ZAPOROZHYE STATE MEDICAL UNIVERSITY

THE DEPARTMENT OF INTERNAL DISEASES №3

Dotsenko S.Y., Afanas`ev A. V., Samura B. B., Tokarenko I. I., Tyaglaya V. N.

OCCUPATIONAL DISEASES

(tutorial for practical exercises)

ZAPORIZHZHYA

2015

Approved by the Central Methodological Advice of Zaporozhye State Medical University as a tutorial for practical exercises for 5th-year students of medicine faculty, protocol № ___ , from _____ 2015

Authors:
1. Dotsenko S.Y. - Head of Department of Internal Diseases №3, Zaporozhye State Medical University, Professor, Doctor of Medical Sciences
2. Afanas`Ev A. V. - Associate Professor of Department of Internal Diseases №3, Zaporozhye State Medical University, Candidate of Medical Sciences
3. Tocarenko I. I. - Assistant of Department of Internal Diseases №3, Zaporozhye State Medical University, Candidate of Medical Sciences
4. Tyaglaya V. N. - Assistant of Department of Internal Diseases №3, Zaporozhye State Medical University, Candidate of Medical Sciences
5. Samura B. B. - Associate Professor of Department of Internal Diseases №3, Zaporozhye State Medical University, Candidate of Medical Sciences

Reviewers:
V. G. Deynega - Honored Worker of Science and Technology of Ukraine, Doctor of Medical Sciences, Professor of Department of Family Medicine and Therapy of FPE
O. V. Kraydashenko - Doctor of Medical Sciences, Professor, Head of Department of Clinical Pharmacology, Pharmacy and Pharmacotherapy with the Course of Cosmetology

Dotsenko S.Y., Afanas`ev A. V., Samura B. B., Tokarenko I. I., Tyaglaya V. N. Occupational diseases (tutorial for practical exercises). – 2015, Zaporizhzhya. – 138 p.

Basic forms of occupational pathologies and their classification have been described in the tutorial for practical exercises. Issues on regional occupational diseases with the consideration of peculiarities and the structure of public economy of Ukraine have been considered. New data on pathogenesis, clinical symptomatology, development, treatment and organization of dispensary care have been suggested. Recommendations on rendering urgent medical assistance in case of some grave conditions and poisoning in clinics to treat occupational diseases have been provided. Principles on organization of conduct of medical examinations and solving expert questions in compliance with the regulations of recent acts have been analyzed.

For 5th-year students of medicine faculty

CONTENTS

FOREWORD

Social and economic development of the society envisages improvement of labor conditions, as well as decrease of the level of general and occupational diseases. As to occupational diseases, their structure, character and progress have changed. Nowadays there is almost no severe forms of acute intoxication by industrial poison (nitric oxide or carbon oxide, hydrogen sulphide, aromatic nitrocompound or Oilsperse), as well as pronounced forms of chronicle poisoning with lead, tetraethyl lead, mercury, manganese, benzol, or other toxic matters. However, radically changing the work character, scientific and technological progress brings up new factors of production environment, which have negative effect onto the employees. In some spheres, there is a threat of the impact of existing production problems due to intensification of production processes. First of all, this concerns the expansion of the production of plastics, synthetic resins, leather, caoutchouc, organic dyes, chemical fertilizers, pesticides, as well as medicinal drugs.

Notions of factors of low intensiveness and combined impact of some harmful factors of production environment have appeared, thus a need have emerged for early diagnostics of occupational diseases.

In the occupational pathology, there is still an actual problem of dust diseases of lungs or pneumoconiosis, as in the structure of occupational diseases they are the most prevalent. The main form of lung dust pathology, as it was before, is black-lung disease or dust disease, and first of all, silicosis. Besides, together with black-lung disease, dust bronchitis has spread much.

Intensive development of electronics and radio technology conditioned the necessity to study the impact of electromagnetic emanation onto those, who work in these spheres of the economy with the purpose of timely elimination of its negative effect.

Implementation of high-pace equipment, which generates vibration and is a noise source, into various spheres of industry, causes vibration disease or cochlear neuritis. And in spite of the developed means and methods to struggle against these factors, vibration disease is one of the most prevalent diseases in the structure of occupational pathology.

Complex mechanization and automation of production processes together with the increase of labor efficiency allowed decreasing burden onto the muscle system. At the same time, partial mechanization and automation of some production lines causes physical overstraining for locomotor system, muscle overstraining, in particular, when monotonous movements are made in haste. That's why occupational diseases of muscles, periphery nerves, and locomotor system can be met rather often and need timely diagnostics, treatment and preventive actions. Thus, under conditions of modern production, occupational diseases still affect the health condition of working people. This situation needs correct evaluation of sanitary and hygienic conditions of work, intensiveness and duration of the influence of these factors in every case with the purpose of due diagnostics and treatment of patients with occupational diseases.

Theme №1. GENERAL ISSUES ON OCCUPATIONAL PATHOLOGY

Notion of Occupational Pathology as a Clinical Discipline. Occupational diseases with occupational pathology is a division of clinical medicine devoted to studying occupational diseases which appeared under the impact of harmful factors in the production environment or work process.

Main objectives of the occupational pathology are as follows:

• study of occupational diseases, their pathogenesis, symptomatology, progress, long-term consequences, therapy issues, medical rehabilitation, and labor ability expertise; early diagnostics of occupational diseases is gaining particular importance;

• study of non-specific action of occupational factors, their meaning for development, progress and consequences of general and non-occupational diseases.

The notion "harmful production factor" means a factor of production environment, as well as peculiarities of production process, which can cause harmful action onto the body of a working person and to cause diseases.

By impact of their nature onto a human body, dangerous of harmful factors of the production environment can be divided into physical, chemical, biological and psychophysiological ones.

- Physical: machinery and moving mechanisms, moving elements of production equipment, falling rocks, high or low temperature of the surface, air in the work zone; increased level of noise, vibration on the work place; increased or decreased pressure within the work zone or its sudden change; increased level of ionizing emanation, etc.
- Chemical: organic and non-organic compounds in the form of gas, vapor, aerosol or liquid.
- Biological: biological objects, which include pathogenic microorganisms (bacteria, viruses, Rickettsia, spirochaetae, fungi, and photozoa), products of their life activity, as well as some organic matters of natural origin. Psychophysiological: physical, nervous and mental strains, which in their turn can be divided into mental strain, analyzer strain, work monotony and emotional strain.

Appearance of dangerous and harmful factors in production can be conditioned by the following: wrong organization of work process (irrational work and rest regime, unnatural body position, extreme pressure on individual organs and systems), low production culture, lacking or insufficient working sanitary or technical devices and equipment; problems with sanitary and technological solutions at some productions (dust control incoal and mining industries, normalization of microclimate at work places, and in deep mines); peculiarities of work processes, connected with strain in emotional sphere (complicated operator work under conditions of time deficit).

Unified classification of occupational diseases has not been developed until now. First of all, this can be explained by the fact that their clinical picture is often polymorph and can be characterized by the change of many organs and systems. Besides, they can be divided into specific and nonspecific occupational diseases.

The former notion includes diseases, which can appear only in case of occupational factors. They can be also called "absolutely specific". The second group of diseases includes "relatively specific" occupational diseases.

There are not that many absolutely specific occupational diseases: black-lung disease, vibration disease, or manganese intoxication.

Relatively specific diseases include intoxications, which sometimes have household origin, but more often they can be met in production conditions (intoxication with lead, mercury, arsenic, or pesticides), as well as radiation sickness, diseases of arms and hands due to functional straining. Particular clinical picture should also be taken into account.

Many diseases can be conditioned by not only occupational, but also harmful factors, though in specific professions and under the impact of specific occupational harms they can be met more often than

in other conditions (bronchial asthma among fur makers and pharmacists; chronic bronchitis among workers of "dusty" professions, etc). These are non-specific occupational diseases.

The most specific classification of occupational diseases is etiological classification. In compliance with the etiological classification, groups of occupational diseases can be identified, which are conditioned by the impact of the following:

1) industrial dust (black-lung disease and dust bronchitis);

2) physical factors of the industrial environment (vibration disease, cochlear neuritis, affections caused by the action of various types of emanation, high and low temperature, etc);

3) chemical factors of production environment (various acute and chronic intoxication);

4) biological factors (infectious and parasite diseases, which develop in those, who are in contact with various infectious material or animals with some infections, as well as among those, who work in tuberculosis and other infection medical institutions; diseases, caused by antibiotics, fungi producers, etc).

Sometimes the classification of occupational diseases includes system and organ principle (occupational diseases of the nervous system, of respiratory apparatus, cardio-vascular system, blood, etc). Diseases, which appear due to the impact of chemical compounds onto the body when working with it under conditions of production environment, are called occupational poisoning. It appears due to interaction of life organism and poisoning. ***The most popular classification of toxic matters, which reflects their practical utilization, is as follows:***

1) *industrial poisons used in production*: organic solutions (dichlorethane), dyes (aniline), chemical reagents (methyl alcohol), etc. ;

2) *poisoning chemicals used against agricultural plant pests*: organochloral pesticides (hexachloran, polychloropinen), organophosphorus insecticides (Trichlorfon, methylmercaptophos); organomercurous matters (granosan) and derivatives of aminoformic acid (Sevin);

3) medicinal drugs;

4) *household chemicals* used as food supplements (acetic acid), sanitary, personal hygiene and cosmetics means;

5) *biological plant and animal poisons* contained in plants and fungi, animals and insects (snakes, and bees); and

6) *military poisoning matters* (sarin, mustard gas or yperite, and phosgene).

Hygienic classification of poisons have been generally accepted, in the basis of which there is quantity assessment of toxic danger of chemical compounds based in experimentally determined fatal dose (DL50) and permitted marginal concentration (MPC). In compliance with this classification, toxic matter corresponds to specific degree of toxicity, which is characterized by its stronger or weaker danger.

The most important for clinical toxicology is the division of chemical compounds on their toxic action onto the body (toxicological classification). The following poisons are distinguished: neuropsychic (organophosphorus insecticides), blistering (dichlorethane, hexachloran, arsenic, or mercury), general toxic (carbon monoxide), asphyxiating (nitric oxide), tear and irritant (vapors of strong acids and alkali).

Background of Development of Occupational Pathology

Studies of occupational diseases have deep roots. Specific data onto the impact of harmful conditions of work onto the human health, as well as existence of particular occupational diseases can be met in ancient documents. As an example, we can name ancient Egyptian and Chinese characters and a code of medical rules and lectures by Hippocrates, Aristotle, Lucretius, Ovid, Plutarch, Pliny, Juvenal, and Galen.

The first description of occupational pathology was done by Hippocrates (460 - 377 BC), who compiled a list of existing at that time the so-called lead professions and described the clinics of poisoning with lead indetail. He also developed the description of negative impact of mining dust onto miners.

Significant growth of production in the late Medieval period (12th - 15th centuries), hard exhaustive labor in such spheres of industry as mineral resource industry and metallurgy, revoked interest in issues on labor hygiene and occupational diseases. In 1530, three-volume monograph was printed by an outstanding doctor and chemist Paracelsus "About Mountain Xerosis and Other Mountain Diseases", where he specifically described labor conditions, as well as occupational diseases of miners in the result of action of dust, sulfur, mercury vapor and other metals onto human body.

Works of an outstanding German doctor, metallurgist, mineral researcher Agricola (Georg Bauer) cause paricular interest, and especially his work "De re metallica" (1556), which describes description of diseases of miners, stating reasons for their appearance as well as prevention methods.

One of the first books on lead poisoning is the edition of 1556 - a monograph by Stockhausen, which has detailed for that time consideration of pathology, which appear due to inbreathing lead vapor. In 1614, Martin Pans, a local doctor from Anaberg (Saxony) wrote a book on miner's diseases. However, the first monograph, which had systemized description of issues on work hygiene of various professions and the description of specific diseases of workers of 52 professions, is the "Diseases of Workers" by an Italian doctor B. Ramazzini (1633 - 1714). It was published 25 times in various languages and brought world fame to its author.

Further development of industry in the Western Europe in the 18h and 19th centuries and the beginning of the 20th century was accompanied by the growth of professional diseases. Since 1782, in Germany and then in France, journals started being published, devoted to issues on general and, in particular, occupational hygiene. Articles told about the character of the impact of lead, copper, chromium, arsenic, phosphorus, as well as iodine onto a human body. Rather detailed monographs on dust, lead, phosphorus and other diseases of workers were published.

The founder of Russian science, culture and technology M. Lomonosov developed principles of occupational hygiene and occupational diseases in mineral resource industry. In the treatise "First Principles of Metallurgy or Ore Business" (1763), he provided specific detailed information on hazard impact of production factors onto the health of workers.

The most complete and systemized analysis of occupational diseases was done by A. Nikitin, doctor at Alexander's Textile Mill in St. Petersburg in his monograph 'Diseases of Workers and Their Prevention Methods" (1847). A. Nikitin used monograph by B. Ramazzini as the basis for his work, however he significantly changed and completed some chapters. The book played an important role in the development of hygiene and professional pathology. The end of the 19th and beginning of the 20th are characterized by rapid development of capitalism in Europe. Millions of workers worked at large enterprises; work conditions and lifestyle were very hard. At that time many factory doctors conducted research on hygiene, studied the impact of production factors onto the health condition and diseases of workers. Valuable contribution in studying occupational pathology was made by representatives of rural medicine - F. Yerisman, Ye. Osipov, and O. Pogozhev. An original manual "Occupational Hygiene or Hygiene of Mental and Physical Work" by F. Yerisman, published in 1877, was in big demand. Scale and depth of research is well shown in the 19-volume edition "Materials on Research of Factories and Plants", compiled based on research of 1008 factories and plants. It includes data on the health condition and physical development of 114 thousand workers. This work, completed by F. Yeresman, O. Pogozhev, Ye. Dementiev et. al., had no analogue in the world practice of that time.

Significant contribution into he development of home science on occupational hygiene and occupational pathology was made by D. Nikolsky, V. Levitsky, V. Novrotsky, G. Khlopin, M. Kavalyerov, I. Liatschenko, O. Navakatikyan, G. Yevtushenko, M. Paranko, and A. Shevchenko.

Among outstanding scientists, who had big impact onto the development of professional pathology as a science, it is necessary to mention such scientists as M. Vigdorchyk and S. Kaplun. M. Vigdorchyk published a number of important works on occupational pathology and statistics of professional diseases; he was also a director for Leningrad Institute of Occupational Hygiene and Occupational Diseases. S. Kaplun was an organizer and the first leader of the Central Institute on Labor Protection.

Together with the realization of practical activity in occupational pathology, research and development work was widely implemented as well. In 1923, Ukrainian Institute on Occupational Medicine (now, Kharkiv National Institute on Occupational Hygiene and Diseases) was created in Kharkiv; in 1924, there appeared Moscow Institute on Occupational Diseases named after Obukhov, and in 1925, Central Institute of Labor Protection, and Leningrad Institute of Occupational Hygiene. In the following years, research and development institutes in occupational hygiene and occupational diseases were formed in Kyiv, Donetsk, Dnepropetrovsk, Sverdlovsk, Krivoy Rog, and Karaganda. Almost all the medical institutions on refreshing courses conducted the work in this direction for doctors, as well as departmental institutes.

Establishment and development of industrial toxicology is connected with the names of M. Pravdin, M. Lazarev, G. Shkavera, O. Cherkes, and L. Medved; studies of production microclimate are connected with the names of A. Letavet, G. Shakhbazyan and M. Karnaukh; S. Andreyeva-Galanina, V. Artamonova and G. Balan studied impact of vibration ontot a human body;and professional pathology in agriculture was studied by Yu. Kundiyev and O. Krasnyuk.

Nowadays, issues on scientific development in the sphere of professional pathology are studied at R&D Institutes on Occupational Hygiene and Diseases. The main institute among them is the institute in Kyiv. Nowadays, four institutes like that function in Kyiv, including one in Kharkiv. As one of their research directions, they all study clinics of occupational diseases. Besides, study of occupational pathology is conducted by professors at chairs of medical institutes and universities, as well as institutes on refreshing courses for doctors. These chairs conduct research either independently (Donetsk Medical University), or together with chairs on Occupational Hygiene (National Medical University), or with chairs on therapy (Dnipropetrovsk Medical Academy, Lviv State Medical University and Kharkiv State Medical University).

Peculiarities of Clinical Examination and Diagnostics of Occupational Diseases

Diagnosing of occupational diseases is a responsible and often a complicated business. People with occupational diseases have a number of social advantages, including pension based on medical list, and payment for medicinal drugs. Clinical picture of the majority of occupational diseases, in particular on early stages, does not differ much from the clinical picture of similar forms of non-occupational diseases.

To consider and solve issues on the presence of occupational diseases, the following documents should be present:

1. Assigning of the medicinal establishment with the purpose of medical examination.
2. Extract from an ambulatory card of the sick person (medical and sanitary part, policlinics and dispensary), put together by the doctor, who attended the patient.
3. Sanitary and hygienic characteristics of labor conditions with the description of specific unfavorable factors of the production environment, their parameters, duration of the contact of the patient with them. In case of combined impact of unfavorable factors, it is necessary to make a detailed description of the production process character. A sanitary doctor of the sanitary and epidemiological station, who carries out state sanitary supervision of the object, where the patient works, puts sanitary and hygienic characteristics of labor conditions together.

Extract from a Work Record Book of the patient, which would prove his/her work term at the enterprise, where occupational disease appeared. The extract should be authorized by the Human Resource Department at the enterprise where the patient works. When diagnosing occupational diseases, especially

on early stages, special clinical and functional, as well as biochemical, immunology, radiological and other methods of examination are very important.

Starting with the examination of the patient, first of all, it is necessary to find out what hazard production factors could have or still have unfavorable impact onto his/her health condition in the work process. It is necessary to study thoroughly the documents, the patient brought with him. The assignment for hospitalization should state the reason of hospitalization, and information on the disease progress before the hospitalization.

Particular attention should be paid to the ambulatory health history record of the patient, where the following should be given:

• all diseases, which were previously encountered, including professional ones;

• time of their appearing with the consideration of work under conditions of unfavorable factor, acting of the production environment;

• information on the health state based on results of previous and periodical medical examination;

• clinical picture of the present disease;

• results of the conducted instrumental and laboratory research;

• contents of the conducted treating and its efficiency.

Sanitary and hygienic characteristics of work conditions should include the information on hazard production factors and their intensiveness, results of measured on the containing of toxic matters, dust, noise parameters, and vibration.

When examining patient with occupational diseases, it is necessary to pay close attention to targeted questioning. Patients should have an opportunity to give a detailed narration on hygienic and sanitary work conditions, life conditions, and disease progress. During the examination, it is necessary to keep to requirements of medical deontology, remember about responsibility to the patient, necessity of attentive care about him/her as well as keeping medical secrecy.

Filling in the Medical History Record for a patient with an occupational disease has specific peculiarities. In the history of disease, the following information should be present:

Passport/ID part: Filled in when a patient arrives to the clinic. It should contain the time of the patient coming, his/her last and first names, age, profession, current work place, what medical institution assigned the patient to come to your clinic and what the diagnosis is.

Occupational disease history/anamnesis: It should start with consecutive list of professions of the patient during his/her career until the moment of visiting the doctor or work termination. After a brief chronological listing of main professions, character of the executed work, as well as the main profession and total work term in unfavorable work conditions.

It is necessary to clarify intentions of the patient regarding continuing work under hazard conditions, as well as where he/she could work after the connection of the disease and work conditions is stated.

Sanitary and hygienic characteristics of labor conditions should include the following:

1) detailed description of work, which was carried out by the patient himself/herself due to peculiarities of the technological process (the so-called "detailed" profession);

2) presence of unfavorable production factors (long uncomfortable body position during the work, noise production, vibration, conduct with dust and toxic matters), category of production;

3) character of individual and collective means of protection implemented at the enterprise (ventilation, sealing-in degree of production processes, utilization of protective uniform, and respirators);

4) working day duration, lunch break, place where lunch is eaten, provision of additional free meals;

5) timeliness of vacations, its duration and its actual utilization, as well as additional vacation and its term; and

6) average salary of the patient.

Occupational anamnesis as well as sanitary and hygienic characteristics, written from the words of the patients, should be completed with the study of sanitary and hygienic characteristics of the work place, which is put together and signed by a doctor on occupational hygiene and is an official document, which confirms the possible impact of specific occupational hazards onto the organism of a worker.

Complaints of the patient: At this stage, questioning the patient should be purposeful. After a list of complaints, each of them should be clarified and worked out in detail. At first, they describe complaints, which refer to the main disease, and then accompanying pathology.

History of the current disease: It is necessary to question the patient in details regarding the beginning and progress of the disease, comparing its development with the work character at the enterprise. It is also necessary to clarify: if a patient was suspended from work in their occupation and for what term; what treatment and/or prevention measures were taken and how effective they were; if the patient went to a resort house to be treated there; if he/she was given disablement certificate (provide the group and character) and what occupation he /she got after then; if the disease had group character (especially, with acute intoxication), if such diseases were encountered before among other workers; if the disease was found during one of the obligatory medical examinations or if the patient addressed to the doctor himself/herself.

It is also necessary to clarify the general anamnesis of the patient: if there were indications of diseases until the beginning of wok under hazardous conditions, if there were periods of exacerbation or complications; what are the results of examination of the patient in other medical institutions. Life history: It is necessary to find out how the patient developed in his/her childhood, what was the material situation, at what age and where he/she started his/her working activity, and where the military service took place (if applicable); what life conditions are now; what previous diseases he/she had and heredity. For correct evaluation of clinical results, it is necessary to clarify what was the patient's health condition before the work started at the given state of production; if he/she had diseases, which increased the sensitivity of the organism to unfavorable action of production factors.

Results of objective examination of a patient. General examination is carried out purposefully in the order developed by therapeutic clinics. However, examination in occupational pathological hospital has its peculiarities. It is necessary to pay attention to those systems, which are most sensitive to the action of specific hazard factors and make an attempt to specify symptoms and syndromes, characteristic to a corresponding form of an occupational disease. For example, fits of whitening of fingertips can be seen among people who work in the conditions of vibration, indications of hemorrhagic syndrome can be met among patients who work closely with aromatic hydrocarbon, "lead colic" can be among those who are in contact with lead and this compounds.

It is necessary to consider that specific difficulty appears at exposure of initial clinic disease indications, which are not always specific.

In such cases, it is necessary to consider patient's examination results in the dynamics with the utilization of additional examination methods.

Preliminary diagnosis: Grounding the diagnosis starts with the analysis of sanitary and hygienic characteristics of work, which is or was carried out and its comparison with the data of the professional anamnesis, complaints of the patient, disease anamnesis and patient's life anamnesis.

During the consideration of objective data, it is necessary to pay attention to a complex of symptoms or syndromes, which reflect changes of various organs and systems that are the most characteristic for the envisaged professional pathology. Also those symptoms should be assessed, which do not have significant impact onto the condition of the sick person, but are important ("radical") for diagnostics, for example, "lead" and "mercury" edging of gums, or "lead" color.

To prove the diagnosis, it is necessary to prescribe additional clinical, laboratory and instrumental methods to examine the patient.

Data after additional laboratory and instrumental examination methods: Important results are provided by clinical and instrumental methods of research, which are often decisive for early diagnostics of professional disease. Research of functions of external breathing, lung radiography, and if necessary, bronchoscopy in case of occupational disease of respiratory organs (black-lung disease, toxic pneumosclerosis, and dust bronchitis); exposure ofpain (algesimetery), vibration (palestesiometery) and temperature (termometery) sensitivity with vibration diseases, as well as electromyography - with affection of nervous and muscle apparatus - are widely used.

A number of toxicological and clinical-biochemical research has particular specifics. Thus, exposure of the very chemical agent into biological environments, which caused the disease or products of its metabolism, can be used as one of the indications to diagnose an occupational disease (poisoning). When diagnosing such peculiarities of poisoning, as the ability to accumulation, creation of depots, ability to combine actions wit other chemical or physical factors, which lead to the change of clinical picture of intoxication, presence of the hidden period, possibility of the development of diseases in many years after the termination of the contact with occupational hazards (e. g. late silicosis, or cancer of urinary bladder).

When examining a patient, it is necessary to pay particular attention to the increased sensitivity of some chemical and biological factors of the production environment. In such cases, utilization of skin and inhalation sampling with the consideration of the possibility of sensibilization of the organism to these matters in case of their repetitive action (e. g. chromium, Ursol, or products of organic synthesis) is particularly important.

Final diagnosis and its grounding. To make diagnosis of an occupational disease (intoxication), it is necessary to complete thorough analysis of the work activity of the patient throughout his/her whole life. It is necessary to determine work duration under conditions of possible influence of unfavorable factors of the production environment, as well as types of these factors (chemical, physical, biological, or production dust).

It is necessary to have detailed understanding of specific sanitary and hygienic conditions of work at work place, where the patient counters the impact of unfavorable factors.

Examination results are grounded based on the analysis of clinical examination data and general symptomatology of the disease - questioning of the patient, physical methods of examination, laboratory and instrumental methods of research.

Here, those symptoms and syndromes can be distinguished, which are encountered in the clinical picture of the envisaged professional disease in the examination of the patient.

Differential diagnosis of non-occupational diseases is carried out, if they have similar clinical manifestation.

Conclusions regarding the reason of the disease: After the diagnosis has been made, it is necessary to make a conclusion regarding the reason of the disease (occupational or non-occupational).

