 442-451, 534-544 kHz

29.4.2. Werner Syndrome

This syndrome is characterized by sclerodermalike, thin and tight skin as well as by bilateral cataracts. Werner Syndrome (WS) is known also as progeria adultorum, progeria of the adults and pangeria. WS is the most common disorder of premature aging. The aging process involves increasing errors in the mitotic machinery of the dividing cells in the postreproductive stage of life; so that the WS serves as a model for studying the human aging process in vivo and in vitro, as many organs of patients with WS prematurely undergo changes, usually associated with aging. In case of this syndrome no mental retardation can be observed. WS is an autosomal recessive disorder, affecting the connective tissues of the body. This entity is caused by a mutation at the locus of WS gene (*WRN*), which gene encodes a conserved protein, belonging to the family of *RecQ* helicases, thus, WS is caused by a helicase enzyme defect. Enzymes of this group unwind the double helix RNA and DNA. This protein is likely involved in the replication and transcription processes. The disease is connected with an excessive synthesis of collagen types I and III, dependent on elevated messenger RNA (mRNA) levels. In case of this syndrome the collagenase level is also many fold increased. Helicase enzymes play an important role in the protecting of cells against chromosome breakage at fork-stalling points, in case of normal replication.
The most frequent resonances: 317-319, 343-344, 353-359, 387-389, 438, 442-451, 482-486, 516-519, 550-552, 563-565 kHz

24.5. Addison's Disease

Addison's disease (AD) is an adrenocortical insufficiency. Addison's classic description of this disorder sounds as follows: „general languor and debility, remarkable feebleness of the heart's action, irritability of the stomach, and a peculiar change of the color of the skin." The disease, if unrecognized and untreated, carries an almost uniformly poor and frequently fatal prognosis [redacted] Chapter 23.17.).

The adrenal glands are sometimes the loci of chronic, granulomatous, infectious diseases predominately of *tuberculosis* but also of fungal infections, f.i. *histoplasmosis*, *coccidioidomycosis*, and *cryptococcosis* or/and of viral infections such as *HTLV*, and *Mycoplasma fermentans*. An idiopathic atrophy of the adrenal glands can be frequently observed, suggesting that an autoimmune mechanism may be responsible for its pathogenesis.

Addison's disease occurs when the adrenal glands do not produce enough hormone cortisol, nor hormone aldosterone in some cases. Its problem may be due to a disorder of the adrenal glands themselves (primary adrenal insufficiency) or to an inadequate secretion of ACTH by the pituitary gland (secondary adrenal insufficiency). Cortisol belonging to the glucocorticoids is normally produced by the adrenal glands and it affects almost every organ and tissue of the body. Its most important function is to help the body to respond adequately to stress. The most important effects of cortizol are as follows:

– Helping to maintain blood pressure and cardiovascular function;
– Helping to slow down the inflammatory response of the immune system;
– Balancing the effects of insulin in breaking down sugar for energy; and
– Helping to regulate the metabolism of proteins, carbohydrates and fats.

Cortisol is of vital importance to health, its amount produced by the adrenals is precisely balanced. Like many other hormones, cortisol is regulated by the hypothalamus and the pituitary gland. First, the hypothalamus sends corticotropin-releasing hormones (CRHs) to the pituitary gland, which responds by secreting hormones that regulate growth, thyroid and adrenal function, and sex hormones such as estrogen and testosterone. One of the pituitary's main functions is to secrete ACTH (adrenocorticotropin), that stimulates the adrenal glands. When the adrenals receive the pituitary's signal in form of ACTH, they respond by producing cortisol. Completing the feed back cycle, cortisol then signals the pituitary to lower the amount of the secretion of ACTH.

Aldosterone belongs to a class of hormones called mineralocorticoids, produced likewise by the adrenal glands. It helps to maintain the blood pressure, water and salt balance in the body by helping the kidney to retain sodium and to excrete potassium. If the aldosterone production gets to be too low, the kidneys will be unable to regulate the salt and water balance, so that the blood volume and pressure will decrease.

Almost every case of Addison's disease is caused by the gradual destruction of the adrenal cortex by the immune system of the body. About 70 percent of the reported cases of Addison's disease **are caused by autoimmune disorders**, in which the immune system produces antibodies that attack the body's own tissues or organs and slowly destroys them. Adrenal insufficiency occurs if at least 90 percent of the adrenal cortex is destroyed, as a result of which, glucocorticoid and mineralocorticoid hormones are often both lacking. Sometimes only the adrenal gland is affected, fi. in idiopathic adrenal insufficiency; though sometimes also other glands are affected, as f.i. in case of polyendocrine deficiency syndrome.