To determine the ability of the patient to work: if the patient is capable to work or not at the current occupation if keeping to specific conditions;ability to work is limited - to show what types of occupation can be carried out by the patient; the patient is unable to work and needs specialized assistance.

Besides, it is necessary to provide recommendations regarding rehabilitation, treatment and disease prevention.

Doctor and labor expertise. Social and clinical conclusion is the basis for decision making by Doctor Consulting Commission (DCC) or Medical Social Expert Commission regarding the type of work ability disorder, disorder level and the character of disablement of the given patient.

After the patient leaves hospital, the conclusion is sent to the medical establishment that assigned the patient.

The list of occupational diseases (Appendix 1), approved by the order of the Ministry of Health of Ukraine and the Ministry of Labor of Ukraine, dated February 2, 1995 (No. 23/36/9), is the main document in compliance with which, the diagnosis of the occupational disease is made, as well as the connection with

completed work or profession is done, issues on work ability expertise, medical and work rehabilitation, reimbursement by enterprise owners, establishments or organizations or agencies empowered by them to employees for the caused harm when fulfilling labor obligations are carried out.

The list includes occupational diseases, which appear only due to unfavorable production and professional factors (black-lung disease, vibration disease, intoxication, etc), as well as a number of diseases, the development of which is connected with the influence of specific unfavorable production and professional factor and only a clear influence of other non-occupational factors, which cause similar changes in the organism (bronchitis, allergies, cataract, etc).

It is necessary to remember that in the corresponding lines of the given document only an approximate list of enterprises, manufacturing enterprises and work done there, as well as etiological factors, which can cause disease, is provided there. Occupational diseases also include closest and distant consequences of occupational diseases (e. g. stable organic changes in the central nervous system after intoxication by carbon oxide). It is necessary to consider the possibility of the development of occupational diseases in long term after termination of contact with hazard factors (late: silicosis, papilloma of urinary bladder, etc). Occupational diseases can also include such diseases, which developed based on the occupational disease (e. g. lung cancer, which appeared with the patient with black-lung disease or dust bronchitis, and should be considered as an occupational disease, which is proved by histological changes in the mucus tunic of bronchi - diffused metaplasia with elements of displasia and development of epidermoid cancer, as a rule).

If occupational disease causes worsening of the development of non-occupational disease, what led to the loss of ability to work, it can beeconsidered occupational (e. g. progressing of a form of arterial hypertension, which appeared on the background of vibration disease). Diagnostics of acute poisoning include:

• clinical diagnostics based in the given anamnesis, results on examination of the event place and study of clinical picture of the disease to establish specific poisoning symptoms;

• laboratory toxicological diagnostics, quality and quantity definition of toxic matters in biological environment of the organism (in blood, urine, or cerebrospinal fluid);

• pathomorphological diagnostics, definition of specific posthumous poisoning indications.

Diagnosis of acute occupational disease (intoxication) can be stated by a physician of any medical establishment after obligatory consultation with a specialist on occupational diseases and a physician on occupational hygiene of a territorial sanitary and epidemiological station (SES). Acute occupational disease (intoxication) appears suddenly, after one time impact of relatively high concentration chemical matters (for not more than one shift), which is in the air of the work zone, or levels or dozes of other unfavorable factors. The connection of acute infectious diseases with the occupational activity of the victim in case of necessity to clarify specialized departments of hospitals, clinics of scientific and research institutes on occupational hygiene and occupational diseases after the obligatory consultation with physicians on occupational hygiene and epidemiology of the SES. Professional etiology of acute contact dermatitis can be established by a doctor of skin and venereal dispensary on the agreement with a territorial SES.

Diagnosis of chronic occupational disease (or intoxication) have the right to state first of all specialized treatment and prevention establishments of Ukraine: Donetsky R&D Center for Occupational Hygiene and Prevention of Traumatism, Institute of Medicine of Labor of the Academy of Sciences of Ukraine (Kyiv), Kryvorizky R&D Institute of Occupational Hygiene and Diseases, Kharkiv R&D Institute of Occupational Hygiene Institute on Medical Radiology, Ukrainian R&D Institute of Ecohygiene and Toxicology of Chemical Matters (in case of utilization of means of protection of agricultural plants), Donetsk Regional Specialized Clinical hospital on occupational diseases, Department on Occupational pathology of Lviv Regional Hospital, Department on Occupational Pathology of Cherkassy Regional Hospital, as well as the clinic of the Institute of Health named after Medved. Diagnosis of chronic

occupational diseases should include its name, main clinical syndromes of affections, degree of affected organ function disorder.

Research and registration of professional diseases should be carried out based on the "Regulations on Research and Registration of Accidents, Occupational Diseases and Damages at Enterprises, Establishments and Organizations", approved by the Cabinet of Ministers of Ukraine, No. 623, dated August 10, 1993.

In compliance with these documents, all first exposed chronic occupational diseases and poisoning are subjects to investigation and research. A notification should be compiled on each victim of an occupational disease by clinics of research R&D institutes on occupational hygiene and diseases, specialized departments of regional (city) hospitals, which have the right to establish final diagnosis of occupational disease.

During three days after the final diagnosis of an occupational disease is made, a notification should be sent to the enterprise where the patient works, as well as to sanitary and epidemiological station and medical establishment where the enterprise is serviced.

An owner of the enterprise should organize investigation of reasons on each cease of the occupational disease during seven days since the moment of reception of a notification of occupational disease. Investigation is carried out by a commission, which is appointed by the decision of the leader of the sanitary and epidemiological station. It should include: an officer of the sanitary and epidemiological station (head for the Commission), representatives of a trade union, a work collectives, medical establishment, as well as a specialist on occupational pathology of the local agency of the Department of Health and the owner of the enterprise. Representatives of the Ministry and other central governmental agencies, to the sphere of which the enterprise belongs, local agencies on public supervision of labor protection of and state executive powers, as well as specialists of R&D establishments and educational establishments of the Ministry of Health of Ukraine can participate in the investigation.

An owner of the enterprise should provide the investigation committee with the data on laboratory research of hazard factors of the production process with instrumental measuring of their meaning, necessary documentation on the process (technological requirements, regulations and normative acts regarding the safety), to provide the commission with the facilities, transportation and communication means, to organize publishing and copying of materials to be disseminated.

The investigation commission should compile a program on investigation of circumstances and reasons of an occupational disease, put together an investigation act with suggested activities to prevent the development of an occupational disease, provide normalization of labor conditions, as well as determine the responsibility of the enterprise and officials for the occupational disease appearing and development. The investigation commission should evaluate hygienic conditions of labor of the patient based on materials of the early conducted attestation of work places, results on examination and research, and if necessary laboratory research of hazard production factors with documentary measuring of their meaning. Itshould analyze present medical documentation: ambulatory cards, disease history, conclusions of establishments, orders of agencies on federal supervision on the labor protection, etc. It has the right to receive written explanations from officials and workers on issues connected with the investigation of occupational diseases. The investigation act on occupational diseases is compiled by the commission in five copies within three days after the termination of the investigation, which are sent to the patient, enterprise, where the occupational disease took place, medical establishment, which services the enterprise and the trade union, the member of which the patient is. One copy of the act stays at the sanitary and epidemiological station to analyze and control the intended measures. Investigation acts are kept at the SES for 45 years, in the rest of organizations they are kept for two years.

Based on the results of investigation of appearing of occupational diseases, an owner of an enterprise should make an order with measures on prevention of occupational diseases whose fault was in violation of

sanitary norms and rules, which caused occupational diseases. When realizing measures on prevention of occupational diseases, suggested by the investigation commission, an enterprise owner should inform the SES within the terms stated in the act.

Registration and records of people, who were among the first to have occupational diseases, are conducted in specialized record books in compliance with the form, approved by the Ministry of Health, which should be filled out:

• at enterprises and establishments on sanitary and epidemiological service based on notifications about occupational diseases and acts of their investigation;

• at medical institutions based on the medical record of a patient, extracts from the medical history, doctor's conclusion on the diagnosis after examination at the hospital, as well as notification about occupational diseases.

Using the data of disseminated acts on occupational diseases, the SES compiles special record cards for keeping and analyzing occupational diseases with the assistance of computers. These compiled cards should be saved for 45 years in the SES.

In compliance with the documents, investigation is carried out on every acute occupation disease and poisoning. A witness, employer, who found out about each case like this, or the victim him/herself should notify the work coordinator, foreman or any other authority of the enterprise and provide first aid medical assistance. In his/her turn, the leader should do the following: organize medical aid to the victim immediately and deliver him/her to a medical establishment, also notify the enterprise owner about the accident; keep the original state of the work place and equipment until the investigation commission arrives, also take measures to prevent similar cases. A medical establishment should notify the leader of the company about each accident like this, which was registered at the company where the victim works within one day, and in case of acute occupational poisoning (disease), they also should notify a sanitary-hygienic station, by sending urgent notification on each victim. The same type of notifications is sent to the owner of the company/enterprise to take urgent measures to prevent further similar accidents. A medical establishment, which clarified or changed the diagnosis of an acute occupational poisoning (disease), compiles an urgent notification and sends it to the sanitary and hygienic station which coordinates the enterprise the victim works at with changed (clarified) diagnosis, as well as its date, within 10 hours.

Upon receiving the notification about the accident, the company owner appoints a commission on investigation, which includes a head (specialist) of the labor protection service of the company (chairman of the commission), head for structural subsection or chief specialist. The commission should also include a representative of a trade union, member of which the victim is, and in case of acute occupational poisoning (diseases) - a specialist of the SES. If the victim is not a trade union member, then an authorized representative of a work team on labor protection should be in the structure of the commission.

Within three days since the moment of the event, the investigation commission is obliged to do the following:

• to examine the accident place, interrogate witnesses and those involved in the accident, and to receive explanations from the victim, if applicable;

• to consider the correspondence of labor conditions and production means on the project and passports, and also keeping to requirements of normative and technical documentation on operation of equipment and normative acts on labor protection;

• to find conditions and reasons of the accident; to determine responsible for the accident; and also to develop measures to prevent cases like this;

• to fill in an act, form H-1, in five copies, where fault for the accident of an establishment, victim or a third party should be stated; the copies should be sent to be approved by an enterprise owner.

Within a day after the end of the investigation, an owner should confirm all five copies of the act, form H-1. The act is sent to a victim or to a person who represents his/her interests, a foreman or another

structural subsection, where the accident took place, to implement measures regarding prevention of similar accidents, to the state inspector on labor protection, trade unions of the enterprise where the accident took place, the head of the sector on labor protection, to who the act is sent together with other materials on investigation. A copy of an act, form H-1, in case of acute occupation poisoning (disease) is to be sent to the SES as well. The SES fills in Records on Occupational Poisoning: (Disease) for recording and analysis based on the act, form H-1.

Organization and Conduct of Medical Inspection of Workers

Medical assistance to patients with some occupational disease is provided by clinics, parts of R&D Institutes, as well as medical and sanitary sectors (MSS). The main task of the MSS is to develop and conduct measures, targeted at improvement of labor conditions and life of the employees and officers, prevention and decrease of total and professional disease, provision of specialized medical assistance and realization of systematic dispensary care together with the administration of establishments.

As until now, amount a list of doctors, there has been no physician on occupational diseases, all the work on servicing the patients with occupational diseases were carried out by general practitioners (workshop general practitioner). Nowadays, when such speciality became officially known, occupational pathological service is at the stage of development.

To prevent occupational diseases, preliminary and periodical medical examinations of workers to start their career or those who work in close contact with hazard factors of the production environment. The order of their conduct is regulated by the Order of the Ministry of Health of Ukraine No. 45 dated of March 31, 1994 "About Approving Regulations on the Order of Conduct of Medical Examinations of Workers of Specific Categories". The order envisages that the organization and medical examinations will be provided by:

- an owner of an enterprise, establishment, organization independently of pattern of ownership and types of activity;
- establishments and agencies of the Ministry of Health of Ukraine, treatment and prevention, sanitary and hygienic, R&D and medical institutions (universities), on the territory where enterprises, establishments, organizations, agricultural companies, agrofirms, rental, cooperative, small, joint venture companies; catering, industrial, and children's sites, elementary schools and other objects.

The owner of the enterprise finances medical examinations, reimburses losses on care, occupational and medical rehabilitation of people with occupational diseases, examination of specific labor conditions to put together a sanitary and hygienic characteristics.

Preliminary medical examinations are dune with the purpose to state physical and psychological ability of a person to work in the specific area, speciality, position, when first admitted to work; as well as to prevent diseases and accidents, exposure of diseases (infectious and others), which threaten with contagion of workers and manufactured products, as well as admission to work for people under 21. Periodical medical examinations are conducted in the process of labor activity for people, who are engaged in hard work, work with hazard or unsafe conditions in compliance with the list of matters, unfavorable production factors and works to implement which, obligatory medical examinations of employees should be carried out. They provide dynamic examination of the health state of workers, expose early signs of the impact of production conditions and hazards onto the body, and which do not give the opportunity to continue working on the given profession: prevent accidents, expansion of infectious and parasitic diseases. They can be conducted within the period of staying of an employee in the hospital or in cases when he/she asked for help.

Results of medical examinations should be drawn up within a month in a relevant act, which is made in four copies (for a medical establishment, owner, trade union committee and sanitary and hygienic station).

During the medical examination, examination in occupation pathologic centers, clinics of R&D and medical institutes (universities) to specify the diagnosis or to determine a role of production factors in the development of diseases, a job and average salary is kept for a worker.

The order of organization of medical examinations:

The enterprise shall:

• determine a contingent of people, subject to periodical medical examination; and draw up a list of them in two copies with last and first names, and then approve it at the sanitary and hygienic station. One copy is sent to the medical establishment, and the other stays at the enterprise (with the organization responsible for medical examination) together with the sanitary and hygienic station and a trade union;

• assign people, who are sent to the enterprise or to change profession and work place to take preliminary medical examination in compliance with a proper document;

• introduce a new-comer to the hazard and dangerous production factors and matters, as well as normative acts on labor protection, which are specific to the profession;

• finance medical examination, and reimburse costs on examination and treatment of workers at occupational pathologic institutes, medical institutes (universities); as well as examination of work conditions with developing sanitary and hygienic characteristics;

• make an order to conduct medical examinations within the terms, approved by the treatment and prevention establishments, appoints responsible for organization of medical examination;

• assist the creation of improvement of material and technical bases of medical sectors, and medical establishments to conduct medical examinations, clinical and other research;

• apportion premises to conduct medical examinations; assign workers to have medical examination in medical establishments and controls the term of its implementation;

• provide implementation of the recommended rehabilitation and prevention measures.

A medical establishment shall:

• make an annual order to create commission to conduct medical examinations with a fixed term, place of conduct and list of physicians, clinical and other research; the commission will be chaired by a vice chief physician of the medical establishment, which trains doctors in occupational pathology;

• develop a plan - schedule of medical examination of workers, which should be approved by an owner and sanitary and hygienic station;

• conduct medical examination of employees, as well as clinical and other types of research;

• involve other specialists to participate in the medical examination; conduct additional clinic research, necessary to assess the health state of employees if applicable;

• inquire sanitary and hygienic characteristics of labor conditions of employees at sanitary and hygienic stations;

• control keeping to the terms of medical examination;

• make conclusions on the health state of each employee, who underwent a medial examination; make a decision regarding medical contraindications as to the possibility to continue work on the profession for those who have general or occupational diseases;

• inform an employee as to the state of his/her health, as well as the possibility to continue work on the profession based on the results of medical examinations or provide conclusions regarding transfer to another job;

• based on the medical indications, send an employee to further examination at medical centers, which have the right to make a diagnosis of an occupational disease;

• assign an employee to meet medical and social expertise commission (MSEC) based on medical indications.

It conducts:

• annual medical examination of people who terminated their work at the enterprise with hazard and dangerous factors, the impact of which can cause late development of occupational diseases;

• analysis and generalizing results on medical examinations, drawing up a final act, which is sent to the territorial SES, owner and a trade union of the enterprise;

• sending out a list with names of those, who are contraindicated to work under unfavorable conditions to an owner within a month after the medical examination took place;

• carrying out dispensary supervision over patients with occupational diseases, who continue their work activity, treatment and occupational rehabilitation;

• keeping records of patients with occupational diseases and poisoning.

A sanitary and hygienic establishment shall:

• determine authenticity of the record keeping made by the owner of hazard and dangerous factors and matters, work with which require medical examinations;

• confirm lists with names of people, subject to medical examination, as well as plans - schedules of medical examinations.

It participates in the following:

• in preparing and training specialists of a medical center; and

• in compiling the final act on periodic medical examination.

• in expert evaluation of the organization and the quality of medical examinations.

It sends decisions as to elimination of exposed violations and drawbacks in the organization and conduct of medical examinations;

• considers issues on temporary termination of medical examination in case of isolation of the existing situation;

• compiles sanitary and hygienic characteristics on work conditions;

• applies with proposals as to prevention of occupational diseases to territorial state administrations.

Research and Development (R&D) institutes on labor medicine, labor hygiene and occupational diseases, as well as chairs of medical universities (institutes) shall:

• develop normative and methodical documents on scientific and organizational principles of conduct of medical examinations, expertise of their quality and evaluation of results; criteria of determination of contingent of people - subjects to medical examinations; indications of the risk of occupational disease development and criteria of determining diseases as occupational ones;

• are engaged in elaborating issues on prevention, early diagnostics and treatment of occupational diseases, medical rehabilitation of workers with the risk of development of occupational diseases and patients with occupational diseases; definition of distant consequences of the impact of hazard and dangerous production agents onto the health. They also carry out refreshing and upgrading courses on occupational diseases in the form of provision of information, training and seminars, and also make final decision as to the connection of the disease with work conditions.

Theme №2. PROFESSIONAL DISEASES OF RESPIRATORY ORGANS, CONDITIONED BY DUST IMPACT

Black-lung disease

Black-lung disease is a respiratory disorder, a type of pneumoconiosis caused by repeated inhalation of coal dust over a period of years and dust depositing in lungs, and is also characterized by the development of diffusion fibrosis.

Classification of dust diseases of lungs changed many times during a long period of time. But at first, dust diseases of lungs were united under the common name "consumption" with giving some specific occupation

of the patient. Later, specific forms of the disease were distinguished, like "byssinosis" or "dust eczema". Then, the term "pneumoconiosis" was introduced (from Greek: "lung" and "dust"), which generalized all forms of dust diseases, which were accompanied by the development of fibrosis process in lungs. For a long time, there was not unified thought regarding the possibility of the development of pneumoconiosis process without inhaling production dust of silicon dioxide, i. e. considered that silicon dioxide is actually a synonym of silicosis. There was not any unified thought as to the existence of dust bronchitis as it is.

Based on modern clinical, radiological and pathological and anatomic data, the dust disease was considered in a wider aspect, what was reflected in its classification as well. Clinical forms of dust diseases are disseminated processes in lungs - silicon dioxide, granulomatoses, exogenic allergy, alveoli disease, which appear under the impact of corresponding types of dust, dust bronchitis, and bronchial asthma.

After introduction of radiological method of lung examination into clinical practice, there was an opportunity for more differentiated approach to determine the presence and character of lung dissemination when the patient is still alive. Thus, particular attention was paid to the development of classification of pneumoconioses as a form of dust diseases of lungs, which are the most difficult to be diagnosed.

As to the evolution of views at the classification of pneumoconiosis, it is necessary to mention that their first classification was adopted in 1930 at the International Conference on Silicosis problems. Then, three stages of silicosis were determined based on mostly radiological picture. In the future, this classification was reconsidered many times (1950, 1958, 1968, etc), it was specified and changed, what let characterize other types of pneumoconiosis, variety of radiological expression of diseases, as well as the presence and character of indications of complications and some accompanying lung diseases. Various radiological and clinical indications inclassifications were coded and marked by symbols. The last variant of the International Classification of pneumoconiosis was approved in 1971.

Home classification of pneumoconiosis, in contrast to international ones, reflected not only radiological indications of the disease, but also the clinical picture, as well as the degree of compensation process. According to the first classification, developed by I. Kavalerov in 1925, the following clinical variants of pneumoconiosis were specified as bronchitis, emphysematous, pleurotic, interstitial cystitis and cardiogenic ones.

So, according to the ***modern classification (2002)*** the following types of pneumoconiosis are distinguished:

1. Silicosis
2. Silicatosis
3. Metalloconiosis
4. Carboconiosis
5. Hypersensitivity pneumonitis (dust diseases)

Taking account of dust aggresivity three groups of dust diseases are distinguished:

I. Dust diseases developing from a dust with high- and medium-fibrogenious influence (containing SiO2 or asbest more than 10%) - a silicosis, silicoantracosis, silicosilicatosis, silicosiderosis, asbestosis.

II. Dust diseases developing from a dust with mild-fibrogenious influence (with the contents free silicon dioxid less then 10%, bonded Si02 or without SiO2) - silicosilicatosis (kaolinosis, talcosis etc.); carboconiosis (anthracosis, grafitosis, pneumoconiosis of coal dust, diamond pneumoconiosis); metalloconiosis (siderosis, kaolinosis, baritosis, manganoconiosis, aluminosis etc.); a dust disease of the electric welders, gascarvers and working other welding professions, polishing, grinding, foundring etc.

III. Hypersensitivity pneumonitis (dust diseases) from a dust (aerosolum) with toxico-allergic influence - berylliosis, pneumonites from influence of chrom, nickel, platinum and others rare-earth metals and alloys; pneumonites from a dust of plastic, polymeric pitches, medicinal preparations (toxico-fibrogenious alveolites - TFA); a byssinosis, bagassosis, papricosis, exogenous allergic alveolitis (EAA) woodworkers, poultrers etc.

The classification also includes indications, which characterize various variants of clinical and radiological picture, functional disorders, complications of pneumoconiosis progress.

Clinical and radiological characteristics of pneumoconiosis are very multi-sided and depend not only on the type of dust, which caused pneumoconiosis. Thus, when making a diagnosis of pneumoconiosis, it is not important only to state the etiological factor - from dust. It is very important for treatment and solution of questions of work ability of patients with pneumoconiosis to know the evidence, form, and speed of progressing of fibrous process in lungs, presence of respiratory compromise and cardiacdecompensation, which accompany the main disease. Based on this, the classification of pneumoconiosis, the following clinical and radiological indications were included, which characterize morphological, functional and clinical peculiarities of various forms of pneumoconiosis.

Radiological characteristics of main indications of coniotic fibrosis of lungs include the following elements: character of shadow (form, outline and size), their expansion, thickness and density. Each radiological indication is coded by a corresponding symbol. Codes of radiological indications are marked with letters from Latin alphabet and Arabic numerals, and stages of pneumoconiosis are marked with Roman numerals. Absence of pneumoconiosis indications on an X-ray photograph is marked with zero (0). If there are some doubts as to the change of lung picture in the way of its some intensification, then when describing the X-ray photograph, it is marked "0-1", what actually means suspicion of *pneumoconiosis* presence. To specify the diagnosis, it is necessary to conduct additional research: increased side photographs, tomograms, as well a repeated radiological examination in 6 to 12 months.

Tuberculosis:

a) with separating forms of tuberculosis (according to the classification)

b) without noting the form of TB (small nodular, big nodular and massive TB-silicon)

Pneumonia

Brochnoectatic disease

Bronchial asthma

Pneumothorax

Atrophic arthritis

Neoplasm As to the character of forms, sizes and outlines of shadows on the radiograms, there are the following fibroses: interstitial, small nodular and big nodular ones.

Interstitial fibrosis on an X-ray photograph is characterized by small change of a lung picture in the form of its intensification and deformation due to the development of perivascular and peribronchial fibrosis, as well as fibrosis of interalveoalr septum and interlobular partition.

Depending on the stage of reflection and localization of fibrosis shadows: linear and cellular (s), heavy (t) and roughly heavy (u).

Interstitial fibrosis of lungs is usually double sided and diffusive. As to the thickness and density of shadows on X-ray photographs can be not much spread (1); much spread, when the picture does not differentiate and there are numerous shadows of irregular forms.

Nodular fibrosis on an X-ray photograph is shown as small rounded shadows, which are conditioned by coniotic nodules. As to the size of nodules, they can be divided into three groups (up to 1. 5 mm (p), from 1. 5 to 3 mm (q), and from 3 to 10 mm (r). Nodular shadows are rounded with clear outlines; their intensiveness depends on dust, which caused pneumoconiosis. Nodular process is double sided, as a rule. As to the number of nodules, there are three categories: small (1), moderate (2), and numerous (3).

Big nodular pneumoconiosis is characterized by the presence of big shadows of rounded or irregular form with clear or unclear outlines on the background of nodular or interstitial shadows on X-ray photographs.

As to the diameter of nodular shadows and the territory, they cover there are A-small nodular variant when nodule diameter is from 1 to 5 cm, with the total territory of spreading - not more than 5 cm2, B - big

nodularprocess, when the diameter of nodules is from 5 to 10 cm, with the total territory of spreading - not more than 1/3 of the lung territory; and C -massive pneumoconiosis, diameter of nodules is over 10 cm with the total territory of spreading - over 1/3 of lung territory.

In compliance with home classification, there are three stages of pneumoconiosis - I, II and III. Main criteria to determine stages, are X-ray indications, though clinical and functional indications are considered as well.

As to the character of pneumoconiosis process, there are the following forms: 1) fast progressing; 2) slowly progressing; 3) late; and 4) "regressing".

With fast progressing pneumoconiosis, stage I, the disease can be diagnosed in 3 to 5 years after starting work with dust, and the acceleration of pneumoconiosis process, i. e. transfer from pneumoconiosis, stage I to stage II, can be seen in 2 to 3 years. This form of pneumoconiosis can include the so-called acute silicosis, which is a fast progressing form of silicosis as it is.

Slowly progressing pneumoconiosis develops in 10 to 15 years after the beginning of work in contact with dust, and with transfer from stage I to stage II, the disease can last for not less than 5 to 10 years.

Pneumoconiosis, which develops in several years after the termination of contact with dust, is called late. Regressing forms of pneumoconiosis can be met only when X-ray contrast dust is accumulated in lungs, which created an impression of more intensified stage of fibrosis in lungs. In case the patient terminates his/her contact with dust, there is partial withdrawal of x-ray contrast dust from lungs. This explains the "regress" of pneumoconiosis process.

Clinical and functional indicators of pneumoconiosis include TB, pneumonia, bronchectatic disease, bronchial asthma, atrophic arthritis, and spontaneous pneumothorax.

Silicosis

Silicosis is pneumoconiosis, caused by inhaling dust with free silicon dioxide (SiO2). This is the most spread form of pneumoconiosis, the progress of which is particularly complicated. The disease got much spreading in the end of the 19th century, mostly due to the development of metal mining industry and machine engineering, where in the process of production, dust is created, which include free silicon dioxide.

Silicosis can be most often met in the following areas:

1) in metal mining industry - among people, who are engaged in mining gold, tin, copper, tungsten and other minerals, which is in the ores with quartz (drill-operators, tunnellers and workers of tunneling teams); in machine engineering among the workers of foundries (fettlers, shakers, etc);

20 on the production of fireproof and ceramic materials - among workers, engaged in production of dinas, fire clay and other fireproof products,as well as repair of industrial furnaces and other operations in metallurgic industry;

3) among workers engaged in tunnel boring, sand grinding, processing and treatment of quartz, granite and other ores, which contain free silicon dioxide.

Changes in lungs due to action of dust take place, as a rule, after a long period of work there. Disease development depends on the amount of dust, which got into the organism content of free silicon dioxide, as well as tendency to the disease. The last thing is very important considering the fact that not all the people who inhale quartz dust will have silicosis provided common work conditions.

The boundary permitted concentration of dust, which contains over 70 % if free silicon dioxide, is 1 mg/m3, from 10 to 70 % - 2 mg/m3 and from 20 to 10 % - 4 mg/m2.

Many research showed that the most pathogenic are the particles of dust with the size less than 5 micrometer as they achieve bronchiole or alveoli, and stay there.

In the development of silicosis, the importance is not only in getting dust into lungs, but also its retention there. At the preliminary stage of fibrosis process, in the result of the fact that the amount of dust, retained in lungs, increases the amount, which can be take out, what creates the so-called "dust depots". Cleaning of

lungs from silica is taken out mostly via bronchitis, but a smaller amount of particles is taken out via lymphatic nodules.

In average, the period from the beginning of action of dust until the development of the disease takes from 10 to 15 years, though under unfavorable conditions of work it can reduce to 2 to 6 years, thus we have the so-called fast progressing silicosis.

This form of silicosis is characterized by a fast progress and rather unfavorable forecast. As a rule, it develops among workers, who work in the most silicosis dangerous professions (tunnellers, polishers of lenses, and before these were sandblasters). They thing that the fast progressing, especially "acute", silicosis with short period of dust exposition (up to six months) develops in case of the action of highly aggressive dust onto young people of asthenic constitution with clear inflammatory process in lungs.

Pathogenesis. Mechanism of appearing and development of silicosis is very complicated and is not completely opened. If to consider this question in historical aspect, then first of all, it is necessary to tell about the so-called mechanical theory of appearing and development of fibrosis process in lungs. From the point of view of followers of this theory, fibrosis changes in lungs are the result of mechanical irritation, microtraumatizing of lung tissue. Soon it was rejected, though even today, mechanical factor is considered valuable in the development of dust pneumosxlerosis. Mechanical theory was later replaced by toxic-chemical theory, which explained the development of silicotic fibrosis by toxic action onto the lung tissue of silicic acid, which appears when gradually dissolving silica.

Further, many hypotheses were suggested as to the mechanism of appearing and development of fibrosis (infectious and piezoelectric), which did not received the necessary support and thus were rejected.

And nowadays, the following two theories are most known: colloid-adsorbing and immunological theories.

The essence of colloid-adsorbing theory is in the recognition of the role of silanol groups of the surface of silica in primary interaction with cellular elements. Thus, pneumoconiosis from the view of the theory followers is considered as chronic disease of lungs, caused by long inhaling of air with high concentration of practically non-dissolved aerosols, which have pathological impact in hard accumulated states based on processes, which take place on the surface of dust particles.

Mechanic destruction of silica is obligatory connected with breaking of many oxygen bridges between oxygen atoms; as in silicon dioxide, four atoms of oxygen surround silicon atoms, and each oxygen atom belongs to two silicon atoms simultaneously. Thus, silica is a continuous link of silica-oxygen tetrahedrons (SiO4)-4.

Thus, with breaking oxygen bridges on the surface of breaking, there are two types of active centers: one of them is unsaturated oxygen atom of a broken pair; the other one is an unsaturated atom of silicon. In the air, under the impact of water vapors, kept in them, and particularly fast in water environment, aquation on the silica surface takes place. Unsaturated oxygen atom is joined with ion of hydrogen, turning it into a silanol group.

An unsaturated atom of silicon attracts hydroxyl group (=Si-)+(-O-H)-. Besides, on the surface of silica break, there is also the third type of active center. This is an oxygen atom of non-broken marginal siloxane bridge.

Researches show that particular cytotoxicity of silica is conditioned by all active centers of the surface of dust particles, capable to create hydrogen links, but silanol groups have the most important meaning. The proof of the important role of these groups in the considered process, there is immediate depressed biological activity of silica at their substitution with inert methyl radicals.

Influence of silica and other fibrous dust particles onto a human organism is done in three stages. The primary and obligatory link in the total chain of pathological changes, observed in lungs when inhaling with fibrous dust, is extensive activity and damage of macrophages with ingestion dust particles. At the second stage, vital products of activated macrophages and matters, which are emanated at the destruction, stimulate

fibroplasts and extensive synthesis of collagen in respiratory organs. Besides, it is necessary to remember that the interaction of dust particles with phagocytes creates free radicals of oxygen, which also cause destruction of lysosomal ferments of phagocytes.

Making conclusions, it is obvious that the primary action of fibrous dust, has significant impact onto the character of pathological developing changes is grounded by on the one hand, mechanism of interaction if the surface of dust particle with the membrane of phagocyte, and on the other hand, by the peculiarity of activation of its oxidase metabolism, and besides, by the ability of the marginal layer of particles to catalyze reactions of transformation of active forms of oxygen.

Immune theory can be well connected with the colloidal-adsorption theory. Significant place in the mechanism of a corresponding reaction onto the dust action is in the interaction: antigen - antibody. Certainly, an antigen with silicosis is a structurally changed protein of coniophagus cytoplasm. After the destruction of coniophagus, particles go beyond the cell environment, and thus the release of structurally changed protein takes place, which is capable to impact the human organism as an autoantigen. Released particles again go through phagocytosis with further destruction of coniophagus, what conditions continuous creation of autoantigens, which stimulate the antibody production. There are also ideas that with the destruction of macrophage dust particles in the result of phagocytosis, liposaccharide complexes are released, which are capable to activate the creation of antibodies non-specifically.

Thus, silicosis can be considered as a disease of non-specific immunogenesis, where phagocytosis and the destruction of coniophagus is an obligatory condition of the development of silicotic reaction.

There is a supposition that lipids or products of their re-oxidation, which are released from destructed dust cells, possess fibrous activity themselves. In the fibroblasts under their impact, oxidation of amino acid of praline into oxiproline takes place, which is considered an important link in the pathological collagen creation.

Lately, works appeared, where the role of fibronectin in the pathogenesis of silicosis is considered. It was shown that fibrogenic dust stimulates synthesis and release of fibronectine by leukocyte and lung macrophages, securing their aggregation and adhesion when forming silicotic granulomas. Besides, in the pathogenesis of silicosis, an important role is played by biologically active matters of tissue basophiles. Based on this position, silicosis is a particular variety of the inflammation - chronic granulomatous inflammation.

Pathological and anatomic picture: With silicosis, changes take place not only in lungs, but also in upper respiratory ways, bronchi, pleura, lymphatic glands and lung vessels. In the mucous tunic of scroll-bones, larynx and trachea, subtrophic, and later atrophic and sclerotic changes can be seen. The mucus tunic of larynx and trachea are thickened and plethoric, and in the submucuous space -hyperplasia of glands with the expansion of their outgoing flows, which are full with mucus and exfoliated epithelium.

At the early stages of silicosis, there are areas in lungs with deposits of dust particles, mostly around bronchi, vessels, and in lumens of alveolar ducts and in alveoli. In these areas there are precolagen and collagen fibers, which are a primary stage of forming of silicotic nodules. Together with it, there is a weakly outlined sclerosis in the form of outgrowing of conjunctive tissue around bronchi and vessels, as well as in alveolar septum.

Diffusive - sclerotic (interstitial) form of silicosis is characterized, first of all, with the presence of numerous bars of collagen tissues and marked sclerosis around bronchi, vessels and in interalveolar septum. Often there is marked emphysema of lungs. Silicotic nodules are absent, but with time in those areas, real silicotic nodules can appear.

Nodular form of silicosis can be met much more often and is characterized with the presence of concentrically located, practically hyalinized fascicles of conjunctive tissue, which look like silicotic nodules. As a rule, they are round or oval, of grey or grey - black color. Silicotic nodules are mostly placed in alveoli, and can be met in peribronchial and perivascular lymphoid nodules.

Patients with silicosis mostly complain on three things: pain in chest, dyspnea and cough.

Complaints on pricking in the chest, mostly in scapula area and underneath them have non-permanent status on early stages of silicosis. Clinical character of pain proves their pleural origin, microtraumatizaton of pleura and the development of conjunctive tissue reaction on deposited sand in them. Along the development of silicotic process, commissures of interlocar and visceral pleura, as well as sclerosis of subpleural tissue, which disturb free smoothing of lung tissue and conditions the feeling of pain, are created between them.

Dyspnea is considered one of the main symptoms of silicosis. Complaints on it appear already on the early stage of silicosis, at first during the work, what proves secret insufficient breathing. Only at the later stage of the disease, patients have dyspnea even at insignificant physical activity, but it is rarely observed in rest (complicated form of silicosis). Dyspnea is conditioned by many mechanisms: spasms of small bronchi and bronchioles; afterwards, the increase of dyspnea can be explained by progressing fibrosis and emphysema, which limit breathing surface of lungs together with cardiovascular collapse.

Dry cough or with small mount of mucoid viscous sputum starts causing problems to patients with silicosis on the early stage of the disease aswell. In lungs, disseminated crepitation, sometimes, fine moist or subcrepitated rales on a lower part of lungs can be heard on the background of vesicular or heavy breathing. These auscultated data correspond to the appearing of primary cataract reaction on dust in the bronchial tree, which can expand from trachea to the smallest bronchi and bronchioles.

Then (second and third stages of the disease), dry rales can be ausculated or not.

With the development of process, catarrhal reaction is replaced by atrophic and degenerated changes, which are accompanied by the damage of epithelium and its desquamation. These can explain the lack of clinical symptoms of bronchitis with silicosis.

Emphysema is a usual companion of silicosis. At first, increased lung pneumatosis develops, which can be seen in the change of percussion sound, mostly in lower parts of lungs, in some prolapse of lung ends with keeping their good motion. This state can be evaluated as refectory reaction onto the fibrosis process, which is in the opening of alveoli. Further, a real emphysema develops, though the chest percussion does not show clear box sound above the total area of lungs even then as it happens with other serious forms of emphysema.

Microtraumatization of mucous tunic of the respiratory apparatus by dust particles causes reactive inflammation. Clinically this state is shown mostly in rhinitis, as nasal cavity is affected by dust factor more than the areas, located in lower, like gullet, larynx, and trachea, for which it serves like a filter. Subjectively the sick are concerned with the feeling of "stuffed" nose, sometimes "scratchy" throat, light throat soreness and coughing.

Besides problems with respiratory organs, patients with silicosis have problems with cardio-vascular system as well, though this problem is not obvious clinically for a long time. Patients do not usually have complaints on that. Heart borders are not changed. Tones are clear and rhythmical in the majority of cases. Progressing of silicotic process, and development of lung emphysema causes narrowing of vessel duct with the development of hypertensia in the system of lung artery, increase of stress onto the right ventricle, and its hypertrophy, and then its widening. Arterial pressure is within norms for a long time, but it has tendency to decrease.

Marked changes in periphery blood at non-complicated silicosis are absent. There can be inclination to the increase of the number of erythrocytes and hemoglobin, and moderate leucopenia, mostly conditioned by decrease of lymphocyte content. With progressing of silicon process, amount of leukocytes increases due to appearing of inflammation. ESR increases, what can be caused by the change of protein content of blood serum to the increase of the level of coarse-dispersed fractions, in particular y-globulins.

Paramount affection of respiratory organs with silicosis conditioned the necessity to research functions of external breathing. The most informativeare such indications as vital capacity of lungs (VCL),

its relationship to the corresponding forced VCL, FEV1 and its correlation with VCL (Tifno test), the data pneumotachometria (in particular, the exhalation power), as well as determination of residual volume of lungs).

VCL of patients with silicosis has the tendency of decrease along the progress of fibrosis process. However, in the primary stages, it can stay at a normal level for a long time. Decrease of VCL is conditioned particularly by the reduction of breathing reserves, particularly of additional air. FVCL indications and pneumotachometria reduce as well, particularly in cases of bronchitis manifestation, what shows the disorder of bronchial permeability.

In the whole, for the silicosis, it is characteristic to have moderate expression of restrictive - obstructive type of disorder of external breathing.

Correspondingly to the existing classification, there are three clinical and radiological stage of silicosis.

Stage I. Patients complain to have dyspnea when having much physical activity, pain in the chest without clear localization, variable dry coughing. Objectively determine indications of basal emphysema, auscultatively - stiff, in some places, vesicular breathing is somewhat weak.

The radiological photograph of lungs at silicosis of Stage I shows double-sided increase and deformation of lung picture, moderate carnification and the change of the structure of lung roots. In case of nodular forms of silicosis on the background of changed lung picture, there is a small amount of punctuate shadows of medium intensiveness, from 1 to 2 mm in size, located mostly in lower and mid lung. Interlobar pleuron to the right is often thickened. On Fig. 1, radiological pictures of normal lungs and lungs with pneumoconiosis of stage I are compared.

As to the function of external breathing, there is moderate compensatory hyperventilation on the background of normal or even some increase of VCL indications.

Stage II. It is characterized by the intensification of dyspnea, pain in the chest and cough. These complaints become permanent. Objective examination shows limitation of the motion of the lower end of lungs, as well as reduction of excursion of the chest.

For silicosis, stage II, more marked intensification and deformation of lung picture. A number of nodular shadows is increased also, size of which achieves from 3 to 10 mm. Sometimes, there is obvious tendency to the joint of nodular shadows. Lung roots are expanded, carnified and start looking "cut". The pleura can be thickened and deformed. Radiological picture starts looking like disseminated miliary tuberculosis ("snow storm").

From the point of view of external breathing, lung vital capacity reduction takes place on the background of the increase of minute volume.

Stage III. It is characterized by dyspnea in rest, intensive pain in the chest, coughing with phlegm discharge, and possible fits of asphyxia. Above the lungs,alternation of sectors of box sound with dull ones can be palpated; and with auscultative methods, it is possible to observe the alternation of breath weakening and rough ones.

Radiologically, at the silicosis of Stage III, massive shadowings are created on the background of changes, which are characteristic for Stage II. Besides that, marked pleurodiaphragm and pleurocarcial commissures, as well as buliosnic emphysema can be observed.

Three clinical and radiological forms can be defined: nodular, interstitial and tumor. It is necessary to determine that for the impact of dust with free silicon dioxide onto a human organism can be characterized by the development of the nodular form of the fibrosis process. A tumor form can be observed with patients with silicosis of Stage III, when all possible disease syndromes in connection with the marked functional disorders are present. Clinical pattern of "acute " silicosis develops into two phases. The first (latent) phase is characterized by the process of fast developing of dyspnea and cyanosis; patients lose weight and have

fit-like coughing; and it is particularly characteristic for "acute" silicosis to have marked dyspnea and cyanosis.

Insignificant emphysema, a big number of rales (mostly in lower parts), acceleration of ESR, quick increase of the amount of P- and a-globulins, and positive tuberculosis sampling determine this form of silicosis.

Radiological pattern of acute silicosis is variable: from nodular ones with small clusters, which are located mostly in lower parts, to tumors due to merging of some elements, in particular in lower parts as well as development of pleural growing.

Late silicosis develops in several years after the termination of contact with dust, which contains quartz. The process of the disease is severe. There are assumptions that this form of silicosis is conditioned by the presence of "depots" of quartz dust, which then is transferred by phagocytes to various portions of lungs.

Differential diagnostics: To differentiate silicosis is possible with many disseminated processes in lungs, which often stimulate dust pathology, and in particular among people, which work in contact with dust. That is why anamnesis of those occupied with dust professions should be taken very carefully, taking into account professional route, work conditions, harmful habits, previous diseases process and development of the found pathology, and radiological changes together with the clinical pattern. Often there is the necessity to involve auxiliary research instruments - tomography, bronchography, bronchoscopy, biopsies, specific sampling, etc. to be able to make a correct diagnosis.

It is necessary to remember that very often it is necessary to differentiate it with the lung tuberculosis, in particular, in cases of hematogenous -disseminated tuberculosis, which can develop in the form of acute miliary and chronic tuberculosis. With lung form of acute miliary tuberculosis, the main criterion is the clinical pattern. Unlike silicosis, the disease starts with the acute form, and is accompanied by the increase of temperature, and in some cases it has a hectic character with profuse sweating. Dyspnea is one of the constant and heavy symptoms. It is so acute that patients cannot sleep, talk, or lie because of it. That is why this form is called "asfiphyxic form of acute tuberculosis". Dry heavy coughing, often in the form of fits, is characteristic for the disease. In the beginning of the disease, objective changes are absent. A patient's face is pale; cyanosis of lips and cheeks is marked. Discrepancy between hard dyspnea and cyanosis, on the one hand, and absence of auscultative changes, on the other hand, can be observed. Then, bubbling subcrepting rales appear, which can go before dry ones.

It is characteristic for it, that the radiological photograph of the chest for the first week of the disease, with marked dyspnea and cyanosis, does not showany clusters. Only during the second week, leveled rush of miliary clusters on both parts of lungs can take place, which are not bigger than a millet grain in size. Such clusters are most typical, but they can be bigger. Density and intensity of shadows, like with silicosis, are more significant in middle and lower parts of lungs, but mostly near the mean wall; with silicosis - it is in lateral and mean ones. Lung roots are not clear and non-differentiated during the first week; then their change depends on the degree of affection of bronchial glands.

Thus, the total clinical pattern of acute miliary tuberculosis differs from the development of silicosis, and radiological changes are characterized by more common rush, and the speed of development; when treating, positive results can be observed, what is not characteristic for silicosis. But there is not such density, expansion or cut-ends as with silicosis. More often it is necessary to differentiate silicosis with chronic tuberculosis. In these cases, the start of this specific process can look like flu.

When making a diagnosis, disease anamnesis is very significant. It is necessary to clarify, in how many years of working with dust, the first indication of the disease appeared? what were the first work conditions? During the period of tuberculosis getting acute, there are symptoms of intoxication, temperature and changes in the clinical analysis of blood, which are very important for differential diagnostics, and the tuberculosis treatment if started then, can solve it completely.

It is also necessary to know well about the compound of this pathology -silicotuberculosis. Most often, tuberculosis is the secondary disease with silicosis. It is also necessary to take into account that the more marked silicosis is, the more often it is accompanied by tuberculosis. In this case, the progress of tuberculosis is characterized by a number of peculiarities. Very often, extrapulmonary tuberculosis develops, and in the mucus, microbacteria of tuberculosis can be often found. TB diagnostics is not very important here, as with silicosis, there is often increased sensitivity to tuberculine. Conducting differential diagnostics between silicosis and silicoturbeculosis, it is necessary to take clinical and radiological patterns into consideration. On the background of marked silicosis, we have to orient more at the clinical manifestation of tuberculosis: frequent catarrhal diseases in the anamnesis with the increase of temperature, symptoms of intoxication, coughing and dyspnea. Objectively, these are local bubbling rales, which appear or increase, when coughing, in particular, in upper parts of lungs, what can prove TB diagnosis. Noise of pleaura rubbing and hemoptysis take place more often than with distinct silicosis.

Changes in blood sampling assist in diagnosis making: increase of ESR, change of leukocyte formula to the left, lymphopenia and sometimes, monocytosis. There also can be anemia, changes in proteinogram towards increasing of coarse-dispersed fractions, mostly y-globulines, and positive reaction onto the C-reactive protein. Radiologically, tuberculosis can be founddue to asymmetrical shadow clusters, infiltrates, and small or big nodular shadows, as a rule in upper areas of lungs, which can be distinguished from silicotic nodules by their size.

Much importance to diagnose tuberculosis is given to positive reaction to antibacterial therapy. However, it is necessary to remember, that with silicotuberculosis, this reaction comes slower, than with pure tuberculosis, that is why it is necessary to be taken into account when making differential diagnosis.

Cases of sarcoidosis can be observed much more often lately. The etiology of this disease has not been found yet. The similarity of radiological changes and sometimes lack of clinical pattern of the pulmonary sarcoidosis reminds silicosis much. Both pathologies can be found by chance at radiological examination.

As with silicosis, the process of sarcoidosis development includes three stages. Stage I is characterized by clear increase of internal pectoral lymphatic glands, vascular - bronchial pattern at this time is not much changed, whereas at Stage I of silicosis there is already an interstitial fibrosis; and the pattern is intensified and deformed, also some individual shadows can be found.

Lung roots are particularly distinguished, they have polycyclic look at stage I of sarcoidosis, and with solicosis they look cut.

Patients with the disease of Stage II, changes in vascular -bronchial pattern remind interstitial form of silicosis, however, they appear mostly in the area near roots, in contrast to silicosis, for which affection of cortical portions of lungs is characteristic.

Stage III of the disease is particularly severe to be diagnosed, as polymorphic fibriosis can take place both with silicosis, and sarcoidosis, and conglomerate shadows, which are created at silicosis, are moved to roots of lungs, what is more characteristic for sarcoidosis.

The main and most reliable indication of silicosis is certificate in lung roots, and particularly the symptom of "egg shell". The latter is never observed with sarcoidosis.

It is important for diagnostics of sarcoidosis of extrapulmonary indications of the disease among patients: skin affection, affection of lymphatic nodules, and affection of locomotorium.

Treatment with corticosteroids gives positive results among patients with sarcoidosis in contrast to the patients with silicosis.

Silicosis should be also distinguished from the syndrome of Haman -Rich. This disease is also called fibrosing alveolitis, or progressing diffusive fibrosis.

The start of the disease can remind pneumonia, however, antibiotics do not usually help as a rule, but on the contrary, they make the state of the patient even graver. The temperature can be febrile, subfebrile

or normal. ESR either grows or stays normal. Coughing is of unstable character, and can forego dyspnea. In lungs, sound bubbling rales can be heard. On the radiological photograph, interstitial small microcellular fibrosis can be seen. This disease can be differentiated only with chronic development. In contrast to the dust pathology, it is characterized by deterioration of the patient. Dynamic observation of the clinical pattern and radiological changes can show "disproportion": with rapid grow of pulmonary insufficiency, small dynamics is observed, i. e. stability in radiological pattern is observed.

At any disseminated process in lungs, moreover among those occupied with "dusty" professions, it is necessary to remember about possible cancer as well as the possibility of development of lung cancer (carcinomatosis of lungs).

Among complications of silicosis, there are pulmonary tuberculosis (silicotuberculosis - STB), pneumonia, bronchiatic disease, bronchial asthma, atrophic arthritis, spontaneous pneumothorax, and coniotic cavity.

Tuberculosis of respiratory organs can be linked with dust diseases of lungs- pneumoconiosis, and especially with the most spread one - silicosis.

Tuberculosis can be met particularly often with small and big nodular forms of silicosis, as well as among patients with grave development of the process.

Silicosis with gradual combination with tuberculosis, tuberculosis with further combination with silicosis as well as silicotuberculosis, where the character of the preliminary affection is impossible.

As a rule, tuberculosis with developed silicosis is secondary. The source of tuberculosis process includes old centers, located in upper and cortical portions of lungs. Spreading of the process goes through lymphogenous, bronchigenous and, sometimes, hematogenous ways. It is considered that the peculiarity of tuberculosis spreading process with silicosis is in selective affection of lymphatic system.

Due to significant compensatory possibilities of the organism, silicotuberculosis is not obvious clinically for a long time. After some time, temperature of the body increases, a patient starts coughing, and weight losses can be observed. Along progressing of the disease, symptoms become more distinguished. General condition worsens, intoxication increases, functions of breathing and blood circulation worsen as well. However with silicotuberculosis, intoxication is less clear, than with similar forms of pulmonary tuberculosis, not connected with silicosis, and the discharge of microbacteria does not take place with marked tuberculosis either.