Polyendocrine deficiency syndrome Type I occurs among children, the adrenal insufficiency may be accompanied by the hypofunction of the parathyroid glands, slow sexual development, pernicious anemia, chronic candida infections and chronic active hepatitis.

common single nucleotide polymorphism in CKN1. No genotype-phenotype correlation exists. Ocular histopathologic findings indicate degeneration of all retinal layers. Pigment migrates into the photoreceptor layer. Nerve fiber bundles of the optic nerve head become markedly thin, while partial demyelination of the remaining nerves occurs.
For patients with sensorineural hearing loss, a significant loss of neurons occurs in the spiral ganglion and brainstem, with retrograde atrophy of the auditory pathways.
As mentioned above, DNA damages and mutations are fundamentally different. Damages are physical abnormalities in the DNA, such as single and double strand breaks, 8-hydroxydeoxyguanosine residues and polycyclic aromatic hydrocarbon adducts.
DNA damages offer a special problem concerning non-dividing or slowly dividing cells, where unrepaired damages tend to accumulate over time. In rapidly dividing cells, unrepaired DNA damages which do not kill the cell by blocking the replication, will tend to cause replication errors and thus mutation. The majority of mutations are not neutral in their effect and are deleterious to a cell's survival. Thus, in a population of cells comprising a tissue with replicating cells, the mutant cells will get lost.
Following a DNA damage, cell cycle checkpoints will get activated. This checkpoint activation interrupts the cell cycle giving thus time enough to repair the damage before the cell divides. The checking of the DNA damage occurs at the G1/S and G2/M boundaries. An intra-S checkpoint does also exist. Checkpoint activation is controlled by two master kinases, ATM and ATR. ATM controlls the DNA double-strand breaks and disruptions in the chromatin structure, while ATR primarily controlls the stalled replication forks. These kinases phosphorylate downstream targets in a signal transduction cascade, leading eventually to cell cycle arrest. A class of checkpoint mediator proteins including BRCA1, MDC1, and 53BP1 was also identified. These proteins seem to be required for transmitting the checkpoint activation signal to downstream proteins. p53 is an important downstream target of ATM and ATR, as it is required for inducing apoptosis following a DNA damage.
If the rate of the DNA damage exceeds the capacity of the cell to repair it, the accumulation of errors can overwhelm the cell and result in early senescence, apoptosis or cancer. Inherited diseases associated with faulty DNA repair functioning lead to premature aging, increased sensitivity to carcinogens and thus, to an increased risk of getting cancer.
Defects in these mentioned mechanisms are responsible for several genetic disorders, including:
Xeroderma pigmentosum, which is characterized by hypersensitivity to sunlight/UV, resulting in an increased incidence of skin cancer and in premature aging.
Ataxia telangiectasia (Louis–Bar syndrome), an autosomal recessive disorder, characterized by cerebellar ataxia, telangiectases, immune defects and by predisposition to malignancy. AT cells are extraordinary sensitive to killing by ionizing radiation.
Cockayne Syndrome, characterized by hypersensitivity to UV and chemical agents,
Trichothiodystrophy, characterized by the sensitivity of the skin, brittle hair and nails.
Mental retardation often accompanies the three latter disorders, suggesting an increased vulnerability of the developing neurons.
The regenerative capacity of an organ is assured by a very complex process. Limb regeneration of newts occurs in two major steps, i.e. by the de-differentiation of the adult cells into a stem cell state similar to that of embryonic cells, and then, by their re-differentiation into a new limb-forming tissue, more or less in the same way as they developed at first in their embryonic state.
Some researchers claim that if the periosteum, the membrane surrounding the rib, is left intact, the human rib could get regenerated. Moreover, the given source states that a piece of the human rib transplanted to the foot could survive at this new place, if the periosteum was transplanted together with it. There have also been attempts to sustain the claim by noting that some transplanted back muscles might restore the functionality of other muscles.