There is no adopted unified classification of silicotuberculosis. In practice, classification of silicosis and tuberculosis (given in Table 3) is usually used.

More often with silicotuberculosis, nuclear form can be met, affection is rarely double-sided with polymorphic nuclears - 1. 5 cm in diameter, which are located, as a rule, in under collarbone areas, and in upper portions of lungs. Diagnosis of nuclear tuberculosis on the background of marked silicosis is difficult to be made, because tuberculosis nucleons are difficult to be distinguished from merged silicotic nodules.

With all forms of silicotuberculosis, changes of lung roots occur, conditioned by the increase or, sometimes, calcinosis of lymphatic nodules, fibrosis of lungs and hypertension in small circle of blood circulation. Classification of tuberculosis is incapable to unite all forms of tuberculosis development at silicosis. Thus, *the clinical and radiological classification* was suggested, where four main groups of atypical forms of silicotuberculosis are given:

1) Silicotuberculous bronchadenitis with major localization of tuberculosis process in innerpectoral lymphatic glands;

2) small nodular form of silicotuberculosis with appearing of individual shadows, up to 3 cm in diameter;

3) big nodular silicotuberculosis with single or many shadows, with the diameter from 3 to 8 cm, rounded forms - silicotuberculoma.

4) massive silicotuberculosis, where the clinical form of tuberculous form is impossible to state.

Among complications of silicosis, it is necessary to mention spontaneous pneumothorax, which is mostly limited, and thus it develops as non-malignant; however in some cases, it can develop into a total or even double-sidedpneuomothorax with air spreading to the mean wall portion. Valve pneumothorax can be observed as well.

Interstitial pneumonia, as a silicosis complication, changes the degree of compensation of the organism. Bronchoectatic disease with silicosis is a rather rare complication. Mostly, combination of silicosis with bronchial asthma can be observed. Cancer with silicosis is observed as often as with pneumosclerosis of non-occupational etiology. Exceptions are the cases when patients are within the work zone, where ores contain radioactive elements. Then the development of silicosis is more often combined with new formations in lungs.

Cases of combination of silicosis with diseases of conjunctive tissues can be observed more seldom, like atrophic arthritis, sclerodermatitis, and system lupus erythematosus. Combination of silicosis with affection of joints like atrophic arthritis is known in the literature as the Syndrome of Coline - Caplan. Whereas, X-ray can show the presence of rounded shadows, located mostly along the periphery of both lungs and consisting of atrophic granulomas and silicotic nodules.

Atrophic arthritis on the background of silicosis can develop without particular indications of visceral affections. Sometimes, in the clinical pattern of silicoarthritis, affection of internal organs is the main. Lethal cases of patients with silicoarthritis from uraemia are known, caused by atrophic affection of kidneys.

Combination of silicosis with sclerodermatitis is not a rare case, especially among miners.

Treatment. Main approaches in treatment of patients with silicosis are based on the understanding of the mechanism of the disease development, character of morphological and functional changes, especially of the progress and complications.

First of all, it is necessary to remember that patients with silicosis of Stage I, who do not have external respiratory function disorder, should work rationally. Strengthening of general state of organism and increase of its protective forces, in particular by training and tempering, are very important. To do so patients should be recommended to do hygienic physical exercises and walking in fresh air. Significant role is played by rational meals (special diet, enriched with protein - 100 - 150 g of cheese, with adding pancreatin or methionine in the dosage of 0. 5 to 1 g a day).

Among means of impact onto the pneumoconiotic process, it is very important to withdraw dust from lungs by inhalation of mineral waters of various composition. It has positive impact onto the mucous tunic of trachea and big bronchi.

Such gradual development of fibrosis process with silicosis served as a basis for clinical utilization of glucocorticoids. But then it was found out that patients with non-complicated silicosis should not take in glucocorticoids. They can be prescribed only to patients with combination of marked stages of silicosis and atrophic arthritis, as well as in case of fast progressing silicosis. The following treatment regimen is suggested: prednisolone in the dosage of 20 to 25 mg a day. Maximum amount is prescribed for 10 to 12 days, then it should be gradually decreased by 2. 5 mg every 5 to 10 days.

Treatment with corticosteroids should be conducted only under protection of anti-tuberculous drugs, among which phthivazide and para-aminosalicylic acid, PAS(A) are the best, whereas these drugs should be prescribed for 1 to 2 months after hormones are cancelled.

Among drugs, which have antifibrous action, it is possible to use 2 % solution of poly-2-vinylpyridine-N-oxide, which is introduced intravenously together with the isotonic solution of sodium chloride (150 - 200 ml) in the drop form every other day. One-time dose of 0. 1 - 1 g, and the course is 15 to 20 infusion.

However until now, there is no unified thought as to the efficiency of this drug; there is data on side effects of poly-2- vinylpyridine-N-oxide (gonadotropic and cancerogenic). Besides, some clinical testing

has not established treatment effect of the drug. Thus, search for efficient polymers, capable to restrain the development of fibrous process in lungs, continues.

There is positive data regarding utilization of glutamic in the dosage of 0. 25 - 0. 5 g three times a day for patients with silicosis. It restrains the development of silicosis and asbestosis in the experiment, decreases the level of progressing and causes reverse development of dust fibrosis in lungs.

The main pathophysicological disorder, which appears in the very first indications of silicosis, is oxygen insufficiency. Thus, in the therapy of patients with silicosis, it is necessary to use oxygen therapy, drugs, which stimulate activity of the respiratory center (Cordiamin - 25 to 30 drops or 1 - 2 ml subcutaneously). Besides pathogenetic methods of treatment, significant place in therapy of patients with silicosis is taken by symptomatic therapy.

If patients cough, they are recommended to take in expectoration drugs: 3 % of potassium iodide solution or 0,5 % of the tincture of termopsis herb. For bigger effectiveness, this drugs should be taken in with much water.

Among physiotherapeutic methods of treatment, the following have been proved to be good: ultrasound, particularly among patients with uncomplicated silicosis of Stage I with the presence of pain syndrome, coughing, disorder of drainage function of bronchi, bronchial permeability, as well as electrophoresis with various medicinal drugs, depending on one or another clinical syndrome.

Particular place in treatment of patients with silicosis is taken by drugs, which increase total reaction of the organism. They include alcohol extract of eleuteroke, which is taken in by 30 to 40 drops 30 min before meal each day for 30 days.

Among medicinal drugs, which create non-specifical stimulation of the patient's organism, it is possible to name prodigiosan, introducedintramuscularly in the dose of 25 to 50 mg of 0. 005 % solution once a day every 4 to 5 days, the course is from 3 to 4 injections.

Treatment measures should be directed at the fight against complications as well (development of cor pulmonale, cardio-vascular insufficiency, or pneumonia). To do so, cardiac glycosides are taken (strophanthin 0. 5 ml of 0. 05 % solution), aminophylline (5 - 7 ml of 2. 4 % solution), diuretic (furosemide and hydrochlorothiazide). Treatment of pneumonia should be purposeful, with the consideration of the character of microflora (ampicillin, 0. 5 g, 4 times a day; sulphalen - the first day - 1 g and then 0. 2 g a day). Protein synthesis in the organism can be stimulated by anabolic steroids (metanedrostenolon - 0. 005 g 1 to 2 times a day before meals). Treatment course should be from 3 to 4 weeks, retabolil - 1 ml of 5 % oil solution intramuscularly, total 8 - 10 injections.

Main method of treatment of silicotuberculosis is chemotherapy with modern anti-tuberculous drugs: isoniazid (dosage - 0. 6 - 0. 9 ga day), rifampicin (average dose - 0. 45 g a day); ethambutol (15 - 25 mg per 1 kg of body mass a day); etionamid (inside, 20 min after meals, once a day 0. 5-0. 75 g); streptomycin (intramuscularly, in the dosage of 0. 5 - 1 g a day).

Total duration of the treatment of active forms of silicotuberculosis - not less than 1 - 1. 5 years. Besides, the whole arsenal of medicinal drugs against silicosis can be used (generally sanative, physiotherapeutical therapy) with the consideration of the development and expression of coniotic process.

Verification of work ability: Silicosis of stage I of the stage is a contraindication of industrial dust impact. Patients should be transferred to another job beyond the conditions with dust, irritating gases and unfavorable metrological factors. Hard physical work is contradicted also. If rational job is connected with the decrease of qualification, the patient should be assigned to attend doctor - labor commission to receive Disablement Group III on occupational disease.

At stage II, the patients always have the right to receive Invalidism Group (mostly Group III, and in case of its combination with tuberculosis, and respiratory insufficiency - Group II).

At stage III of the disease, there is marked respiratory and cardio-vascular insufficiency, and sometimes the need of assistance from others (occupational disablement of group II or I).

Preventive measures of silicosis envisage the conduct of the following:

• complex mechanization of production processes;

• sealing-in of the machines;

• organization of efficient production ventilation;

• hygienic norming of professional hazards;

• record-keeping and research of specific cases of occupational diseases;

a) biological methods of prevention methods:general sanative ones (rational organization of the work and leisure, rational meals, and physical exercises);

b) special (respiratory exercises, inhalation of aerosols, and rational meals with vitamins);

• preliminary and periodical medicinal examination of people, who work under conditions of professional hazards;

• utilization of individual means of protection.

Silicatosis

Silicatosis are pneumoconiosis, which develop in the result of inhaling of silicate dust.

Besides, free silicon dioxide, in the nature there is a number of complex mineral compounds, which include silicon dioxide, which is not in a free mode, but in connection with other elements (silicates). Silicate ores can be met in mountain mines: in asbestos - talc mining industry. Silicates are used in fire clay and dinas production, when manufacturing rubber products, perfume and many other industries.

Work processes, connected with extraction and utilization of silicates, are often accompanied by inhaling silicate dust. Long inhaling of this dust can cause the development of pneumoconiosis - silicatosis. The type of dust, which caused it, determines type of silicatosis.

Asbestosis is silicatosis, which is caused by inhaling asbestos dust.

Asbestos is a mineral with characteristic fibrous structure, which is widely used to produce thermal isolation materials. Creation of asbestos dust takes place in mining asbestos, as well as during its sorting and processing. The dust degree of the environment directly depends on the character of production process and is very high when crushing asbestos. Boundary permitted concentration in the work zone for aerosols of natural asbestos, as well as mixed asbestos-natural dust with the concentration of asbestos in them over 10 % is 2 mg/m3; for asbestos bakelite - 8 mg/m3 and for asbestos - cement - 6 mg/m3.

Clinically, asbestos is apparent in a number of complexes of chronic bronchitis symptoms, lung emphysema and pneumosclerosis, which are accompanied by dyspnea and coughing.

Dyspnea is one of the first symptoms of the disease. At first, it appears at physical activity, and in more serious conditions, it can be observed in rest as well. Together with dyspnea, dry rales can be observed, which later develop into viscous expectorating mucus; sometimes, coughing is in the form of fits. Rarely, astmatic phenomena can take place as well.

Characteristic complaints onto dyspnea and coughing are often accompanied by pain in the chest, and in particular when making deep inhales. Patients with marked asbestosis have disorders of the general conditions: headaches, general weakness, and undue fatiguability. Sometimes, dyspeptic phenomena take pace as well. Appearance of patients can have peculiar grayashy color with light cyanosis of lip mucous tunic. Patients can start losing weight.

The chest often has a usual form. When examining, emphysema can be found in upper portions. Breathing is rough with prolonged exhaling, in the lower portions - weakening is observed, often with dry disseminated crepitations; in lower parts, fine and medium moist rales can be observed. Cor pulmonale develops late. At first, there is accent of tone II on the lung artery. Labial pulse develops with time, as well as tachycardia and phenomena of decompensation on the bigger circle with characteristics changes for that time on the electric cardiogram (tall waves P2 and P3, reduction of interval S - T in II and III sectors, dextogram).

Sometimes in the mucus, asbestos corpuscles can be found, which are light yellow lumps of prolonged form with clavate or circular endings.

Asbestosis appears among those who working contact with asbestos dust for about 5 years or so, whereas in contrast to silicosis, clinical indications are usually ahead of radiological.

In compliance with the clinical and radiological indications, usually there are three stages of asbestosis: I, II and III; stage III can be observed in single cases - in particularly unfavorable work conditions or with complication of chronic pneumonia and bronchoectatic disease.

Patients with the disease of stage I have marked diffusive emphysema of lungs, diffusive intensification of vascular- bronchial pattern, more intensified in lower portions of lungs (bronchitis and bronchiectasis) and fine cellular pattern in mean portions, found after radiological examination.

Lung roots are slightly widened, shadows are dense, and their structure is rough.

Patients with asbestosis of stage II have the same indications, only in more marked form based on radiological examination. Vascular - bronchial pattern has more coarse cellular structure. Sometimes, there are numerous punctuate shadows of nodular character. Lung roots are denser and widened. There can be indications of the beginning of cor pulmonale.

With the disease of stage III, there are marked phenomena of pneumosclerosis and emphysema. Often there are significant changes of pleura and characteristic indications of cor pulmonale. In contrast to silicosis of stage III, with asbestosis of stage III, there is not many pneumosclerotic fields.

As it is, pneumosclerotic process from asbestos dust action does not have a tendency of fast progressing. The severity of the condition of patients with asbestosis depends on the degree of the expression of bronchitis, emphysema of lungs, development of bronchoectasis, and infection joining. The given complications are the main reason of significant disorders of respiratory function and disorders of hemodynamics of small blood circulation circle.

Talcosis is silicatosis, which appears due to the talc dust action. Talc is magnesia silicate, which does not dissolve in water, and slightly dissolve in acids and alkali. In the production, talc is used in rubber, textile, and paper industries. Highest sorts of crushed talk are used in perfume industry.

Pneumoconiosis caused by pure talc, when it is mined and used, develops usually not earlier than after 10 years of work. The process of the disease is moderate. Complaints: dyspnea at physical activity, pain with unstable character in the chest, coughing, mostly dry with some mucus. Weight losses are observed also. With percussion, box sound is heard in lower lateral portions of the chest. With auscultation, coarse breathing can be heard.

Radiologically, fine cellular pattern of lungs and single shadow spots are observed, which cover the lung area levelly. Lung roots are somewhat widened.

However, the clinical and radiological pattern of the talc pneumoconiosis can be more marked as well. Workers, engaged in talk mining and primary processing of ores, can have diffusive fibrous changes in lungs, which on the x-ray remind a pattern of silicosis of groups I and II. It is necessary to consider that the bigger degree of affection in this case can be explained by the additive of talc to free silicon dioxide, i. e. silicotalcosis takes place here.

There are *three stages of talcosis*:

Stage I. Patients complain to have dyspnea when having physical activity, unstable coughing, and pain in chest. With percussion, it is possible to determine indications of basal emphysema, and with auscultation - coarse, and in lower lateral portions - slightly weakened respiration, and dry tales. After radiological examination, it is possible to observe the increase and deformation of vascular pattern due to the development of fibrous process, as well as deformation of lung roots.

Stage II is characterized by the increase of dyspnea, coughing and mucoid sputum. Objectively, complex of symptoms of bronchitis with emphysema indications. Radiological examination shows a marked intensification of vascular - bronchial pattern and emphysema. Lung roots are widened and deformed.

Stage III. Dyspnea in rest, coughing and intensive pain in chest, as well as presence of cyanosis. After percussion of lungs, box sound is observed, quick restriction of movements of the lower portion of lungs. Dry and moist rales can be heard. As to the cardiac-vascular system: widening of the right border of heart, cardiac tones are muffled, accent of tone II is over the pulmonary trunk. Radiological examination shows pneumosclerosis. Lung roots are widened.

Cement pneumoconiosis is silicatosis, caused by the action of cement. Cement is silicatosis, but it includes free silicon dioxide as well. In the production dust of Portland cement, there are from 3 to 7 % of free silicon dioxide, and in the production dust of acid-proof cement - up to 67 %. The permitted concentration for cement dust is 6 mg/m3.

Cement pneumoconiosis is characterized by complaints on coughing, mild pain in chest and dyspnea at physical activity. The progress of pneumoconiosis depends on the type of cement, which cased the disease. Thus, with prolongedinhaling of Portland cement dust, interstitial dust fibrosis gradually develops among workers. Workers, producing acid-proof cement, can have the disease with comparatively low term of work (from 7 to 9 years). During radiological examination in this case, besides interstitial fibrosis, there can be fine-nuclear formations in mean portions on both sides, which remind silicosis. Complications with tuberculosis are rare. Besides, pneumoconiosis, there is development of bronchial asthma, skin irritation, and "cement rash" and conjunctivitis.

Treatment. To treat silicatosis, means are utilized which stimulate protection forces of the body (solux, ultra-violet radiation, oxygen therapy, and respiratory exercises). Bronchological, antihistamine and inflammatory drugs, as well as vitamins (P, ascorbic acid and nicotinic acid)In case of complications of silicatosis, the following can be prescribed: antibiotics and sulfanamides (pneumonia), cardiac glucosides - strophanthin, corglucon, and diuretics - lasix, and hydrochlorothiazide (cor pulmonale).

For further rehabilitation, it is advised to take resort treatment (Crimea) under conditions of marked cardio-vascular insufficiency and exacerbation of the inflammatory process.

Verification on work ability. Patients with silicatosis of stage I are subjects to rational work, in particular if they have bronchitis, pneumonia or signs of further development of fibrous process (asbestosis, rarely talcosis, or olivinosis).

If the stage of silicatosis is II or III, patients should get disablement group II or I of occupational character (first of all, if there is chronic obstructive bronchitis, cor pulmonlae, marked decrease of external breathing function).

Preventive measures. To prevent appearing of silicatosis, it is important to take sanitary - technical measures (sealing in and mechanization of production processes, ventilation, utilization of individual respiratory organ protection means) as well as conduct of preliminary and periodical medical examinations.

Carboconiosis

Carboconiosis is pneumoconiosis, caused by the action of dust, which contains carbon (coal, graphite, or coke).

With carboconiosis, moderately marked mostly fine nuclear and interstitial fibrosis is observed. Anthracosis is one of the most spread and practically the most important disease in this subgroup of pneuconioses, which usually develops among miners, engaged in mining coal, as well as workers of ore-dressing plants and some other manufactories.

Among workers of coalmines, depending on labor conditions and domination of some type of dust, there are three types of pneumoconiosis: anthracosis, silicosis and anthrasilicosis. Anthracosis. It appears and develops in case of long work period in mines (from 15 to 20 years and inhaling the air with high concentration of coal dust. The permitted concentration of dust of coal, which contains less than 2 % of silicon dioxide, is 10 mg/m3.

Pathological and anatomical pattern. Coal dust depositing in lungs is primarily characteristic for anthracosis. Lungs become of gray - black color. In marked stages of anthracosis, conglomerations of coal dust pigment is levelly spread along the total area of lungs.

In alveolar septum, around vessels and bronchi there is union of conjunctive tissue, in some places clusters of cells with particles of coal dust are observed which got their name from anthracotic nucleuses. In contrast to silicotic nodules, they do not have concentrically placed beams of conjunctive tissue. Pneumosclerotic changes are joined with emphysema of lungs and chronic bronchitis.

With anthracosis, silicotic nodules can be observed in lungs.

Clinics. For coal pneumosclerosis, more marked bronchitis and lung emphysema are characteristic, what distinguishes it from silicosis. More marked and functional disorders of the external respiratory apparatus can be observed. Radiological changes contain diffusive interstitial fibrosis and numerous small nuclear formations on the background of emphysema of lungs. There are *three stages of the disease*.

Stage I. Patients complain on fast fatiguability, dyspnea at physical activity, coughing and pain in the chest. Sometimes, complaints are absent, though radiological photograph register clear changes. Radiological pattern is characterized by the appearance of small nuclear shadows on the background of cellular deformed lung pattern in mean portions (mostly to the right) with the presence of bigger shadows of lung roots. The diameter of clusters is between 1 to 3 mm, and sometimes 1 to 5 mm.

Stage II. Complaints on dyspnea, and sometimes in rest. fast fatiguability, and pain in the chest. Objectively, emphysema and bronchitis are observed. Radiological pattern is marked with the increase of the number and sizes of fine nuclear shadows, located not only in the mean, but also in subcollar bone portions. Shadows of lung roots are widened; their intensity is increased. Pleural changes can be changed often in the upper portion and emphysema is marked.

Stage III (is rare in the conditions of coal dust only). Complaints on general weakness, dyspnea even in rest and at light physical activity, coughing, often with mucus, and pain in the chest. During radiological examination, massive homogenous shadows of irregular form with clear outlines can be observed, which are located symmetrically or on different heights in both or one lung among fine nuclear and porous formations.

As to clinical and radiological development, anthracosis is comparatively a non-malignant, moderately progressing chronic disease. Combination withtuberculosis worsens the forecast much. However with anthracosis, the danger of complication with tuberculosis is much less than with silicosis.

Coal miners can have both anthracosis and silicosis. Anthracosis is usually observed among miners, who work full-time on coal extracting and face lavas. Tunnellers, who conduct preparatory works and inhale dust with silicon dioxide, have anthracosis or silicosis developing. That is why to solve the problem as to the character of pneumoconiotic process, it is necessary to study occupational anamnesis and work conditions well.

Graphite pneumoconiosis. It is carboconiosis, caused by graphite dust. Graphite is a dark gray substance with fair chemical activity, one of the variations of coal. It is used to produce cast iron, stainless steel, electrodes, in electrical devices, as well as to make pencils and paints.

Pneumoconiosis caused by graphite dust, develops slowly, after the work period of over 10 years, and has non-malignant character. Patients with graphite pneumoconiosis complain to have pain in the chest, dyspnea and fast fatiguability. After objective examination, chronic bronchitis and lung emphysema are observed. Harmful impact of graphite dust onto the ENT-organs is observed (atrophic pharyngitis and rhinitis).

Radiological examination shows cellular fibrous process, which correspond to stages I or II of the disease. However, in rare cases with graphitosis, graver changes in lungs can be observed, i. e. large fibrous fields with portions of necrosis, which can be explained by particularly unfavorable conditions.

Verification of work ability. In case of appearing of pneumoconiosis of Stage I, without complications of disorders of external respiration, a patient can work without preliminary attending the Expert Commission. The obligatory condition is dynamic control of the health state by doctors and work conditions normalization. If pneumoconiosis of stage I is combined with bronchitis, and moreover in case of appearing of pneumoconiosis of stage II or III, further work in dust conditions is contra-indicated.

Preventive measures: prevention of dust creation (wet drilling and mechanization of work processes, as well as medical examination of workers.

Metal-coniosis

Metal-coniosis is characterized by depositing of radiological contrasting dust in lungs with moderate fibrosis reaction (siderosis, baritosis, etc). These pneumocioses are distinguished by non-malignant development. According to the current classification, berylliosis, aluminosis, pneumoconiosis caused by cobalt dust and toxic air with repeated reaction of pulmonary tissues are included to the group of metal-coniosis.

Aluminosis is metal-coniosis, caused by aluminum dust action.

Aluminum is white silvery light metal. Metal aluminum and its compounds are widely used in the industry. Contact of workers with aluminumdust or vapors takes place when producing metal dyes, artificial abrasive elements, pyrotechnic aluminum powder, etc. The permitted concentration for the aluminum and its compounds is 2 mg/m3.

Aluminum gets into the organism when inhaling its vapor and dust. The term of development of aluminosis varies from 6 months to many years.

With such concentration of aluminum dust, changes in lungs can take place within the first year of work in the result of probably allergic reaction of the organism.

Mechanism of the action of aluminum dust onto the pulmonary tissue has not been completely understood yet. Obviously, under the impact of the tissue fluids, aluminum ions, starting reaction with proteins in the organism, create strong complex compounds, which violate normal life activity of cells. With time, interstitial regenerative union of collagen- hyaline tissue takes place. Around of some particles of aluminum, non-dyed membranes are observed, which are called "aluminum corpuscles".

Clinics. Patients often complain to often have catarrhal diseases, fits of coughing with mucus or without it, tension in the chest, flabbiness, dyspnea, absence of appetite and pain in stomach. Then dyspnea at physical activity is observed, in some grave cases - clear dyspnea and strong suffocating coughing even with insignificant physical activity.

Objectively, clinical data is not very characteristic in the beginning. Breathing is hard, dry rales can be heard. With the development of the process, cyanosis, degrease of vital capacity of lungs, and sometimes up to 1000 ml or less are observed.

In spite of significant disorder of external breathing, radiologically it is impossible to find any changes at this stage. On the radiological photograph, first changes appear in the form of intensification of vascular pattern of lungs, especially in mean portions. Sometimes, there can be single, fine and average sized mottled formations. These changes are located symmetrically. Lung tops in the majority of cases are free. In the future, merging of spotted shadows takes place with the creation of homogeneous carnifications. In severe cases, lung roots are widened and carnified. In contrast to silicosis, there are no nodular formations and tumor-like carnifications at aluminosis.

As a rule, even after the termination of the contact with aluminum dust the dust, accumulated during the production period in lungs, continue acting, and this process progresses inevitably. In blood, limphocytosis and eosinophilia are observed.

Siderosis is metaloconiosis caused by inhaling metallic ferruginous dust. Mostly it is observed among workers of blast-furnaces and agglomeration factories.