The human liver is one of the few glands in the body that has the ability to regenerate from as little as 25% of its tissue. This is largely due to the unipotency of hepatocytes. The resection of the liver can induce the proliferation of the remained hepatocytes until the restoration of the loosed mass takes place, where the intensity of the liver's response is directly proportional to the mass resected. Since almost 80 years the surgical resection of the liver of rodents proves to be a very useful model for studying cell proliferation. Healing, ment physically, is the process by which the cells in the body get regenerated and repaired, reducing the size of a damaged or necrotic area. Healing incorporates both the removal of the necrotic tissue (demolition), and the replacement of this tissue.
The replacement can happen in two ways: by regeneration, in case of which the necrotic cells are replaced by the same tissue as originally, and by repair, in case of which the injured tissue is replaced by scar tissue. The healing of most organs happens by using these two processes. Cells need also a collagen framework along which to grow. Alongside most cells there is either a basement membrane or a collagenous network made by fibroblasts that will guide the growth of the cells. As ischemia and most bacterial or viral toxins do not destroy gridfibres and collagens, they will continue to exist even if the cells around them are dead.
Following an acuté injury, the regeneration of the tubular component of the mammalian kidney is well known. The regeneration of the glomeruli has recently also been documented. Following an acute injury, the proximal tubule is more severely damaged, the injured epithelial cells will slough off the basement membrane of the nephron. The surviving epithelial cells, however, undergo a certain migration, de-differentiation, proliferation and re-differentiation in order to replenish the epithelial lining of the proximal tubule. Recently, the presence and participation of kidney stem cells in the tubular regeneration have also been experienced. More over, in addition to the surviving tubular epithelial cells and kidney stem cells, the bone marrow stem cells are also found to participate in the regeneration of the proximal tubule, though the mechanisms remain highly controversial.
Mutations and heritable alterations in the genetic material may be *gross* (at the level of the chromosome), or *point-like alterations* (which latter means mutations not visible as cytological abnormalities and/or can involve even just a single nucleotide pair in the DNA). The consequences of base substitution mutations in protein-coding regions of a gene depend on the substitution and its location. They may be silent, without resulting in a new amino acid in the protein sequence, or can be a missense mutation causing an amino acid substitution (for example CTC in the DNA sense strand specifying a glutamate residue in the protein; is altered into CAC in the DNA resulting in a valine residue in the beta-globin protein chain causing thus **sickle-cell anemia**.)
Missense mutations may have very serious consequences, as in case of sickle-cell anemia, mild consequences as in case of **hemoglobin C** (a different amino acid substitution in position 6 of beta-globin) and no concequences concerning the phenotype, as in case of two known amino acid substitutions at position 7 of beta-globin. Finally, base substitutions in a protein-coding region may mutate an amino acid codon to a termination codon or vice versa. The former type, which results in a prematurely shortened protein is referred to as a **nonsense mutation.** The effects of nonsense mutations are variable depending upon how much of the truncated protein is present and how much this mutation influences its functioning.
Base substitution mutations may also occur in the promoter and 5' regulatory regions of genes or in introns and may affect their transcription, translation, or splicing. Many forms of **Beta-thalassemia** are the result of these non-structural mutations that affect the level of expression of the globin genes.
All types of mutation described above have been observed in human globin genes. Their consequence depend on their influence on the level of the expression of the gene product,

on the kind of the amino acid substitution caused and on the locus of this substitution in the protein.

The general regeneration process is inhibited in the **DRIS syndrome**. *Coxsackie B4* and *certain other viral infections* can inhibit the cellular repair mechanisms. These viral agents prohibit the functioning of the human DNA repair genes and thus also the cellular regeneration. The most important question is: in which way these viral infections inhibit the functioning of human DNA repair genes. Neither the premature aging syndrome, nor the becoming pathologically old can be healed by convencional methods.

RFR method: detects and eliminates the pathological microorganisms.

The most frequently found resonances are:

those of Mycoplasma fermentans: 442-451 kHz,

those of Human T-cell Lymphotropic Virus: 370-376 kHz and

those of other viral agents: 268-278, 534-545 kHz (These resonant frequencies may belong to the Coxsackie B4 virus, or to a species of the HPV group, or perhaps even to a virus not identified as yet), **and other frequenties: 353-358, 387, 482, 510, 517, 545 kHz**

All inherited characteristics are encoded by genes. Some deficient characteristics, such as the Hutchinson-Gilford Progeria Syndrome, Werner Syndrome, Cockayne Syndrome, Bloom's syndrome, Fanconi's anemia and other similar diseases are caused by the damage of repair functioning. However, abnormal characteristics expressed by an abnormal gene may be caused by hereditary diseases as well.

Accordingly, by eliminating the inhibitor viruses, the repairing of DNS and thus the regeneration of tissues can offer a new possibility of healing. I plan to cover these developments in my forthcoming book.

29.5. Neuroacanthocytosis

Neuroacanthocytosis (named also Levine-Critchley syndrome and Chorea-acanthocytosis) is a rare inherited movement disorder characterized by progressive muscle weakness and atrophy, progressive cognitive loss, chorea, involuntary twisting movements of the body and acanthocytotic (spiked) red blood cells. The symptoms of this disorder are resulted by the degeneration of the basal ganglia that helps control movements and by the loss of neurons in the brain and the spinal cord. Neuroacanthocytosis has been described as an autosomal recessive way inherited disorder, as an autosomal dominant way inherited disorder and as a part of an X-linked inherited disorder named McLeod syndrome. Acanthocytosis has also been mentioned associated with the rare hypobetalipoproteinemia, acanthocytosis, retinitis pigmentosa and pallidal degeneration (HARP) syndrome, a disease of childhood akin to Hallervorden-Spatz disease and a defect in the gene for pantothenate kinase.