Pathologic and anatomic pattern. Volume of lungs is increased. On their sections, nodules are observed, the diameter of which is up to 6 mm. Lung coloration is black (if impacted by ferrous oxide) or yellowish (if impacted bythe dust with ferrioxide). Lymphatic glands are increased, and are or red color in the section.

At histological examination, excrescence of conjunctive tissue and dust deposits with iron are observed in alveolar septum, as well as around bronchi and vessels. Also, fine nodules (dust pictures, pieces of conjunctive tissue, histiocytes and lymphoid tissues are observed.

Clinics. Clinical pattern of siderosis is very poor. Patients have no complaints during long times, which would prove the affection of lungs. Function of breathing stays unchangeable. Only on the radiological photograph of lungs, lightly marked interstitial fibrosis and disseminated fine nodular shadows with clear outlines, where dust accumulated, are observed. Thus, the diagnosis of siderois is sometimes determined only based on results of radiological examination with the consideration of dust composition, present at the production where the patient works.

Practically, there are no complications with siderosis. Patients with uncomplicated forms of siderosis do not require treatment in the majority of cases. They are completely capable to work.

Berylliosis is metaloconiosis, caused by inhaling beryllium. Beryllium is silvery-gray light metal. In production, beryllium compounds are used (beryllium oxide, beryllium sulfate, beryllium chloride, and beryllium fluoride) to produce X-ray tubes, luminescent lamps and to receive atomic energy. Beryllium metal is used to receive alloys of strong and sparkle tools, beryllium steel, as well as in ceramic production. The boundary permitted concentration of beryllium is 0. 001 mg/m3.

Beryllium gets to the body through lungs in the form of smoke and vapors. It is mostly deposited in bones, liver and kidneys. It permeates through placenta, and can be found in urine many years after termination of contact with it.

Both beryllium and its alloys are toxic (the most toxic are compounds, especially beryllium oxyfluoride). Beryllium and its compounds have local (onto respiratory tracts and skin) and resorptive (onto the central nervous system and parenchymatous organs) impact and is a carcinogenic matter.

There are two forms of berylliosis: acute and chronic. Dissolved beryllium compounds mostly cause acute intoxications, and non-dissolved compounds -chronic ones.

In pathogenesis of berylliosis, an important role is played by autoimmune processes, conditioned by sensitization the proteins of the body itself, what is significantly changed under the influence of beryllium.

According to contemporary understanding, berylliosis is close to colagenoses. Beryllium interrupts the activity of a number of ferments (alkali phosphotasis, and magnesium interaction).

Pathologic-anatomic pattern. With acute poisoning with beryllium, quick plethora and swelling of mucous tunic of trachea and bronchi areobserved. In gaps between bronchi and around vessels there are some erythrocytes and lymphoid cells. Interalveolar septums are thickened due to their infiltration with lymphocytes. In alveolar cavities, there are accumulations of exudation with a big amount of fibrins, separate gigantic cells, Langhasen' s type, as well as peeling of epithelium. Blood vessels are expanded; in the parenthem there are separate hemorrhage focuses. In later stages, industrial alveolitis rakes place in the form of carnificating pneumonia.

At chronic berylliosis, lungs are much enlarged on the macroscopical stage; they are dense of gray red color. The surface of lungs is small-grained. There are numerous fine nodules, which are dense when touched. Morphological pattern with chronic berylliosis consist of epithelial cells, and a small number of lymphoid, plasmatic and multinuclear gigantic patterns of Langhans, located mostly along the periphery. The evolution of granulomatosic process with berylliosis is characterized by formation of fine sclerotic nodules, which form large granulomatosic nodules after merging.

Clinics. Acute form of the disease develops in the following forms:

• in the form of acute affection of conjunctiva and upper respiratory tracts; transition into a sever form with lung affection is possible (disease duration: several days or weeks);

• in the form of "beryllium fever";

• the most sever case - acute brochobronchiolitis or the so-called pneumonitis, the progress of which can develop in two phases: in these cases, the disease starts with symptoms of metal fever, after which a non-symptom stage starts (4 - 6 days), and after that, bronchobronchiolitis takes place.

Patients often complain to have pain in the chest as well as very strong coughing (dry or with heavy expectoration of mucus, often with a mix of blood, asphyxia, committing and clear cyanosis. Tachycardia and hypotensia are well observed as well. Lungs are emphyzematosic; dry and sometimes bubbling rales can be heard. Liver is often bigger in size, and painful. The temperature is increased up to 38 to 39 0C. In the blood there are neutrophilic leukocytosis with stab changes, ESR is increased. In urine, beryllium is found. The progress is wavelike with periods of worsening, long - up to 2 - 3 months. There are possible relapses in case of returning back to work, as well as beyond contact with beryllium under the impact of intercurrent diseases or without any obvious reason. Radiologically, it is possible to find indications of confluent pneumonia large in size, which covers one or portions of lung at a time and develops with acute reaction of lung roots. With the second version of this affection, together with diffusive decrease of transparency of lung, on the bigger or smaller portion of lungs there are small nuclear shadows from 1 to 2 mm in diameter. Lung pattern is changed on a large scale or diffusive, unclear and of small porous character. Roots are widened and non-differentiated. The described pattern stays in place for 2 - 6 - 8 weeks. Scars can stay forever, Chronic berylliosis. It develops gradually. At first there are asthenic complaints: weakness, increased fatiguability, then breath catching during walks and later in rest as well, fit-like coughing, pain in chest. Some patients lose weight fast: within a short period of time patients lose from 10 to 20 kilos. At fast progressing transition, the body temperature can increase up to 39 - 40 0C with chill, grave general feeling and explicit asphyxia. Cyanosis becomes much clearer now, which gets diffusive character with time. Lymphadenopatia is observed.

During pulmonary percussion, box sound can be heard, which proves the presence of emphysema, restriction in the movements of lung end though union process in pleural cavity. In lower parts of lungs, there are small bubbling rales, rarely, they are dry and scattered; pleura friction noise is observed also.

During the research of functions of the external breathing, hypoxemia can be heard, the degree of which grows in parallel tot the severity of the case. The most characteristic is the change of diffusion ability of lungs, connected with clinical infiltration of interalveolar septums and development of alverolar and cailar block. Deficit of saturation of the arterial blood with oxygen, increase of the content of reduced hemoglobine are caused by early development of cyanosis.

In case of further development of the disease, lung hypertensia takes place with further development of cor pulmonale: tachycardia takes place, right sections of hears increase, myocardium tonic reduces. ECG shows tall wave P in II and III standard ducts, relative increase of PV1 wave.

Granulomatosis process can be determined in parenchimatoous organs -liver, spleen, as well as in lymphatic nodules. Often, increase and pain in liver is observed with disorder of its functioning as well as spleen expansion.

In the blood, increase of erythrocytes is observed, as well as increase of the level of total protein of blood serum, mostly due to hypergammaglobulinemai (20 - 30 g/l), as well as increase of IgC, in case of acute disease - IgA.

The listed above clinic indications are characterized by the acute berylliosis, in the period of remission, the disease does not have many syndromes. Depending on the character of radiological changes in lungs, there are two forms: interstitial and granulomatous; depending on the explicitness of the latter - stages are I,II and III.

The interstitial form is characterized by diffusive changes of lung pattern, clinically it s more non-malignant and as a rule it is restricted by stage I. The grave form is granulomatous, which is characterized by the presence of fine or large nuclear shadows (granulomas), widening of roots due to hyperplasia of lymphatic nodules, early development of pulmonary and cardiac insufficiency in the result of alverolar-capilar block, which causes the violation of oxygen diffusion. The disease in Stage I is characterized by relative diffusion increase and deformation of lung pattern. The increase of radiological photograph enables to find spot shadows opf granulomas.

Patients with Stage II have marked deformation of lung pattern, numerous fine spotted shadows of nodules. Changes are located mostly in mean and lower portions.

In Stage III, further increase of the number and sizes of nodules, diffusion fibrosis, as well as emphysematous changes are observed.

Berylliosis often develops after a short period of work in contact with beryllium or after many years of the termination of contact with it.

Berylliosis can be observed among those, who are not in contact with beryllium, but live not far from beryllium production, and sometimes even at a bigger distance from it. Thus, to develop a sever form of the disease; sometimes it is enough to have a short-term contact with beryllium under conditions of its small concentrations.

Skin manifestations appear at direct action of berillium action, its vapors and aerosols. There are dermatitis with such types as contact and allergic; on the place of former microtrauma, there can be ulcers, which gradually heal. In case of permeation of undissolved compounds of beryllium into the skin, underskin granulomas appear, also fistulas are possible; sometimes it can develop for a long time (for months). Bone affection with thickening of periostis of ribs and long cortical bones

Diagnostics of berylliosis is based on the contact with beryllium, as well as characteristic clinic and radiological pattern, disproteinemia; beryllium can be found in bioenvironmental (urine); magnesium content in blood plasma is reduced, and its increased excretion with urine.

Significant diagnostic criterium, especially with granulomatous form, is positive allergic skin sampling with beryllium. Compress of 0. 25 - 0. 5 % water solution of $BeCl_2$ is put onto a healthy patch of shoulder skin. At positive result, scattered follicular papules appear in 8-12-20-24 hours, sometimes there are erythema or swelling (they stay from 5 to 12 days and then pigmentation is left).

Differential diagnostics of berylliosis (chronic form) is a very serious problem, as its clinic - radiological form has many similar moments. First of all, it concerns sarcoidosis, miliary tuberculosis, syndrome of Haman - Rich, silicosis and other pneumoconiosis.

In spire the fact that berylliosis is referred to metalconiosis, in the action of beryllium there are moments, which are not chatracteristics to the impact of other types of dust. It proves to be more like poison with allergic action mostly affection respiratory organs. It is considered that silicosis is a chronic disease, referred to real occupational nosologies, then the development of berylliosisis possible among people, who do not have direct contact with beryllium, and the severity of intoxication is often unadequate to the amount of poison, which permeated into the organism. Sever cases of diseases (often lethal) can be among people, who lived not far from beryllium production (1 to 2 km away). Some cases have been described when the disease developed after a short-term contact with beryllium(20 min contact). The beginning of chronic berylliosis differs from silicosis. Patients lose weight fast, they become weak, fatigued, often do not stand any medicinal drugs, antibiotics in particular. When conduction pulmonary percussion, box sound can be heard. With auscultative methods, scattered dry and fine moist rale can be heard; physical pattern is more vivid, usually a patient has well marked asphyxia. A patient can also have fever. Good results can be observed after glucocorticosteroid therapy. In contrast to other pneumoconiosis, berylliosis can affect not only respiratory organs with various clinical manifestation, but also skin, and lymphatic apparatus; it can produce marked hepatolienal syndrome, and affect joints, what differ it much from silicosis and brings

closer to sarcoidosis. There is also such a definition that berylliosis is sarcoidosis with known etiology, though its progress is milder.

Indications of berylliosis can appear in about several months, and sometimes in many years (15and more) after the termination of any contact with beryllium. That is why it is always necessary to remember about the possibility of berylliosis development under conditions of unclear diagnosis, as well as well remember about the existence of acute berylliosis, where syndromes of rhinitis, pharyngitis, tracheitis can develop, as well as the development of bronchiolitis, and toxic pneumonia can be observed which develop for a long period of time and in severe forms with its episodes and relapses. Acute form can remind the syndrome of Haman - Rich.

To make a final decision as to the diagnostics of berylliosis can be done with the help of skin sampling of Curtis with the solution of sulfate or beryllium nitrate.

When conducting differential diagnosis of berylliosis with sarcoidosis, it is necessary to remember that for the latter, it is characteristic to have simple progressing, absence of alveolar-capillary block, large polymorphism of clinical manifestations (affection of skin, lymphatic nodules, locomotor apparatus, eyes, nervous system, heart, liver, and kidneys). Positive reaction of Quame is also very important.

As to differentiating berylliosis with the syndrome of Haman-Rich, it is necessary to take into consideration the presence of contact with beryllium, positive skin sampling, as well as results of puncture biopsy of lungs.

Exception of the diagnosis of tuberculosis is based on absence of clear symptoms of tuberculosis intoxication, negative tuberculosis testing (based on the positive Curtis testing) as well as on the results of specific test-therapy.

Numerous research of mucus to have atypical cells, results on broncioscopic research, conduct of transbronchial puncture of lung tissue, definition of the state of function of external breathing permit exclude a possible diagnosis of miliary tuberculosis. Treatment. Treatment measures for patients with berylliosis differ much from the therapy of other types of pneumoconiosis. With acute forms of the disease, it is important to terminate any contacts with beryllium compounds. If upper respiratory tracts are affected, it is necessary to use inhalations: warm alkaline or oil ones with menthol. With acute bronciolitis and pneumonia, treatment should be complex and include drugs, which are directed at the treatment of pulmonary and cardio-vascular insufficiency (oxygen and cardiac glycosides), anti-infection means (antibiotics and sulfonamides), as well as desensitizing drugs (Dimedrol and Suprastin). In grave cases, it is necessary to use dexamethasone and prednisolone.

Autoaggressive character of changes with chronic berylliosis is the basis to use glucocorticoid drugs. When choosing the treatment scheme for patients with berylliosis, it is necessary to consider their age, concomitant diseases, as well as the disease stage. Usually, the treatment is conducted in courses, 30 to 50 days each, with 30 to 40 mg of prednisolone to start with. The dosage reduction should be done gradually. It is necessary to remember about the possibility of complications due to glucocorticoid therapy (metabolism disorder, worsening of diseases of alimentary canal, increase of arterial blood pressure, and reduction of resistance to infections.

Verification of the ability to work. Considering the quickness and fast development of berylliosis already within first days and weeks of work, as well as ability to relapses, severity of development and complications, relapsing character of skin affections and tendency to development of pulmonary pathology among these people, and with chronic or marked acute poisoning, it is necessary to restrict person from working in the conditions with beryllium and its compounds, and to provide full-time rational job.

Contraindications to continue work with beryllium can become positive skin sampling. With full-time work, all patients with marked form of berylliosis are assigned to visit doctor-expert commission (full restriction of work), as well as with mild forms - those who need to be re-qualified. Issues temporary

termination of contact with berylliumcan be raised in case of isolated affection of eyes (conjunctivitis) or light affection of upper respiratory tracts.

Preventive measures. Among the measures to prevent intoxication with beryllium, it is necessary to do the following:

• to utilize protective measures and first of all to use uniform with its further treatment;

• to utilize technical devices and equipment which would reduce the risk of beryllium impact of the worker;

• to utilize effective general ventilation;

• to conduct preliminary and periodical medical examination of workers.

Pneumoconioses Caused by Mixed Dust

Pneumoconioses of this type develop in case of combined action of various types of dust. Clinic and radiological manifestations within this group are various, what depends on the composition and physical and medical properties of the dust, especially from the mixture of free silicon dioxide.

Peumoconiosis, caused by the action of mixed dust, which large amount of free silicon dioxide, are well spread and are close to silicosis on their manifestations. It can be met among workers of coalmines, iron ores, as well as ceramic and china - faience industry. Depending on the character of the mixture, the following forms are distinguished: anthracosilicosis, sidersilicosis, and silicosilicatosis.

Among pneumoconioses from the action of mixed dust with insignificant mixture of silicon dioxide, there are pneumoconiosis of electric welders, gas welders, and grinders, when radiological dust of metal depositing takes place.

Pneumoconiosis of grinders. This pneumoconiosis can be met in 7 - 14 % of cases among workers of metalworking industry among those, who work on grinding - and - polishing operations. It appears in the term from 15 to 30 years from the start of working on the profession, connected with dust impact. The disease usually progress gradually, and very rarely it get to stage II.

Clinics. The clinical pattern is characterized by symptoms of bronchitis and emphysema of lungs. Patients complain to have dyspnea, coughing, and pain in the chest. In contrast to the silicosis, coughing is observed more often with pneumoconiosis of grinders. Often, this is coughing with expectoration. At objective examination, as a rule, lung emphysema is observed (primary moderately expressed) and more often than with silicosis, dry and sometimes bubbling rales can be heard.

Thus, pneumoconiosis of grinders is more often manifested with bronchitis (and perhaps in some cases it goes before pneumoconiosis or accompanies it). Workers on damp grinding often have the diagnosis of inflammatory disease of respiratory tracts, caused by inhaling small drops of liquid, used when grinding (petroleum oils and their emulsions, alkaline solutions, kerosene, etc). But the dust concentration with damp grinding is smaller, thus pneumoconiosis develops more rarely.

Radiological and morphological pattern among patients with pneumoconiosis is characterized by diffusive interstitial fibrosis with main localization in lower and mean zones of lugs. Fine nodular forms of fibrosis can be met much more rarely.

Pneumoconiosis of grinders can be rarely complicated with tuberculosis (3. 1 %) and generally it is characterized by non-malignancy of the progress. Forecast of this pneumoconiosis is mostly connected with bronchitis development, how marked emphysema of lungs is and the complications of a non-specific infection. Pneumoconiosis of electric welders. Prolonged inhaling of multi-component of electric welding aerosol can condition the development of pneumoconiosis, which refers to pneumoconiosis from inhaling mixed dust with insignificant among of free silicon dioxide and iron and has some clinical and radiological peculiarities.

Pathologic and anatomic pattern. For pneumoconiosis of electric welders, a morphological substrate is interstitial fibrosis with relatively small accumulation of dust. In lungs, sclerosis is present with

dust depositing, and thickening of alveolar septums; in lymphatic glands - dust, and development of conjunctive tissue, which in contrast to silicosis, does not cover the total territory of a lymphatic nodule. It is not characteristic for it to have nodules like with silicosis.

Clinics. The disease appears after a long period of work as an electric welder (in average 15 - 16 years). When working in closed premises, pneumoconiosis can develop within shorter period of time (up to 5 years). On early stages of the disease in most cases, complaints are absent or unclear (some dyspnea after physical activity and rare dry rale). The results of physical examination are rather insignificant too. General state is rather satisfactory as a rule; cyanosis is not cleat; chest form is not changed. In some cases, there is percussion sound with moderate box sounds in lower lateral portions of the chest. Some dry rales are non-permanent. When doing physical examination, the majority of ventilation indications are not changed: in some cases there is some increase of the amount of residual air in compliance with moderate emphysema of lungs.

On the radiological pattern of lungs. The primary period of the disease shows diffusive intensification and deformation of lung pattern. Then, there is spread porous looping, more marked in mean and lower portions of lung fields. On the background there are round formations, which differ by fine sizes, sharp outline and increased intensity of shadows. Absence of the tendency to merge these shadow formations is characteristic. as can be observed with nodular form of silicosis, mostly clusters of radiologically contrasted dust, which contains iron, and in a less amount, it is conditioned by finely popular shadows, and by the development of conjunctive tissue. Dynamic observations prove non-malignant progress of pneumoconiosis of electric welders. As a rule, progressing of the disease to stage III is not observed.

Complication of pneumoconiosis with pulmonary tuberculosis can be met much more seldom, than with silicosis and mostly nuclear forms of tuberculosis are observed. In some cases there is regressing development of the process, connected with graduate emptying of lungs from radiologically contrast dust. Combination of pneumoconiosis of electric welders with chronic bronchitis worsens significantly the disease development. In such cases, clinical pattern of the disease depends on the activity of non-specific infection, as well as clear obstruction of bronchi and lung emphysema. Treatment. Patients with stage I of pneumoconiosis, especially if it is not complicated, does not need specific treatment. They are recommended to have general means of to increase the resistance of the body to the infection, balanced meal, physical exercises, as well as keeping to a work/leisure regime. In case of complication of pneumoconiosis with bronchitis, lung insufficiency, and possible bronchial asthma, a corresponding treatment is carried out.

Verification of the ability to work. Electric welders with pneumoconiosis of stage I with the absence of clinical symptoms can continue working under attentive observation of doctors, but it is necessary to restrict their work in narrow restricted premises. At stage II of pneumoconiosis, in case of its combination with chronic bronchitis, emphysema or tuberculosis, the work as an electric welder is contra-indicated.

Preventive measures. Prevention of pneumoconiosis of electric welders is provided by the improvement of technological process (replacing arc welding with contact one and utilization of special welding machines); provision of effective ventilation; utilization of means of individual protection of respiratory tracts and eyes; as well as medical examination of workers.

Pneumoconiosis Caused by Organic Dust

Occupational diseases of lungs, conditioned by the impact of organic dust, can be referred to pneumoconiosis conditionally, as not all of them progress with the development of diffusive pneumofibrosis. Thus, with biocenosis, which is caused by the action of dust of herbal fibers (cotton, flax and hemp), there are mostly functional disorders of bronchial permeability, sometimes joined with bronchiatic syndrome.

In the result of dust action of grain, flour, tobacco and some types of plastics, there can be changes in lungs with moderate diffusive fibrosis, accompanied by general or allergic reaction.

Biocenosis is an occupational disease among people, who were under the impact of organic fibrous materials for a long time (cotton, flax and hemp).

The main hazard at ginneries and cotton-spinning factories, as well as flax mills is the dust of complex composition, which contains organic and mineral fractions.

With preparatory operation on treating and processing cotton, flax and other fibrous materials, especially when processing coarse low quality raw materials, the dust can contain 20 % and more of free silicon dioxide at the expense of ground contamination. Such dust is silicon hazardous. However, at the majority of textile mills, dust includes, as a rule, only matters of organic origin. It can be contaminated with bacteria and fungi (mostly mold). The permitted concentration for dust of herbal and animal origin (grain, cotton, wool, down, etc) with a mixtures of silicon dioxide is as follows: a) over 10 % - 2 mg/m3; b) from 2 to 10 % - 4 mg/m3; and c) less than 2 % - 6 mg/m3. Pathogenesis. In the basis of clinical pattern of biocenosis, there are disorders of bronchial permeability, which obviously have double origin. On the one hand, they are the results in direct action of agents, which narrow bronchi. These agents are contained in the dust of flax and cotton. Besides, under prolonged impact of organic dust, another mechanism turns on: in bronchial wall, matters are deposited, which are withdrawn during the next contact with dust, and also create bronchial and obstructive, but slower action. Subjectively, this is accepted as breathing obstruction, feeling of pressure and pain in the chest. Specific role in forming biocenosis is played by matters of protein origin, which are capable to sensibilize the body of workers: histamine, etc, contained in organic dust, as well as fungi and bacteria, which contaminate it.

Clinic. Biocenosis symptoms, which appear in several years under work conditions of high concentration of dust, are very characteristics. Patients complain that they feel squeezing, pressure and pain in chest, obstructed breathing, and dyspnea at physical pressure, dray rales and weakness. At first the mentioned disorders appear only when renewing work after a break ("Monday symptom"). With time, they do not stop during workdays too, and then can become permanent. In the basis of subjective symptoms of biocenosis was the peculiar dynamics of disorders of bronchial permeability; it is mostly expressive on those days when after a break, contact with industrial dust takes place again. Repeating themselves, these disorders cause the development of cardiac pathology of bronchi and pulmonary apparatus and cardiopulmonary decompensation, combine with other diseases.

Biocenosis progress is divided into the following stages:

Stage I. When renewing work after a break, complicated breathing, squeezing in chest, coughing and weakness are observed. In the majority of patients, auscultative symptoms are absent; sometimes there are physical indications of initial emphysema and bronchitis.

Stage II. Dyspnea and coughing become more expressive and take place during work, but Mondays are still "one of the hardest days". Expectoration, either mucous or pus-mucous, appears. Dyspnea can be significant, sometimes becoming characteristic asthmatic fits. As a rule, there are clinical and radiological indications of bronchitis and emphysema of obstructive and restrictive type.

Stage III. Light gaps disappear; subjective disorders take place throughout the whole week, beyond work place as well. Objective symptoms include chronic bronchitis, lung emphysema, and sometimes bronchial asthma. Ventilation disorders take place; pulmonary-vascular decompensation is observed. Radiologically, indications of emphysema, Carnification of lung roots and intensification of lung pattern are observed.

There are a number of changes in the organism besides respiratory system, like arterial hypertensia, dyspeptic phenomena, scent depression (amongworkers of flax mills), atrophic rhinitis, laryngitis and dermatitis. Skin tests with the extract of production dust are positive.

Treatment. It is done in compliance with the general principles of pneumoconiosis treatment. In case of complication of biocenosis with inflammatory process, antibiotics are prescribed; and with the development of cor pulmonale - cardiac glycosides and diuretic are prescribed.

Preventive measures. Measures to prevent biocenosis development are similar to those, which are carried out to prevent silicosis.

Theme №3. DUST BRONCHITIS, OCCUPATIONAL BRONCHIAL ASTHMA AND EXOGENOUS ALLERGIC ALVEOLITIS

DUST BRONCHITIS

Dust bronchitis is one of the occupational diseases, caused by prolonged impact of production dust. It is characterized by diffusive inflammation of bronchi and is an initially chronic diffusive endobronchitis.

As a nosological form, dust bronchitis was enlisted in the list of occupational diseases in 1970.

Under modern conditions, dust bronchitis develop rather slowly, in 8 to 10 years of work under dust impact, and can be met in various industries in Ukraine.