A deletion mutation in the gene localized to chromosome band 9q21 was identified as the locus for the defect generating the autosomal recessive form of NA (2001). This mutation leads to chorein protein deficiency or absence. In 2005, researches involving affected French-Canadian families presenting temporal lobe epilepsy, an expanded conceptualization of the molecular genetics of the autosomal recessive form NA was attained. 70-80% of the family members had large deletions in the NA gene, now known as VPS13A, on chromosome 9. Some family members with no epilepsy but with milder features, such as tics and dysphagia for example, may be representatives of a heterozygous expression of the deletion, suggesting that variations in the VPS13A gene may lead to a dominant pattern of inheritance. Antibodies to the GM1 ganglioside component of peripheral nerves have been described. This GM1 ganglioside is also present in RBC membranes and in the central nervous system. Decreases in GM3 and sialoparagloboside components of RBC membranes have been noted. These gangliosides are also present throughout the nervous system.

The pathophysiology of all forms of neuroakanthosis syndrome is based on different gene abnormalities causing multisystem membrane defects. The common derangement is in the malformation of the RBC shape and the induction of various levels dysfunction of the central nervous system, the neuromuscular, and cardiac system. Some kind of accelerated senescence and autoimmune damage of the erythrocytes and the nerve tissue holds a key in fully appreciating the triggering of acanthocytosis and neurodegeneration in case of these syndromes.
Parkinsonism has been associated with the disorder in some patients.
Diagnosis: symptomatically, there is no specific test as yet.
Treatment: symptomatic and supportive (f.i. antipsychotic, anticonvulsant and antidepressant drugs, etc.)
RFR method: can find the Mycoplasma and neurogen viruses.
The most frequent resonances are: 288-300, 324, 332-339, 356-359, 370-372, 402-410, 427-429, 442-451, 493-495, 518-519 kHz

29.6. Cystic Fibrosis

Cystic fibrosis (CF) is an autosomal recessive disorder, in which most carriers of the gene are asymptomatic. Cystic fibrosis is a disorder affecting the function of the exocrine glands, involves multiple organs and leads chiefly to chronic respiratory infections, pancreatic enzyme insufficiency and to complications of the untreated pancreatitis, systemic infection such as *mumps, rubella, Coxsackie virus B, cytomegalovirus (CMV), Mycoplasma, Human Immunodeficiency virus (HIV)* and *other HTLVs*, pancreaticobiliary malunion, congenital anomalies of the pancreato-biliary junction, pancreas divisum, congenital sphincter of Oddi abnormality, choledochal cysts, or choledocholithiasis.
Genetic predisposition: cystic fibrosis is caused by defects in the gene for cystic fibrosis transmembrane conductance regulator (CFTR), which encodes a protein that functions as a chloride channel and is regulated by cyclic adenosine monophosphate (cAMP). Mutations in the CFTR-gene result in abnormalities of the cAMP-regulated chloride transport across the epithelial cells on the mucosal surfaces. The damage of this protein leads to a decreased secretion of chloride and to an increased reabsorption of sodium and water across the epithelial cells, as well as to a thinner epithelial lining fluid and to an inspissated mucus, which sticks much more to bacteria and viruses causing infections and inflammation. The pancreatic acini will be substituted by fibrotic tissues, multiple cysts, inspissated mucus and eventually by fat.
Symptoms: These patients suffer from pancreatic enzyme insufficiency and have digestive problems, malnutrition and steatorrhea, so that they can not normally develop in their early life. The thick meconium of newborn infants suffering from cystic fibrosis can cause meconium ileus. The failure of chloride conductance by epithelial cells and associated water transport abnormalities result in viscid secretions The secretion in the respiratory tract, pancreas, GI tract, sweat glands and in other exocrine tissues shows an increased viscosity which can but hardly clear up and might lead to chronic recidive infections.
Diagnosis: by ultrasound, CT, MRI, by gene examinations, enzime function testing
Treatment: by pancreatic substitution therapy; by high calorie, high protein, low fat diet; by administering broad-spectrum antibiotics in case of acute or chronic bacterial infections and in case of congenital anatomic defects by surgical intervention.
RFR method can detect and eliminate the infective agents
The most frequent resonances of CF are: 335-339, 344-346, 364-366, 370-373, 397-399, 402-403, 408-412, 442-451, 472, 492, 498-500, 534-536, 570-572 kHz

ADDENDUM

The susceptibility of people to infections differs. The cause of which is, besides others, their genetic predispositon. People with certain gene disorders can show an enhanced susceptibility to infections caused by certain pathogens. It is to be hoped, that by activating the DNA repair mechanism this susceptibility can be reduced, and if that proves to be true, it might become one way of the prophylaxis of diseases.

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