List of industries, potentially hazard for the development of dust bronchitis (Yu. Kundiev and O. Krasnyuk)

preliminary processing of fibrous technical cultures (operators of hackling machines and units, raw material sorters)
silicon dioxide
asthma
Plant growing (tractor and combine drivers)
Fibrous herbal dust, fungi and bacteria insemination
Pneumoconiosis, bronchial asthma
Cattle breeding (operators of poultry farms and animal farms)
Ground, plant gases, mineral fertilizers, and pesticides.
Bronchial asthma, exogenic allergic alveolitis
Feed production (operators and shift men)
Dust of herbal origin, biologically active matters (microelelents), fungi and bacterial insemination
Bronchial asthma, exogenic allergic alveolitis
Bakery (elevator operators, millers and bakers)
Grain and flour dust, fungi insemination
Bronchial asthma, exogenic allergic alveolitis
Tobacco production (sorters of leaf tobacco, operators of cigar and cigarette machines)
Tobacco dust
Bronchial asthma

Etiology. The development of dust bronchitis depends in prolonged inhaling of much dispersed dust with small amount of quartz or even without it (cement, herbal, or wooden dust).

The appearance of disease is influenced by the presence of unfavorable conditions of production components: work conditions (microclimate, heavy work, or noise); a number of unprofessional factors (sex, age, smoking, infection in the past, or diseases of upper respiratory tracts).

Pathogenesis. In case of action of dust onto the body, disorder of some systems of protection of bronchi-pulmonary apparatus is observed, like mucociliary transportation, local immunity, and surfactant

system. There are disorders of evacuation of dust portions and secretion function of bronchi on the background of structural changes of ciliary epithelia.

Dust bronchitis is characterized by atrophic and sclerotic changes in all the structures of bronchial tree, which form on the initial basis of the disease already, also by changes of bronchi motor activity, and hypersecretion.

In the pathogenesis of the disease, bronchospasm is very important. It appears in the result of reflector reaction of bronchial muscles onto dust particles or sensitization to allergens, contained in the industrial aerosol (chromium, manganese, nickel, phenol-formaldehyde resins, etc). Pathogenic microflora of respiratory tracts influences the development of the inflammatory process in bronchi and allergization of the patient.

At this disease, decrease of cell and humoral immunity decreases also, and significant meaning is also possessed by some genetic factors, in particular deficit of p1-inhibitor of the protease. Pathologic and anatomic pattern. At dust bronchitis, bronchi, bronchial tubes and alveoli are affected. The action of dust first causes relevant reaction from the side of mucous tunic in the form of bronchi hypersection. Number of goblet cells increases. Reological properties of mucus change, its viscosity are increased. Then cells of ciliary epithelia die, basal membrane, infiltration with lymphoid cells starts. This period is clinically determined as endobronchitis, or dust catarrh.

With time, endobronchitis transits into panbronchitis, and then into peribronchitis. Centers of infections in perobroncial cell are accompanied by perineal sclerosis and transition of inflammatory changes into parenchyma of lungs. This phase of reamed inflammation, which is along formation of various grades of sclerosis expression and obliteration of fine bronchi transits into the third stage - reconstruction.

Thus, evolution of chronic bronchitis can be presented by consecutive pattern of hyperthrophic changes of bronchi with atrophic ones with further development of catarrhal mural deforming bronchitis. Spreading of inflammatory changes in distal sections of bronchial tree is accompanied by the violation of production of surface active matter - surfactant, which causes the development of bronchospasm and assists the appearance of sever complication - obstructive emphysema of lungs.

Clinics. Modern classification of dust bronchitis envisages the evaluation of the stage, period (phase) of the disease, as well as presence of complications. There are three stages of dust bronchitis:

Stage I: irritation is weakly marked bronchitis. After many years of work in contact with industrial dust, there is dry rales with some mucus appears. Gradually with years, it becomes stronger; dyspnea appears, as well as general worsening of health state. Worsening of the disease is rare and does not last long.

During objective examination of the patient, there is clear pulmonary sound with box hint, mostly ion lower portions of the chest. According to auscultative examination, breathing is coarse, dry and, sometimes, bubbling rales can be heard. No significant radiological changes are observed. Pulmonary insufficiency is absent, and within the period of acuteness corresponds to 0-1 degree. Changes in periphery blood are absent.

Bronchitis at this stage has clear phases: relapses or exacerbation. Under conditions of timely treatment and normalization of work conditions and the life method, this process is rarely reverse.

Stage II: inflammatory. Clinic manifestations of dust bronchitis in this stage of the disease are conditioned by a variant of bronchitis progress: obstructive, asthmatic, and inflammatory, which mostly depends on etiological factor. Thus, under the action of mostly quartz dust, as a rule obstructive bronchitis with lightly marked inflammation develops with fast developing emphysema of lung with obstructive genesis. Miners, electric welders, workerswho are in contact with organic dust; mostly asthmatic variant of dust bronchitis develop. Presence of toxic components (metal oxide, formaldehyde, or sulphuric compounds) assists the development of bronchitis with manifestation of infectious process in bronchi, which reminds chronic toxic bronchi with the development of bronhcoectases and pneumosclerosis. In such cases,

frequent exacerbations are observed, with secretion of mucopurulent or purulent sputum, as well as corresponding changes of indications of clinical and biochemical blood analysis.

After the radiological examination, slow intensification of lung pattern, which at the period of exacerbation becomes more marked. Pulmonary decompensation of I or II degree. Initial symptoms of cor pulmonale can be observed. Exacerbations are longer (2 to 3 weeks), and their frequency is up to three times a year.

Thus, bronchitis of II degree enables to determine one variant of progress or another, as a rule, it is complicated bronchitis, which is not reverse in full, in spite of rather intensive treatment.

Stage III - deep bronchitis. Clinical manifestations in this stage are characterized by stable coughing with mucus, dyspnea in rest and pain in chest. During the examination, it is observed that patients are cyanotic, and have hydropic face. Percussion examination shows box sound above lungs, and after auscultative examination - big number of scattered dry and bubbling mixed crepitations can be heard.

Radiological changes of II degree are characterized by marked intensification and deformation of lung pattern (as an indication of dust impact), as well as indications of emphysema. Pulmonary decompensation and cor pulmonale are well marked. Exacerbations are frequent and relapses are rather short, and sometimes are even absent.

Thus, dust bronchitis in this stage is a complicated bronchitis with rather grave progress, for which it is characteristic to combine several syndromes (inflammatory and obstructive or inflammatory and emphysema). The most frequent and grave complication is formation of cor pulmonale with the development of cardiopulmonary decompensation, which leads to the loss of work ability by the patient.

Depending on clinical peculiarities of the disease, there are the following forms of dust bronchitis: emphysematous, bronchospasmodic and inflammatory forms of dust bronchitis.

Emphysematous form is characterized by dyspnea of various degrees, coughing with insignificant mucus, which expectorates hard. Objectively, indications of emphysema, coarse breathing and dry crepitations. Radiologically, it is possible to determine increased transparence of lung fields, restriction of movement and low condition of domes of the diaphragm, as well as increase of bronchial vascular pattern. The function of external breathing is changed after the restrictive type within I-II degrees. On the side of cardiovascular system, presence of cor pulmonale in the stage of compensation, and more seldom - with the insufficiency of blood circulation of I-II degree are possible.

Stage II of bronchitis is particularly marked. Patients complain to have dyspnea in rest, discharge of small among of mostly phlegm. Objectively, acrocyanosis and emphysematous form of chest are found, as well as lower portions of lungs is lowered. There are symptoms of cor pulmonale with insufficient blood circulation of II and III stages with obstructive restrictive type. In the peripheral blood, there might be inflammatory changes. In case of overbalance of asthmoid syndrome, in stage I of bronchitis, some breathing obstruction is observed, which appears periodically during the contact with the dust factor and changes of weather conditions. Physical changes are insignificant in this case: during percussion examination, lung sound can be heard with some dry crepitations, in particular during forced breathing when the patient is in horizontal position. In the peripheral blood, eosinophile is found. As to the functions of external breathing there can be Ist degree disorders of obstructive type. During stage II- periodical fits of dyspnea, as well as asphyxia increase. Over the lungs, there is box sound in basal portions, as well as coarse breathing, exhaling is prolonged, and crepitations are dry and whistling. In the blood, eosinophile is increased, and in the prhlegm, there are elements, characteristic for bronchial asthma. As to the cardio-vascular system, there are indications of cor pulmonale in the stage of compensation or with insufficient blood circulation of Ist degree; the function of external respiration - the reduction of within I-II degrees of obstructive type. Clinical pattern of stage III reminds the secondary bronchial asthma. Present frequent fits of asphyxia, and dyspnea in rest, coughing, expectoration is difficult to come out. There is emphysema of

lungs, big number of dry crepitations. In the blood and phlegm testing - sings of allergic component. Cor pulmonale with insufficient blood circulation diagnosis is often to be made.

In case of overbalancing of inflammatory process in bronchi - clinical pattern of bronchi reminds common infectious bronchitis. Characteristic indications of the version are coughing with phlegm. At the initial stage, the latter has mucous, then mucopurulent and purulent character, increased temperature, fatiguability and increased hyperhidrosis. In lungs on the background of box shade of percussion sound, there are dry, and further crepitations. There might be areas of muted percussion sound. In the periphery blood, there is neutrophyl leukocytosis and ESR increase. Function of external respiration is decreased.

Diagnostics. Diagnostics of dust bronchitis is carried out in two stages: at first, it is envisaged to make a diagnosis of the chronic bronchitis as nosological form and definition of the degree if its severity, then dust etiology of bronchitis and thus occupational category of the disease. The first stage of the diagnostic process is based n the record of clinical manifestations of the disease (anamnestic data, patient's complaints, results on physical examination) and auxiliary methods of research (functional, radiological and endoscopic ones).

When deciding on referring chronic bronchitis to an occupational disease, it is necessary to follow the following criteria:

1. Presence of sufficient stage of work under conditions of dust action (7 to 10 years and more). Work period under dust conditions should be supported by a corresponding written in a work record book of the patient.

2. Unfavorable conditions of work, supported by sanitary and hygienic characteristic (presence of dust on a work place with mentioning its concentration and composition), irritating gases, unfavorable microclimatic factors - changes of temperature, humidity and hard physical work).

3. Peculiarities of the development of chronic bronchitis - beginning and character of the disease development, presence of carried disease, especially pneumonia, described and proved by the extract from the outpatient record book of the patient.

Dust etiology of bronchitis is rather easily stated in the case when there is explicit development of the disease of bronchial and pulmonary system under conditions of the dust factor. Anamnesis indications of frequent diseases of bronchial and pulmonary system and much smoking can make the process of stating the professional character of the disease more complicated. But it is necessary to remember that in case of long work period, connected with the action of the production dust, in spite of the previous acute infections of respiratory breathing, it is difficult to exclude the impact of dust onto the development of chronic bronchitis. In case when a worker, whose past has an indication of acute bronchitis and pneumonia, but at the time of getting a job, which deals with dust factor, he/she was considered as healthy (what is indicated by a corresponding record), then was considered healthy and only in some time, he/she got chronic bronchitis, then this disease should be considered occupational as well.

When chronic bronchitis is a direct outcome of acute infection disease of respiratory organs, the issue on the connection of chronic bronchitis with the work conditions is solved individually first of all, with the consideration of work conditions and work period of the patient. Very often it is necessary to exclude unfavorable impact of production factors onto the development of the disease, which will enable to say about a joint genesis of hazard factors, first of all, dust and infections. In this case, they say about chronic bronchitis of the joint genesis (dust infection).

Based on complaints of the patient, changes, found during clinical examination, as well as presence of data from the listed above officialdocuments, and differential approach diagnosis of dust bronchitis can be made. In compliance with the traditional clinical classification of dust bronchitis in making the diagnosis after nosologic form of "dust bronchitis" in compliance with the stage (I, II and III) show clinical syndrome, which prevails in the clinical pattern (inflammatory, asthmatic, and lung emphysema) and its explicitness, as well as the degree of luminary and heart decompensation. To clarify the degree of lung decompensation

by following the traditions of deviation of indications of external respiration from the norm (M. M. Kanayev)

When diagnosing dust bronchitis, it is necessary to determine the process activity. Dust bronchitis develops with periodic exacerbations, with which its progressing is connected, but these exacerbations do not manifest with clear indications usually, what shows the activity of pathological process. Such generally accepted indicators f the activity of the inflammatory process, as body temperature, ESR, number of leukocytes, leukocytic blood formula, biochemical indicators (C-reactive protein, sialic acids, sulfur-mucoid, as well as haptoglobin), and during acute condition of bronchitis, they can be very unclear. That is why during the period of exacerbation, it is necessary to pay particular attention to the changes of clinical manifestation of the sick, reduction of their ability to work, sign of brocnhospasms, weakness, increased hydrosis, increased coughing, signs of bronchospasms, and appearing of mucopurulent sputum show the exacerbation of dust bronchitis.

With dust bronchitis, there is not always an opportunity to consider the dynamics of the process in two opposite phases - exacerbation and remission. Often after massive course of treatment in hospital, patients are released with some indications of delayed exacerbation. This condition should be considered as the stage of fading exacerbation, which envisages corresponding recommendations regarding the following outpatient treatment and regime. Sometimes, patients have clear clinical exacerbations, however some objective sings of bronchitis are increased (a threat of exacerbation). Corresponding job, and out-patient treatment) can prevent the appearance of exacerbations and loss of ability to work.

Thus, diagnostics of dust bronchitis is based on thorough recording of results of clinical and assisting research, career data and data on labor conditions.

Treatment. Tactics of treatment of dust bronchitis is based on the results of examination of patients and is conditioned by mostly symptoms, functional state of the external respiration, blood circulation, nervous and other systems, presence and explicitness of an allergic component, as well as the state of immune reaction.

Considering that at dust bronchitis, there is the development of atrophic processes in mucous tunic of the bronchial tree on the first stages of the disease; main treatment should be directed at the increase of general reactivity of the organism, stimulation of general regenerative processes in the mucous tunic of bronchi, as well as liquidation of bronchospasms.

First of all, it is necessary to tell about the utilization of means, which stimulate processes of epitelization. Such properties are possessed by methyl-uracyl, which is given in the dose of 1 g 3-4 times a day after meals. It is also possible to prescribe 4 % solution of calcium pantothenate, which is given in the form of 4 % aerosol inhalation - 10 ml every day. The course consists of 10 to 12 inhalations.

Patients, who mostly have bronchospasms, are prescribed sympathomimetic agents: isadrin and novodrin, which are taken in the form of aerosols. Of some advantage are medicinal drugs of the same group in small dosated tanks: asthmopent, alupent, and berotek. Optimal dose for inhalation is two inhaling, which are repeated 4 to 5 times a day. When treating patients with dust bronchitis with disorders of bronchial permeability, aminophylline is widely used - 2. 4 % IV solution 10 ml dose.

When treating patients with dust bronchitis, medicinal drugs are used which improve phlegm discharge, like althaea root, termopsis herb, and potassium iodide, as well as means which have mucous solving action, like mucous solving inhalations and sodium chloride.

Considering the important role of an allergic component in the development of bronchitis, patients with this pathology are prescribed to use antihistamine products, like Dimedrol, diazoline, and phencarol.

When treating patients with inflammatory version of dust bronchitis, main place is occupied by medicinal drug therapy, aimed at liquidation of inflammatory process and prevention of the process transfer into a chronic form. Infectious etiology of the given form of the disease is conditioned by utilization of a

corresponding therapy (antibiotics, sulfanilamide, etc) with simultaneous conduct of measures to increase protective immunity of the body.

The most well spread medicinal drug to treat patients with inflammation, caused by pneumoconiosis and streptococcus is penicillin. One-time dose is 300 000 - 600 000 OD every 3 to 4hours, or 2 000 000 - 5 000 000 OD a day. Medicinal drugs of the group of semisynthetic penicillins, like ampicillin and oxacillin are widely used (daily dose is up to 4 g, 0. 5 g - 4 to 6 times a day, one or up to three hours before meals).

Out of sulfanamide medicinal drugs, sulfalen and sulfadoetoxia are prescribed (the first day - up to 2 g, and then 1 g for 8 to 10 days).

As preventive measures against dysbacteriosis, Nystatin is prescribed: 2 000 000 - 4 000 000 OD a day.

An important role in treating diseases of lungs are played by endobronchial sanitations with introduction of necessary medicinal drugs.

Verification of the ability to work. Issues of the verification of the ability to work for those, who have dust bronchitis, are solved individually, with the consideration of the severity of the disease, age, work period, occupation of the sick and work conditions.

The patient with dust bronchitis of stage I can continue working in the competence of his/her occupation with obligatory dynamic medical examinations (not less than twice a year).

Patients with bronchitis of stage II are subject to rational employment, which is not connected with the influence of dust, irritating matters, and unfavorable weather factors as well as without significant pressure. The decrease of qualification at transfer to another job is the basis to send the patient to the Treatment and technical verification commission to get disablement status (as a rule, it is group III) due to the occupational disease. Patients with stage III of bronchitis due to frequent cases of exacerbation of inflammatory process and the development of cardiac and pulmonary decompensation lose the workability completely and often need external help, what conditions the need for them to get disablement group II or I in the result of the occupational disease.

Preventive measures. Main preventive measures of bronchitis is the conduct of technical, sanitary and hygienic measures, aimed at further improvement of the work environment for workers of dusty professions.

Measures of medical preventive measures, first of all, a quality conduct of medical examinations, both preventive and periodical, are very important. Another important preventive measure for dust bronchitis is timely and rather long treatment of acute inflammatory diseases of respiratory organs, active anti-smoking campaign.

OCCUPATIONAL BRONCHIAL ASTHMA

Occupational bronchial asthma is the disease, main manifestation of which include fits of asphyxia, conditioned by bronchospasms, hypersecretion of bronchial glands, swelling of mucous tonic of bronchi, and which is etiologically connected with the action onto the bronchial apparatus by the agents on the workplace of a worker. Thus, occupational bronchial asthma, which is observed under various production conditions, is etiologically connected with the impact of occupational factors.

At the meeting of the WHO in Geneva in 1980, bronchial asthma was listed among other occupational diseases. Whereas it was stressed that the main criterion to recognize occupational etiology of bronchial asthma is the presence of connection of its appearance with the work conducted.

Some epidemiological research showed that 2 to 14 % of all the patients with asthma suffer from professional bronchial asthma. The frequency of professional bronchial asthma much varies in various occupational groups. Thus, it is considered that among those farmers who contact with animals and birds, about 6 % of the people have asthma, and as to those who work in bakeries - about 10 % have asthma.

Etiology. In the etiology of occupation bronchial asthma, an important role is played by the following matters: allergic agent of animal (wool, silk, hair, feather, pieces of epidermis, bees and helminthes) and plant (pollen of herbs, bushes, trees, flowers, wooden, grain and flour dust, volatile oil, flax and tobacco) origin; a large number of chemical matters (Ursol, metal compounds -chromium, nickel, cobalt, manganese; formalin synthetic polymers, dyes, and pesticides); medicinal drugs (hormones, vaccines, ferments, protein and vitamin concentrates, as well as forage antibiotics). Among medicinal drugs, the most important are antibiotics (especially penicillin, more seldom - streptomycin, biomycin, and tetracycline), as well as vitamins, sulfanilamide, analgetics, hormonal drugs and aminazine. In principle, etiological factors can be divided into the two following groups: allergic agents and asthmogenic agents. The former include flour, especially, wheat flour, natural silk, epidermis of animal fur and skin, castor oil; dust of green coffee beans; detergents; and various medicinal drugs. The latter one includes wooden dust, cotton, PVC, lacquers, pesticides and phenol.

Pathogenesis. In case of contacts of a worker with occupational allergic agents, int the body there is the increase production of antibodies of class IgE. The latter are fixed on mast cells (immune stage), after what degranulation of mast cells with the discharge of a great number of bronchospastic and vasoactive matters - histamine and serotonin (pathochemical stage) takes place. Under the impact of biologically active matters, permeability of microcirculatory flow is increased; swellings, severe inflammation and bronchospasm (pathophysisological stage) develop. Clinically, this is manifested by the disorder of bronchial permeability as well as the development of fits of bronchial asthma. This is a so-called atopic occupational bronchial asthma, in the genesis of which reagent type of immediate hybersensitivity takes place.

Prolonged impact of astmogenic agents causes changes in the reactivity of target cells (first of all, mast cells, located along the respiratory tract). Change of reactivity of these cells is first of all accompanied by excessive production of biologically active matters (histamine and leukotriene). In the response, bronchial spasms, swelling of mucous tunic, and hypersecretion of bronchial glands develop. All these change the permeability of bronchi much and cause asphyxia fit.

An important meaning in the development of professional bronchial asthma is also caused by heredity and genetics, as well hormonal disorders, misbalance of vegetative nervous system, and respiratory infections. As to the last factor, it is considered that there are several variants of the interaction of allergy and infection: as to the first - infection in he bronchial tree causes formation of bacterial allergy, which causes asphyxia; as to the other one -infection improves the permeability in the tissue of infectious allergic agents, and as to the third one in the opposite- sensitization of the organism is an infection "conductor". The development of infectious - inflammatory process in bronchial tree of those who have occupational bronchial asthma are assisted by atrophic processes in the mucous tunic (result of the contamination of the production environment with matters of irritating action: solvents, acids, alkaline, vapors and gases of various toxic matters). This is manifested by intensified hemorrhage of tissue metabolites and stimulates the production of autoantibodies. The same is caused by sensitization of organism to agents of infectious processes in the bronchial tree.

Along the intensification of infectious and allergic component on the background of the occupational allergy in the pathogenetic process, besides the reagent type of allergy, there are other types of allergic reactions. In the blood, the number of circulating immune complexes is growing (joining of industrialand infectious allergy agents with antibodies of IgA class). These complexes activate the system of complement, and in the result, the pathologic process includes kinin, neutrophilic and macrophage, as well as some other systems. Pathological process develops with more severity with asthmatic exacerbations. This very joining of infectious and allergic component causes the development of hypersensitivity of slow type (there are chemical and toxic factors of lymphocytes, and T-factors are activated). Thus, pathogenesis of the given version of the occupational bronchial asthma is close to infectious-allergic one.

Possible variants of the participation of the infection in the development and formation of a specific pattern of bronchial asthma in the clinics of professional diseases are shown in the following scheme:

Infection

Non-infectious-allergic occupational bronchial asthma (occupational asthma caused by occupational allergic agents

Infectious-professional bronchial asthma (complications of a chronic occupational respiratory pathology of nonealergic genesis)

First manifestation of sensitization to occupational allergic agents on the background of a respiratory disease

Acute stage on the background of acute respiratory disease

Sensitization of microflora respiratory tract

Acute stage (complication) on the background of an acute respiratory disease

Assisting stabilization on the background of an occupational non-infectious allergy with the development of conjunctive sensitization - non-infectious+ infectious

Pathologic and anatomic pattern. People, who died from asphyxia, had swollen lungs. Bronchitis has viscous glass like content, with a big number of eosinophilic granulocytes, Kurchman's spirals, and Charco-Layden crystals. Eosinophilic infiltration of bronchi walls and the thickening of the basal membrane of their mucous tunic take place. Formation of perivascular infiltrates with mononuclear cellular profile and granulation changes in the mucous tunic of the perivascular genesis should be determined as a specific peculiarity of the occupational asthma.

Classification. There are two main forms of bronchial asthma:

• occupational bronchial asthma, which is very much like atopic asthma; and

• occupational bronchial asthma of the joined sensitization (occupational and bacterial allergic agents).

In comparison with the general clinics, these are correspondingly: atopic (immune) and infection-dependant (non-immune) bronchial asthma.

Besides, it is necessary to take into consideration the gravity degree (light, mild and severe), progress phase (acute state, fading acute state and remission) and complications (lung emphysema, pulmonary collapse, pneumothorax, miocarda myocardium dystrophy, cor pulmonare, cardio-pulmonary decompensation, etc).

Clinic. Main clinical manifestations of the occupational bronchial asthma is a fit of asphyxia. Mostly it appears at night. The beginning of it is the feeling of stuffed nose, coughing and complicated breathing. Inhaling and especially exhaling are obstructed. Abdominal muscle tensing is observed. Breathing is noisy and accompanied by distant buzzing and whistling crepitations. the patient has to take a specific position (sitting and supporting himself/herself with arms), where shoulder belt is fixed: shoulders are moved up and forward, the head is like drawn into the shoulders, the chest is in the aspiratory state, and movement are limited. Cyanosis of lips, and the top of the nose is observed.

Above the lungs, there can be hears box percussion sound. And after auscultative examination, breathing is coarse with prolonged exhaling. Dry buzzing and whistling crepitations can be heard.

Pulse is frequent, heart flattening is not determined (the result of the emphysema presence), and the body temperature is normal or increased. On the ECG in the IInd and IIIrd standard portions, more pointing waive is observed.

The fit is over with the discharge of viscous phlegm of gray color and the renewal of normal breathing.

As to the frequency and expression of asphyxia or asthmatic syndrome fits, as well as the respiratory compromise, complications and the disease character, the progress of the occupational bronchial asthma can be divided into light, mild and severe.

At the light progressing of the occupational bronchial asthma, asphyxia fits are rare (1 to 2 times a month or less), they last for several minutes to half an hour, and usually they are light on the background of pleural administration of bronchiolitic means. Sings of worsening of bronchial permeability appear in during mild or significant physical activity, sometimes, on the background there are light whistling crepitations, coughing or asthmatic fits. In-time termination of the contact with the professional allergic agent, as a rule, leads to clinic convalescence.

Light disease progressing is characteristic to occupational bronchial asthma, which is like atopic one, as after the timely termination of the contact with production factors, remission takes place. With these forms of the disease, the development of emphysema can be observed though a comparatively long period (8 to 9 years), and some patients can have periodical light asphyxia fits, caused by the action of sharp odors, as well as physical and emotional tension.

For the mild disease progress, it is characteristic to have asphyxia twice or three times a week, which last for one hour (rarely, more); they terminate after an injection or aerosol inhalation. Between the fits, patients can have periodical crepitations in the chest and some complicated breathing. Worsening of the state is observed with moderate or insignificant physical activity. Termination of the contact with an occupational allergic agent is accompanied by significant improvement - typical fits of asthmatic state terminate, though expiratory dyspnea and coughing appear periodically.

Severe disease progressing is characterized by the appearance of frequent, often daily fits of asphyxia, up to the development of asthmatic state. Complicated breathing takes place during insignificant physical activity. To receive therapeutic effect, there is the necessity to use corticosteroid hormones. Termination of the contact with the production is not accompanied by the improvement of the patient's state.

Mild and severe progress of the occupational bronchial asthma is characteristic fro asthma of joined sensitization (professional allergic agent and bacterial one). Due to frequent acute condition and absence of remission, lung emphysema and signs of cor pulmonale among these patients, in spite of the rational change of occupation, appear already in 3 to 5 years after the beginning of the disease. There is also an opportunity of the development of broncvhoectasies, chronic pneumonia and asthmatic status. Severe and prolong fits of bronchial asthma, as well as spread obstruction of bronchiole with viscous phlegm can become a direct reason of the death.

Between fits, clinical signs of bronchial asthma can be absent. This state is more characteristic for initial stages of he disease, and in more marked stages of bronchial asthma, even between fits, there are the following indications: complicated breathing, moderate dyspnea at physical activity, coughing with mucous phlegm. Coarse breathing can be heard in lungs, often with dry crepitations, especially when breathing is forced.

Patients with occupational bronchial asthma have changes in peripheral blood (eosinophilia, Kurshman's spirals, crystals of Charko and Leyden), protein spectrum of blood serum, increase of the level of histamine, reduction of excretion of 17-hydroxy-costicosteriods with urine. Development of occupational bronchial asthma depends on the peculiarities of the occupational anamnesis (character and type of allergic agents). Thus, for the occupational bronchial asthma, which is like atopic one, presence of symptoms of exposition and elimination (appearance of fits of asphyxia when contacting with the allergic agent and its termination after the termination of the action of an allergic agent). Especially, it is obvious after the rest (vacations and weekends) during the period of the so-called monovalent sensitization. Timely rational work change at the stage of pathologic process can prevent its further progressing. In case of continuation of work under conditions of occupational factors, which had caused the disease, its progress leads to worse conditions due to the development of polyvalent allergy. During this period, termination of the contact of

patients with occupational factors does not bring the improvement of their condition (the period of polyvalent sensitization).

The first fits of asphyxia of such patients follow allergic affections of upper respiratory tracts and skin. In the peripheral blood, there is eosinophilia. And in the phlegm there are eosinophiles as well as Kurshman's spirals.

Under the condition of the influence of matters, which cause local irritation onto the respiratory organs or cause dust (toxic and dust) bronchitis or pneumoconiosis, occupational bronchial asthma of joint sensitization develops. For such a form of disease, it is characteristic to have no clear elimination syndrome, thought worsening of the state with more frequent fits of asphyxia takes place among the patients of the group as well, when they renew their contact with occupational factors (exposure symptoms). When the sick terminate their contact with an allergic agent, asphyxia is not replaced by the complete remission though. In the clinical pattern, there are symptoms of inflammatory process in the bronchial tree, and upper respiratory tracts. Mucopurulent sputum is discharged, where pathogenic bacteria are seeded.

The sick are characteristic to have subfebrility as well as insignificant leukocitosis. Gradually, the number of asphyxia fits increases, they also have worsened dyspnea, and not only at physical activity in contact with an allergic agent, but also due to irritating cold. And with this form of occupational bronchial asthma, a pattern and frequency of joining asthma with allergic changes in the upper respiratory tracts and skin exist. Usually there is no heredity in complicated allergic diseases. As a rule, in all the cases, initial fits of asphyxia are interconnected with infectious - inflammatory diseases of respiratory organs in the form of repeated respiratory infections, acute bronchitis and pneumonia. Inhalation testing with occupational allergic agents proves the development of an allergic reaction on the immediate-slowed down type.

After radiological examination, patients with asthma joint with allergies, have the intensification of vascular - bronchial pattern in lower portions of lungs. In some cases together with this, there are pleurodiaphragm commissures in the result of infectious-inflammatory diseases of respiratory organs. The equivalent of the bronchial asthma is the asthmatic bronchitis, which is evident through expiratory dyspnea, absence of large-scale fits of asphyxia, as well as presence of catarrhal phenomena in the lungs when production allergic agents with production dust or irritating matters. In the anamnesis of the development of asthmatic syndrome, there is acute respiratory viral infection, bronchitis and pneumonia. The symptom of elimination in the clinical pattern of the disease is absent. Radiological examination allows determining the intensification of vascular-bronchial pattern in lower portions of lungs, and pleural-diaphragm commissures. As a rule, inhalation testing eliminates positive reaction to immediate-slowed down and slowed-down types.

Diagnostics. Clinical manifestation of the occupational bronchial asthma does not differ from those, which take place with the asthma of different etiology. Specific difficulties can take place in the process of definition of the etiologic factor in the genesis of this or that form of asthma. Thus, it is very important to study the occupational anamnesis of the patient, sanitary and hygienic characteristics of his/her workplace, as well as the data on allergen anamnesis, clinical manifestation and immune methods to examine a patient.

The presence of the contact with industrial allergic agents, production dust and irritating matters, fits of asphyxia at work and significant improvement of the state during vacations or staying at hospital on sick leave, correspondence of the clinical pattern, as well as all the factors, which can assisting the development of asthma (heredity, hormonal disorders, diseases, life conditions, etc), enables to suspect occupational bronchial asthma which needs specific allergen examination.

Methods of allergen examination, which need immediate participation of the patient (skin allergen tests and provocative inhalation testing) is conducted in case of satisfactory feeling of the patient during the remission stage. General contra-indications to use these methods of diagnostics are acute fever states and inflammatory processes; active TB form, pregnancy, decompensation diseases of heart, liver and kidneys; thyrotoxicosis; as well as complicated forms of bronchial asthma.

Mostly, scratch test or internal tests are used. To carry out scratch test, one drop of allergen is put onto the palm portion of the forearm, and through it the scratch is made. The reaction is assessed in 20 to 30 minutes, then 24, 48 and 72 hours. As a rule, immediate positive reaction takes place. When conducting of the allergen reaction under skin, it is necessary to administer from 0. 05 to 0. 1 ml of the allergen, which contains one skin dosage. Positive reaction is of the slowed-down type and it is assessed in 24, 48 and 72 hours since the administering of the allergen.

Provocative inhaling testing is conducted only in the phase of bronchial asthma remission and only in hospital. After the percussion and auscultative examination of lungs, spirogram is taken with the definition of Tifno index. Then within 3 to 5 hours, test-control liquid is given to the patient through anaerosol inhaler. If within 5 to 10 minutes, the patient does not feel worse, another spinogram is made and in case of absence of significant sighs, inhalation of the least concentration of allergen is conducted for 2 to 3 minutes. After this, characteristics of the Vital Pulmonary Capacity are checked, as well as indexes of forced exhaling in 20 min, 1 hour, 2 hours and in 1 day. Provocative inhalation testing is considered positive, if VPC is reduced by 10 %, and Tifno index - by 20 % comparing with initial data.

Among the *methods of allergen laboratory diagnostics* to find out sensitization to industrial allergens, the following are used:

• reaction of a blood cell to the hapten in vitro - reaction of specific blood leukocyte accumulation (RSAL), tests on damage and alternation of blood neutrophyls (PPN) and reaction of direct specific damage of blood basophiles (RSPB);

• serologic reactions - reaction of compliment binding (RZK) and reaction of passive hemagglutination (RPGA).

Specific cell reactions on hypersensitivity in vitro - reaction of specific rosette formation (RCR), reaction of termination of blood leukocyte migration (RGML)

Each method of diagnostics with the attraction of the given above reactions is based on specific peculiarity. Thus, RSAL of the periphery blood -on the effect of intensification of adhesion of white blood cells in case of adding to it a specific allergen of the reaction cell, which is one of the first phases of specific allergic reaction of the blood cell. Reaction is assessed as positive when RSAL is equal to 1. 4and higher. PPN - on the immune phenomenon, which develops according to the reaction type of target cells onto the immune complex, which is created in the serum in the result of adding a specific antigen. Reaction is defined as positive when the indicator is 0. 05 or higher. RSPB - due to the fact that blood basophiles and mast cells of the connective tissue serve as target cells in realization of reactions of immediate action. Reaction is positive if the indicator is 1. 4 and higher.

Only complex evaluation of the occupational and allergologic anamneses, of the corresponding documentation regarding the conditions of work and dynamics of the disease, and also results of specific allergologic and immune examination of the patient enable professionals to state the professional genesis and etiological factor of bronchial asthma.

Treatment. Treating methods with occupational bronchial asthma should take into consideration the data on etiological and pathogenesis. With atopic non-infectious form, especially in the initial stages, termination of the contact with production factors can cause disappearing of fits.

The most grounded method of treatment of bronchial asthma is specific hyposensitization of the body. However the complexity of defining the majority of allergens of the occupational character, short term of the achieved effect, athreat of the development of complications (anaphylactic shock) do not let us consider this therapy method as efficient.

In complex treatment, it is important to liquidate the concentration of the chronic infection. Among recent medicinal drugs, particular attention is paid to the drugs which mostly stimulate p2 -adrenoreceptors of bronchi. In particular, they include salbutamol, terbutalin, and alupent. It has been proved that in comparison with other adrenoceptor agonists (adrenalin and ephedrine), which influence not only p1 and a-

adrenoreceptor and assist to the increase of the arterial blood pressure, tachycardia, anxiety, increase of and asphyxia; but they have less influence onto the cardio-vascular system.

At the same time, aminophylline (IV of 5-10 ml of 2. 4 % solution into 10 - 20 ml of 20 % solution of glucose) is still the very therapeutic method of treatment of patients with occupational bronchial asthma, used most often. To prevent fits of asphyxia, it is possible to use retarded forms of theophylline -theopec and retafil.

Besides bronchodilatory methods, antihistamine drugs are often used to treat patients with bronchial asthma: Dimedrol - 0. 03-0. 05 g 1-2 pills a day; phencarol - 0. 025-0. 05 g 1-3 pills. Ketotifen inhibits release of histamine from mast cells, and they are prescribed in 0. 001-0. 002 g in the form of pills or capsules twice a day. Disodium cromoglycate as a method of biochemical preventive measure, stabilizes the membrane of mast cells and does not let release of biologically active matters from them, and they are prescribed in the dosage of 20 mg in the from of microionized powder four times a day using an inhalator. Calcium channel blocking agent are prescribed to patients with bronchial asthma on the background of physical tension, as well as to those who suffer from ischemic heart disease. Glucocorticsteroids are administered only then when all usual methods of treatment did not give the expected effect. Prednisolone is prescribed in pills 0. 005 g; in acute cases, treatment starts with 20-40 mg a day, after it achieves the curing effect, the dosage is reduced to 5-10 mg and less. In emergency cases, prednisolone is used for injections. It is prescribed intravenously or intramuscularly in the dosage of 100-200 mg a day. It is also possible to use synthetic steroid hormones - beclometasone in the form of aerosol for inhalations.

Expectorant and antitussive methods: 3 % solution of potassium iodide in the dosage of 0. 3 to1 g a day; Tarasov's mixture internally - 1 teaspoon - 1 table spoon with warm milk - 3 - 4 ties a day after meals.

Antibacterial means, particularly when there is purulent bronchitis, arthritis, and pneumonia; ampicillin and biceptol.

Immunomodulators: considering the fact that patients with bronchial asthma have reduced activity of the T-immunity, decaris is used, 100 mg - the first four days in a row with a two-day break.

Among non-medicinal methods of the therapy for patients with occupational bronchial asthma, reducing diet therapy, needle reflexo-therapy,curing gymnastics, respiratory gymnastics, physiotherapy (ultraviolet, ultrahigh frequencies, and electrophoresis), sanatorium-and-spa treatment (Crimea) and pneumatotherapy.

Verification of workability. When making decision on workability and job of patients with bronchial asthma, it is always necessary to remember that independently from the degree of the disease severity, they are contra-indicated the contact with matters of sensitized and irritating action, staying under unfavorable meteorological conditions and significant physical activity.

Workability of patients with bronchial asthma of mild degree is usually kept, but they need rational job.

When bronchial asthma of mean severity among patients can be significantly restricted or completely lost. In connection with the development of respiratory insufficiency and decompensation of chronic cor pulmonale of patients with bronchial asthma of the severe degree, as a rule, inability to work, and many of them require external assistance and supervision.

Preventive measures. The task of medicinal preventive measures is to keep workability of workers and employees, and to prevent development of occupational medicinal examinations to select those who had to start working under conditions of possible contact with allergens. It is also important also to define initial sings of the disease and rational work beyond contacts with production allergens.

EXOGENOUS ALLERGIC ALVEOLITIS

It is a general term of the group of allergic pneumonias, which progress with involvement into a diffusive dispersed inflammatory process of some groups of alveoles.

Etiology. The reason of the development of exogenous allergic alveolitis is the allergen, which enters the organism with inhalation, together with the inhaled air. Such allergens can be weevil (wheat), extract from the dust of cacao beans (cacao beans), serum protein, antigens of bird droppings (feature and droppings of pigeons, chickens, and parrots), thermophilic actinomycetin (rotten hay), penicillin (medicinal drugs), salts of heavy metals (chemical matters), etc.

Size and number of particles are very important in the development of alveolitis. It is considered that particles up to 5 micromicrons easily achieve alveoli and are capable to cause sensitizations.

Pathogenesis. Allergen, which gets to the organism, causes sensitization, accompanied by the creation of antibodies. These precipitant antibodies together with allergen create immune complexes, capable to deposit in the walls of alveoli, and bronchial tubes. They cause inflammation (bronchiolitis and alveolitis), increased permeability of vessel walls (due to discharges of mast cells and basophiles of vasoactive amines), formation of granulomas (granulomatous pneumonitis), which leads to the development of interstitial fibrosis and disorders of ventilation function of lungs of the restrictive type. Pathologic and anatomic patter. For an allergic alveolitis, it is characteristic to have granulomas in the walls of alveoli and bronchioles, as well as inflammatory infiltration of lymphocytes and plasmatic cells, as well as accumulation of exudation. Granulomas consist of epitheloid cells, which in the center are surrounded with lymphocytes and plasmatic cells. On the later stages of pathological process, pulmonary fibrosis is present.

Clinics. Clinical pattern of the disease is characterized by general symptoms (fever, pain in muscles, reduction of body weight). Sings, connected with the affection of respiratory organs, show involvement of bronchioles and alveoli into the pathologic process.

Often the disease starts with the growing dyspnea and coughing. When using auscultative methods, it is often possible to hear crepitations, mostly in interscapular regions. Acute form can be recognized rather easily. At functional research, decrease of blood saturation with oxygen, increase of partial pressure of CO2 in the arterial blood, also clear respiratory alkalosis can be observed. Pulmonary capacity is reduced in the majority of cases, in particular, lung vital capacity.

Alveolitis can be chronic. It develops in the result of repeated less intensive influences of disease causing agents in several months after coming across them and are characterized with progressing respiratory insufficiency. Patients are bothers with dyspnea, sometimes with moderate fever and drowse. With X-ray examination, interstitial fibrosis can be observed.

One of the examples of allergic alveolitis is "farmer's or thresher's lung". That is the disease when inhaling of organic dust causes the reaction of increased sensitivity on the alveolar level, connected with the production of precipitin, and which is characterized with allergic diffusive affection of alveolar interstitial structures of lungs. The disease can be met among agricultural workers, which come across damp moldy hay, grain, silo and other herbal materials. It is more often can be observed in winter and autumn period of the year, when hay stocks are used as feed for domestic animals. Mostly, the development of the "farmer's lung" disease is caused by thermophilic actinomycetes: Micropolyspora faeni and Thermoactinnomyces vulgaris.

Acute forms are characterized by their sudden initiation. In 3 to 6 years after the exposure, temperature suddenly increases up to 39 to 40 °C, headaches appear, as well as pain in muscles, and coughing with poor phlegm, and sometimes with the mixture of blood. Sometimes, there is nausea and vomit, voluminous hidrosis, and progressing dyspnea. During examination, cyanosis, tachycardia, frequent breathing at rest can be observed; and crepitations and single dry rales can be observed during auscultative examination. After radiological examination, intensification of pulmonary picture and small nodular types of different intensiveness can be observed. If the action of the allergen is eliminated, symptoms of the disease disappear in 7 to 10 days. The repetition of the contact with disease causing agents leads to the development of subacute form of the disease, where clinical and radiological indications disappear much slower. There are indications of growing respiratory decompensation in restrictive or obstructive type, but

the latter does not happen often; they have dry coughing, and chill at night. When examining, it is possible to find out that the chest acquires barrel-like look; crepitations in lower portions of lungs can be heard. Radiological examination shows more marked changes in the form of diffusive nodular shadows can be observed in lungs, which are located mostly in mean and lower portions of lungs. The disease stops in 4 to 8 weeks, if further contact with herbal dust is terminated.

Chronic form of the disease appears in the result of constant exposure to insignificant amount of dust of moldy hey to the organism of the human body. In the clinical patter, there is mostly dry coughing, dyspnea at physical activity, subfebrile temperature; total condition is worsening, and body mass is decreasing. At auscultation, crepitations, as well as fine and mean bubbling rales can be determined. If contact with dust continues, irreversible changes can take place - fibrosis of lungs and decompensated cor pulmonale.

At the functional research of external respiratory, restrictive form of ventilation decompensation can be observed. Lung vital capacity is decreased, and their diffusion ability decreases.

Diagnostics. Diagnosis can be made based on the occupational anamnesis (sick people, which do not have inclination for atopic reactions; the disease develops in a rather long-term contact with the allergen), peculiarities of clinical patterns (duration of the latent period, and characteristic signs), as well as radiological changes. The diagnosis is proved after skin testing (with blood serum or an extract of placenta) and serologic research (to find precipitant antibodies with methods of immune electrophoresis and radioimmunology). In some cases, biopsy of lungs or analysis of bronchoalveolar lavage is recommended (increase of T-lymphocytes).

Differential diagnostics. Exogenic allergic alveolitis should be differentiated with sarcoidosis, for which it is characteristic to have absence of the connection with the profession, affection of other organs, besides, lungs, development of hypercalcium areas, increase of near root lymphatic nodules on the radiogram, weak or negative reaction onto tuberculine and positive Quame's reaction.

Alveolitis should be also differentiated from pneumonia of infectious origin, for which it is characteristic to have the connection with colds, segmental or area shadowing on the radiological photograph, as well as expressed intoxication syndrome.

Treatment. The most efficient method of treatment is the termination of contact of the patient with the allergen, which caused the disease. To treat exogenic allergic alveolitis (in particular of subacute and chronic forms), corticosteroids are used. Prednisolone is prescribed in the dosage of 1mg/kg a day for 7 to 14 days, then the dose is gradually reduced.

Verification of the ability to work. Issues as to the ability to work of patients with the disease of lungs, conditioned by the impact of rottening herb dust, is solved the same way as in case of corresponding forms of dust diseases of lungs, caused by other types of dust.

Preventive measures. Main preventive measures for the patients with exogenous allergic alveolitis are in preventing the contact of the patient with corresponding allergens by the change of technological process (decrease of concentration of the allergen in the exhaled air), as well as the usage of respirators and other means of individual protection of respiratory organs.

Theme №4. OCCUPATIONAL DISEASES, CAUSED BY PHYSICAL FACTORS. VIBRATION DISEASE AND SENSORINEURAL HEARING LOSS. ALTITUDE SICKNESS AND DECOMPRESSION SICKNESS

Vibration disease

Vibration disease is a professional disease, caused by vibration. For the first time, this pathology was described by Loring in 1911 as a syndrome of "dead fingers" among scabblers, and in1955, it got the name of "vibration disease".

The main factor, which causes the development of the disease, is vibration. From the physical point of view, vibration is a mechanical oscillation, which is repeated at definite periods.

There is a l o c a l vibration, which impacts mostly onto hands of a worker when working with vibroinstruments, and a g e n e r a l vibration, which impact the whole organism. Local vibration takes place when workers use pneumatic and electric instruments (chisel hammers, riveting hammers and chopping hammers). The impact of the general vibration can be observed in case if a worker works with the vibrating equipment (vibroplatform and automatic concrete distributors), as well as in case of transfer of vibration from working engines, machines and equipment to the floor.

Expression and the time for the development of the disease is determined by the zone of the amount of oscillation energy, which is transferred to the body of a human being or his/her limited part, as well as factors, which assist to the development of vibration disease: forced body position, cooling and noise.

Among occupational diseases, the vibration disease still holds a leading place and is mostly encountered among those, who work in metal processing, machine engineering, metallurgic, construction, aircraft manufacturing, mineral resource industry, agriculture, transportation and many other spheres of national economy.

In Ukraine, vibration disease appears mostly among workers of such professions as cutters, drillers, fettlers, face-workers (impact of low frequency local vibration), riveters, polishers, tool dressers (impact of high frequency local vibration), as well as drivers of heavy earth-moving machines (impact of general vibration).

Pathogenesis. In the basis of the vibration disease is a complicated mechanism of nervous and reflector disorders, which cause the development of nucleuses of stagnant agitation and to further stable changes both in receptor, as well as in various sectors of the central nervous system. A significant meaning in pathogenesis of the vibration disease is played by specific and non-specific reactions, which reflect adaptation and compensatory processes of the organism. It is considered that the vibration disease is a specific angiotrophoneurosis, when spasm of fine and bigger vessels can be observed. There are also thoughts thatan angiotrophoneurous syndrome at this disease, connected with the affection of laminated bodies (Fater - Paccini).

Recent data proves that in the pathogenesis of vessel changes at the vibration disease takes place in the following:

• disorder of mechanisms of membrane transportation of calcium with the increase of basal concentration of the latter in unstriped muscular cells of blood vessels;

• increase of the speed of both active and passive transportation of potassium;

• replace of properties of pre-membrane spectrin and actinic complex, decrease of urgent skeleton proteins of erythrocytes - spectrin and actin;

• accumulation of primary and secondary products of lipid peroxidation and inactivation of ferments of antioxidant protection.

In the genesis of trophic disorders, which develop at this disease, a significant role is played by changes in microcirculation, rheological properties of blood, disorders in obtaining and utilization of oxygen. There are also disorders in hypothalamic-pituitary-adrenal system, changes in correlation of

vasoactive substances of rennin-angiotensin-aldosteronogenic system and hormones of pituitary-thyroidcompex, content of nucleotide, increase of prostaglandins in blood, affection of vitamin and microelement balance, as well as change of immune indicators. Besides that, neurohumoral and nervous-reflector disorders have phase character. They depend on the degree of the expression of vibration pathology. Thus, in initial stages of the disease, there is an increase of the functional activity of sympathyco-adrenal system due to activation of mechanisms of adaptation and overexcitation of peripheral vegetative formations. Further, in case of progressing of pathology, this state changes by the normalization of excretion of catecholamines, and then inhibition of sympathico-adrenal mechanisms correspondingly to the decrease of adaptation possibilities of the organism.

Clinics. Due to the fact that manifestation of vibration disease is many-sided and polymorphous, and until now the classification of E. Andreyeva-Galanian and V. Artamonova is still pressing; it considers its various forms, conditioned by the action of local and general vibration. Prolong study of this pathology enabled to state various variants of its progressing with mainly the manifestation of neuro-vascular disorders or pathology of a locomotor system. E. Drogichyna and N. Metlina isolated seven syndromes of the disease: angiodistonic, angiospastic, syndrome of vegetative polyneuritis, neuritis, vegetomyofascitis, vestibular, and diencephal with neurocircular disorders. Isolation of these syndromes is conditioned by the fact that the impact of additional production factors together with vibration (cooling down, microtraumatism, and physical tension) enabled to isolate some syndrome of the disease in the clinical pattern. At the same time, the progress of the disease haschanged lately, what conditioned the necessity of the consideration of this classification, taking into account the action of local vibration of various intensiveness.

The clinic of the vibration disease is complicated, and it is distinguished by its variability and is not always specific. The disease develops gradually, thus for a long time patients might not go to the doctor due to seeming improvement of their state when working. Only an active medical examination can help to make a diagnosis. Depending on the character of the work, physical parameters of the acting vibration, total amount of time of its impact, place and area of collision with the source of vibration, symptoms manifest differently and peculiarly.

The clinical symptoms of any form of the vibration disease consist of the neurovascular disorder, disorders in neuro-muscular system, locomotor system, and metabolism. The main place in the clinics is possessed by angiodistonic syndrome with phenomena of angiospasms of peripheral vessels. At the same time depending on the form if their manifestation is a localization of vascular and nervous - muscular disorders can be conditioned by the character of the action of vibration, in the range of which there are high and low frequencies. Thus, complaints of patients in the result of the action of low frequency vibration and significant "recoil" of a vibrating instrument can be numerous in the first stage already. These patients develop a vibration disease with mostly the affection of muscles, bone changes, angiodistonic manifestation, vascular hypotonia, atony of lesser vessels, pattern of polyneuritis, mostly vegetative, and a rather marked pain syndrome.

The vibration disease in the result of vibration, in the range of which high frequencies prevail, differs by the peculiarity of vascular disorders, and more marked cardiovascular syndrome.

Vibration disease in the result of local vibration

In compliance with the existing classification of this form of the vibration disease, it has three forms of severity:

I - initial manifestations: 1) peripheral angiodistonic syndrome of upper extremities, including rare angiospasms of fingers; 2) syndrome of sensor (vegetative-sensor) polyneuropathy of upper extremities.

II - moderately marked manifestation: 1) peripheral angiodistonicsyndrome of upper extremities often with angiospasmic fingers; 2) syndrome of vegetative-sensor of polyneuropathy of upper extremities: a) often with angiospasms of fingers; b) with stable vegetative-trophic disorders of hands; c) with distrophic

disorders of a locomotor system for upper extremities and their belt (myofibrosis, periathrosis and arthrosis); d) with cervicobrachial plexopathy; and e) with cerebral angiodistonic syndrome.

III - marked manifestations: 1) syndrome of sensomotor polyneuropathy of upper extremities; 2) syndrome of encephalopolyneuropathy; and 3) syndrome of polyneuropathy with generalized acroangiospasms.

Initial manifestations of the disease progress in the form of peripheral angiodistonic syndrome or the syndrome of sensor polyneuropathy hands. The disease starts slowly with passing paresthesia. Patients complain to have numbness, pricking, feeling of butterflies in the stomach, ache in distal parts of arms, increased sensitivity to cold in fingertips. Pain and paresthesia can be noted only in calm state, after work and at night. Besides, manifestations take place when cooling, and in case of the change of atmosphere pressure, when doing heavy physical work. After prolong breaks in work, unpleasant sensations in hands disappear.

Patients with vibration disease in this stage the following symptoms can be observed: cyanosis, hypothermia of hands, hyperhidrosis, sometimes, palm dryness, and mottled skin. All these show the disorder of peripheral blood circulation in palms; these symptoms are not stable. Peripheral angiodistonic syndrome can be accompanied by finger whitening at general or local cooling. Acroangiospasms develop either on both palms simultaneously, or at first on the hand, which suffers from the vibration impact. At first they usually appear during cold periods of the year at general cooling down: suddenly there might be sudden whitening of tips of one or several fingers (except the first one) and lasts for several minutes, and then are replaced with cyanosis (stage of angiohypotonia), which can be accompanied by paresthesia. If the process develops, angiospasms can be spread onto other phalanxes, and then appear on the other hand. In the initial stage of the disease, Reino syndrome takes place rarely (about 1 or 2 times a month).

The perception of vibration and pain sensitivity decreases. At initial manifestations of vibration disease, there can be hyperstesia of fingers, which is replaced with hypoesthesia. Zone of the decrease of sensitivity gradually spreads onto palms and forearms. Trophic disorders in this stage of the disease are limited by the worn out pattern of fingers and X-disease of palms.

Peripheral angiodistonic syndrome is actually a clinical manifestation of vegetative polyneuropathy of upper extremities. With intensified pain and paresthesia in distal sectors of arms, expanded zones of hypoesthesia beyond palms diagnose the syndrome of vegetative and sensor polyneuropathy of upper extremities.

Moderate marked manifestations of the disease can be characterized by more marked intensiveness of pain and paresthesia in arms and intensification of the frequency of the development of acroangiospasms. Pain and paresthesia in distal sectors of upper extremities become more stable during a day. After work and at night they are intensified; sleep is affected; patients toss in bed and rub hands at night. During vacations or treatment, this unpleasant sensitivity in hands usually decreases, however it does not pass completely. The growth of expression of peripheral vegetative and vascular, sensor and trophic disorders takes place. In the morning, patients have swelling fingers, slow moving of fingers, which together with pain and paresthesia usually disappear or decrease soon after the work is commenced. Cold angiospasm of fingers develops after washing hands with cold water, or sometimes spontaneously. Fits of angiospasms can involve all the fingers; its duration grows up to 30to 40 minutes. It is over with angiohypotonia with pain reaction and prolong ones (1 to 2 hours) with cyanosis of fingers. Here, the spasm of capillaries changes with their atony. Peripheral vegetative and vascular disorders at this stage do not develop independently, but are a part of the syndrome of vegetative and sensor polyneuropathy of upper extremities. Further increase of the threshold of vibration sensitivity, decrease of superficial sensitivity not only in distal, but also in proximal sectors of extremities can be observed. Sometimes, the zone of hypoesthesia expands to the chest and head.

In case of presence of stable swelling of fingers and palms, slow moving and moderately marked bending contracture of fingers, deformation of interphalanxes, limbs, X-disease of palms, changes of form

and feeding of nails, a diagnosis can be made as to the syndrome of stable vegetative and trophic disorders. Here, nail plates can be in the form of watch crystalline lens, often they are thickened or thinned and dim.

Distrophic disorders in tissue of a locomotor upper extremities and their belt, and also can be manifested in the form of myalgia and myositis of extensors of palms and fingers, suprascapular muscle, periarthrosis and deforming of arthoris of elbow, shoulder and interphalanxes limbs.

From the side of the central nervous system, there is a neurosis-like syndrome, with mild vegetative dysfunction. In the measure of the development of the disease, patients have intensified irritability, fatigability, headache, sleep disorder, cardialgia, dizziness, lability of pulse and arterial blood pressure.

Thus, if to sum up, it is characteristic for Stage II of the vibration disease to have deepening of clinical manifestations of the syndrome of a vegetative and sensor polyneuropathy of upper limbs with more diffused decrease of superficial sensitivity, more marked peripheral angiodistonic syndrome, stable vegetative and trophic disorder of bones, frequent angiospams of fingers, as well as the development of myofibrodistrophic syndrome.

Nowadays, marked manifestations can be met very seldom. Such patients have a syndrome of sensomotor polyneuropathy of upper extremities with the intensification of pain and paresthesia, manifestation of weakness in hands, as well as decrease of force in them. Here, there is hypotrophy of bone muscles, forearms, and the decrease of tendinous reflexes.

Some patients might have generalization of angiospasms and manifestation of the latter on toes as well. In such cases, a syndrome of vegetative and sensor polyneuropathy with generalized acroangiospasms can be diagnosed. It is also necessary to make a stress, which is in the basis of clinical pattern of these forms of the vibration disease, which can be encountered today, initial manifestations of the disease and symptoms are put in the basis of the local vibration, which can be considered as a transitional one from initial to moderately marked manifestations. Besides that, there are symptoms of visceral pathology, e. g. change of the level of arterial pressure with overbalancing of hypertensia, functional disorder of the activity of alimentary gland, dyskenesia of a stomach, intestines, gall tracts, disorder of vitamin balance, as well as carbohydrate, protein, and mineral exchange. It is considered that these changes have reflector character and are conditioned by the disorder of endocrine and vegetative regulation.

Vibration Disease, Caused by the Impact of General Vibration Classification. There are three stages of the severity of a pathological process:

I - initial manifestations: 1) angiodistonic syndrome (cerebral or peripheral); 2) vegetative-vestibular syndrome; and 3) syndrome of sensor (vegetative-sensor) polyneuropathy of lower extremities.

II - moderately marked manifestations: 1) cerebral-peripheral angiodistonic syndrome; 2) syndrome of sensor (vegetative-sensor) polyneuropathy together a) with polyradicular disorder (syndrome of polyradiculaoneuropathy); b) with secondary lumbosacral plexus syndrome (due to osteochondrosis of the lumbar sector of the spinal cord); and c) with functional disorders of the nervous system (syndrome of neurasthenia).

III - marked manifestations: 1) syndrome of sensormotor polyneuropathy; and 2) the syndrome of dyscylindar encephalopathy together with peripheral neuropathy (syndrome of encephalopolyneuropathy).

Clinics. Patients in the initial stage of the disease complain to have headache, irritability, fatigability, and sleep disorder. Here, we can observe the lability of pulse and arterial pressure, though predominant signs are hypertensia, hyperhidrosis, and affection of dermographic reaction. All these prove the development of cerebral angiodistonic syndrome. Together with this syndrome, there are manifestations of peripheral angiodistonic syndrome, for which it is characteristic to have unstable and moderately expressed paresthesia and pain in lower limbs, sometimes cramps of sural muscles. During the examination, it is possible to observe cyanosis or mottled skin, hypothermia of feet, hyperhidrosis of soles, reduction of perception of vibration and pain sensitivity on toes. If there is intensification of pain and paresthesia in feet,

expression of peripheral vegetative and vascular disorders, decrease of superficial sensitivity on the polyneurotic type, mostly in distal sections of lower extremities prove the presence of the syndrome of vegetative and sensor polyneuropathy of lower extremities. In case of the development of the disease of Stage II, there are moderately marked symptoms of the syndrome of vegetative and sensor polyneuropathy of lower extremities. In some cases, similar symptoms may take place in hands as well. Together with vegetative and sensor polyneuropathy, secondary lumbosacral syndrome develops in the result of osteochondrosis of the lumbar part of the spinal cord.

Marked manifestation (Stage III) of the disease can be observed very rarely. For this stage of the disease, it is characteristic to have sensomotor polyneuropathy (pain and weakness in lower limbs when walking, reduction of force and hypotrophy of specific muscles of shins and feet, pain in nerve trunks when palpating).

Vibration Disease, Caused by Combined Affection of General and Local Vibration Classification. There are stages of three stages of the disease:

I - initial;

II - functional;

III - marked manifestation.

Clinics. The disease starts gradually. Patients complain to have headache, dizziness, increased irritability, general sickness, and fast fatigability, ache in lower extremities, as well as their numbness and paresthesia. There can be fits of whitening of toes. The disease at this stage (initial) is manifested through neurasthenic syndrome with phenomena of vegetative dysfunction. The disease has compensated character, and after the termination of contact with vibration, the ability of the sick to work is renovated.

Further, headache becomes constant, agitation increases, and desire to whine appears. Periodically, there are "vegetative crises": nausea, short-term lapse of memory, dizziness, and increased general hyperhidrosis. Skin coverlets become pale, eyes become brighter, pupils widen, muscles are tensed, and the body temperature increases. Fits end up with profuse hidrosis with further development of prostration. This stage (functional) can be characterized by the development of general angiodistonic phenomena with "vegetative crises", lability of cardio-vascular system, and vegetative-sensor polyneuropathy of extremities. There is a marked asthenic syndrome.

At later stages of the disease, there are following sings: worsening of memory, sleep disorder, formation of diencephal syndrome (weight loss, anorexia, acute asthenia, microorganic symptoms of the affection of stem portion of brain and hypothalamic sector), there are changes in the cardiovascular system (bradycardia, and arterial hypotensia). This stage (marked manifestation) has a number of peculiarities: headaches become more permanent. Crises with short-term loss of consciousness become more frequent, vegetative and sensitive polyneuropathy of extremities develops, as well as encephalopathy and diencephal syndrome. Differential diagnostics. Differential diagnostics of the vibration disease is conducted to determine such diseases as Reino syndrome, syringomyelia, vegetative polyneuritis and myositis.

Reino disease mostly develops at women; its development does not depend on the occupation. Clinically, it can be characterized by marked angiodistonic syndrome of peripheral vessels, presence of disorders of vibration, pain, temperature and tactile sensitivity, as well as changes in the internal organs, and locomotor system.

Syringomyelia starts gradually. It is characteristic for it to have segmental disorder of sensitivity, atrophy, pareses, and paralyses; also finger whitening can sometimes take place. Together with disorders of pain temperature sensitivity according to the segmental type, there is tactile and muscle sensitivity.

Vegetative polyneuritis can be characterized by disorders of sensitivity on polyneuritic type; disorders on polyneurotic type; it is not characteristic for it to have affection of vibration sensitivity and fits

of angiospasms. In addition, affection of trophism, temperature and tactile sensitivity decrease can take place.

Myalgia and myositis have definite connection with the occupation. They can be characterized by pain when palpating muscles, absence of peripheral vessels, specific disorders of sensitivity at plexitis (disorder of pain sensitivity at the absence of disorders of vibration, temperature and tactile sensitivity takes place).

Treatment. Etiological principles of treatment of patients with vibration disease involve the principles of elimination (temporary for the period of treatment or full-time in case of absence of therapeutic effect) from the work under conditions of the impact of vibration and other unfavorable factors of production environment.

Among generally strengthening and treatment/preventive measures, it is necessary to consider aero-, gelio and hydrotherapy with the utilization of natural factors of the external environment: air baths, dosed sun irradiation, and swimming in open pools in summer.

As to special treatment - preventive measures, it is necessary to recommend vitamin therapy (ascorbic acid, B1, PP and B12), irradiation with UV rays, preparations, which increase non-specific reactivity of organism.

To conduct pathogenic therapy, it is recommended to use anticholinergic drugs, ganglionic blockers and acupuncture. Among anticholinergic drugs, good results are shown by spasmolytin and benactyzine; and ganglionic blockers -pachykarpin, benxohexamethonium and hexamethon. More positive result is given by combining ganglioblockers and anticholinergic drugs with preparations, which are capable to expand vessels (nicotine acid and papaverine). Nowadays, new data as to positive effect of the recommended calcium channel blocking agents, first of all, the group of nifedipine (corinfar and cordafen, 10 mg three times a day for three weeks), and particularly, corinfar and unithiol (5 ml of 5 %solution, 10 injections), show fastimprovement of the condition of patients, which is accompanied by weakening of pain, acroparaesthesia in hands, disappearing of angiospasticattacks, earlier appearing of the feeling of warmth in hands, and sleep improvement. Together with this, structural and functional state of membranes of erythrocytes, indications of peripheral and central hemodynamics, and rheological properties of blood get normal.

Among physical methods of treatment, it is recommended to have iontophoresis of 5 %solution of Novocain onto hands; diathermy on cervical ganglions; UV irradiation of cervical ganglions; and utilization of two or four chamber galvanic baths.

It is recommended to conduct a spinal blockade 0. 25 % solution of diphacyl together with Novocain, UV irradiation on the level of segments C3-C4 and Д5 and Д6, starting with 2 or 3 biodoses, increasing it to 3 or 4; course is 7 to 8 sessions. It is also recommended to undergo hydrogen sulfide, nitric-thermal, rhodon baths and mud cure (37 - 38 °C) as well as rational meals.

Verification of the ability to work. At the disease of Stage I for patients, they are temporarily (for one month) employed at work beyond the action of vibration (with the provision of a leave on occupational inability to work in case of the decrease of earnings). When qualification of a worker at change of employment is decreased much, then a percentage of the loss of the ability to work for the period of re-qualification is set by the decision of a treatment -expert commission (one year).

Similarly, issues can be solved in case of vibration disease of I to II stages. Only to achieve a stable effect of such diseases, patients are transferred to work beyond the action of vibration for the period of two months.

Treatment of patients with Stage II of the disease should be done in hospital with further transfer to work, which is not connected with the impact of vibration or cooling down to fix results of treatment for 1 or 2 months. In case of acute decrease of qualification at the change of work, thy can be assigned to

undergo expert examination to determine the degree of the loss of the ability to work for the period of re-qualification (1 - 2 years).

As a rule, patients with vibration disease of Stage III have limited ability to work. They obtain a percentage of the loss due to occupational inability to work or an invalidism group (III) due to the occupational disease.

Patients with vibration disease of the 1st degree do not lose their ability to work due to general vibration. They undergo treatment, and then to stabilize its results, they are transferred for a month or two to work that is not connected with the impact of vibration, intensive noise, and receive a leave as to their inability to work.

At well-marked pathological changes of the 2nd degree, it is necessary to undergo rational employment with the definition of the degree of the loss of the ability to work (for one year). For patients with vibration disease of Stage III, it is characteristic to have the decrease of occupational and general ability to work. They can obtain 2nd or 3rd group of invalidism in the result of the occupational disease.

Preventive measures. Technical measures - decrease of vibration in the source of their formation, utilization of carious shock-absorption means, provision of normal microclimatic conditions in premises, where work on vibration instruments and equipment is carried out; hygienic formation of the level of vibration; as well as organization of the regime of labor at minimal contact with those, who work with vibrating instruments.

It is recommended to conduct hydraulic procedures - bathes for hands with the temperature of water 37 °C together with self-massaging; UV irradiation sub erythematous dosages of mostly cervical areas; gymnastics and regular medical check-ups.

OCCUPATIONAL PATHOLOGY, CONDITIONED BY THE IMPACT OF NOISE

Noise is a chaotic combination of sounds, i. e. mechanical vibrations in the zone of frequency from 20 Hz to 16 kHz, which are perceived by a hearing analyzer.

Under conditions of the production of the noise impact, there are engine testers, riveters, cutters, copper-smiths, weaver and spinners. Noise is an unfavorable factor of production environment impacts mechanization experts in agriculture, as well as repair shop workers.

A hearing apparatus of a human being can perceive a sound with the frequency from 16 to 20 000 vibrations a second. As to the spectral composition, it is possible to distinguish high frequency noise with the predomination of levels on the frequencies higher than 800 Hz, and low frequency - with most levels on frequencies lower than 300 Hz and mean frequency, which cover an intermediate diapason of frequency (300 to 800 Hz). The character and degree of expression of the action of noise onto the hearing organ is determined by its intensiveness, key, periodicity, as well as joining of noise with other occupational factors, in particular with vibration.

Pathogenesis. Until 1960's, it was considered that noise causes affection on only a hearing analyzer. It was stated that in the basis of occupational partial deafness, there are destructive changes of both hair cells of cochlea, and also in spiral ganglion and in hairs of a cochlear nerves. And only for the last twenty years, a possibility was proved of a non-specific action of noise onto an organism was proved, which manifested itself in the affection of the functional state onto the organism, which manifested itself in assumption in disorder of the functional state of the nervous and cardio-vascular systems.

At first, hair cells of the lower cochlea of helix, which perceive sounds of high tones. If further impact takes place, support cells of Deiterse and internal hair cells are involved. A number of nervous fibers of external hair cellsdecreases. Cells of spiral ganglion look pressed together and their number is decreased. At the occupational partial deafness, sound-perceiving apparatus is affected (a spiral organ and a plexus of fibers of a helix of vestibulocochlear nerve around hair cells), i. e. occupational worsening of hearing belongs to perceptive partial hearing.

It is necessary to remember that in the development of pathology of the vestibulocochlear nerve, a significant role is played by the affection of nervous and cardio-vascular system, conditioned by disorders of blood circulation and tissue trophism. Under the impact of intensive and prolong noise, agitation of the hearing center is transferred to the mesh substance and reticular formation.

High frequency noise is transferred subjectively worse and it has more dangerous impact onto the organism. Impulse noise is considered harmful than constant one.

Clinics. With the development of occupational partial deafness, there are four stages of loss of hearing. Occupational partial deafness develops according to the type of cochlear neuritis and can be characterized by gradual development. At first, there is noise in ears, which becomes more intensive and stable gradually. At research with a tuning fork or with audiometry already at early stages, there is a decrease of perception of high frequencies (4000 - 6000 Hz) and the reduction of bone conduction. Gradually, worsening of hearing, there are also other tones, and the level of perception of whispering reduces as well. Bad hearing of whisper attracts attention as well, though hearing of speech is still good. The latter is affected only in case of presence of a very large work period under conditions of the impact of noises (20 years and more). Otoscopic pattern goes without changes.

Thus, for patients with occupational partial deafness of the 1st degree, whisper can be perceived at the distance of 5 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 50 dB.

For the 2nd degree (cochlear neuritis with a mild degree of hearing worsening) - whisper can be perceived at the distance of 4 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 60 dB.

For the 3rd degree (cochlear neuritis with a mean degree of hearing worsening) - whisper can be perceived at the distance of 2 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 65 dB.

For the 4th degree (cochlear neuritis with a severe degree of hearing worsening) - whisper can be perceived at the distance of 1 m and at audiometric research, at the audio frequency of 4 kHz, it is possible to register the decrease of hearing up to 70 dB and more.

Thus, at the initial stage of the disease, perception of whisper (diapason of frequencies within limits of up to 2. 5 kHz) does not almost change, and workers do not notice the decrease of their hearing. However, special checking with the help of audiometer at frequencies of 4 to 6 kHz demonstrates the decrease of hearing well. At this, both air and bone perception is affected on the same level; the process has symmetrical character, affecting both right and left ear. Along with progressing of the disease under the impact of noise, perception in the area of audio frequencies of 2, 1 and 0. 5 kHz decreases; it usually develops gradually and slowly, and it increases with the period of work on the position, what curves of hearing threshold are shown (Fig. 6 - 9).

Complaints to have complaints on general sickness, increased irritability, bad sleep, headache, dizziness, and sound of noise or ringing in ears take place. Some patients complain to have pain in hear, often of complaining character with the irradiation under the left shoulder-blade. In future, there are complaints to have the decrease of hearing of both ears. At objective examination, there are vegetative disorders on the general neurotic background, which takes place in the form of instability in the position of Romberg, trembling of stretched our arms, red stable dermographism, as well changing of a reflector sphere.

At noise pathology, there are disorders in metabolic process. Change in protein exchange is manifested through the increase of general protein and globuline.

Clinical observations show the change of heart activity among those, who are subject to noise impact. Thus, on electric cardiograms, there is lability of pulse and slowing down of intra-ventricle and intra-atrial heart conductivity.

The majority of researchers consider that under the impact of a prolong and systematic noise, arterial blood pressure is increased, thus noise can be a factor of risk in the development of hypertonic disease.

For occupational partial deafness, it is characteristic to have the following: gradual development of the process; presence of correlation between disorders of the function of the central nervous system and a number of other organs and systems with changes of hearing sensitivity; presence of similar cases of the disease among workers of the professional group; absence of indications in the anamnesis as to the beginning of this disease during the period, which was prior the contact with occupational hazardous factors; and loss of hearing takes place in the result of affection of air and bone conductivity; whereas deafness is stable and permanent even when the source of noise is eliminated.

Treatment. Considering the peculiarity of the clinical manifestation of syndromes, conditioned by the action of noise, it is necessary to approach selection of therapeutic means in a differentiated way. First, it concerns generally strengthening therapy, organization of regular meals, sleep regime, obligatory staying in the fresh air for 1 to 2 hours, every day. Among medical grudges, it is recommended to use bromide, elenium, trioxazine, benactyzine, as well as glutamine acid and rutin, depending on the expression of an accompanying syndrome and cochlear neuritis.

With the purpose of generally strengthening action, it is recommended to use ascorbic acid in dosage of 300 to 500 mg, as well as a complex of vitamins of group B.

At the presence of angiodistonic syndrome, it is recommended to use spasmolitic means and ganglionic blockers (bromides, aminazine, meprotane together with bensohexamethonium and penthamine).

In case of development of neurocirculatory dystonia of hypertonic type at the patient, it is recommended to prescribe bromide, valerian, and diazepam in combination with spasmolytics. As neurotropic drugs, it is recommended to prescribe reserpine and rhaunatin, which initiates processes of the connection of noradrenaline and dopamine in depositing granules of ends of post-angliar simpatico fibers, and do not let to deposit noradrenaline, which circulates in blood, also has tranquilizing and neuroleptic action.

Among physical methods of treatment, galvanization on the method of Scherbak, darsonvalism, and UVF onto carotid sinus zone are used. To conduct balneotherapy, it is recommended to take salt-coniferous, carbonic acid gas, and hydrogen sulfide baths.

To treat neuritis of hearing nerves, it is necessary to use diabasol, nicotine acid, sulfate atropine and tropacine.

Among physiotherapeutical methods, good results are shown by diathermy onto the zone of mammiform process, and mud application onto the ear area. Verification of the ability to work. At the neuritis with mild decrease of hearing, the work ability of a patient is saved as a rule. It is necessary to conduct dynamic doctor observation, and conduct of outpatient treatment with utilization of sanatorium-preventoriums.

At cochlear neuritis with a mean degree of the decrease of hearing of a qualified worker with long occupational period of work, it is also possible for them to keep their work under thorough observation and conduct of outpatient treatment. In a number of cases, if general disorders prevail, in compliance with occupational medical leave, it is expedient to transfer a patient to another occupation, which is not connected with the impact of sound. However, young people with a short period of work, as well as unqualified workers and people who are subject to impulse noise, especially in case of fast progressing of the process, it is necessary to recommend rational employment beyond noise.

At cochlear neuritis with a sever degree of hearing worsening, it is recommended to have rational employment, which is not connected with the impact of noise. In all these cases, if rational employment is impossible without the demotion of qualification, patients are to be sent to the expert commission to determine a group of invalidism of an occupational character.

Preventive measures. Introduction of various earplugs and cotton wool; anti-noise bushes of the type of "Earplug"; as well as utilization of earphones and helmets. Preliminary and periodical medical examinations are recommended.

OCCUPATIONAL DISEASES BOUND WITH ATMOSPHERIC PRESSURE CHANGES

Altitude sickness

The altitude sickness is a disease that results from a considerable and fast decrease of partial pressure of oxygen (p02) in ambient gas medium.

In 1918 Schneider had offered to aggregate pathologic conditions that arise in time of flight and climb up an altitude in a unified nosological unit that have received a title of altitude sickness. It originates in pilots, and also in people, who work on high-level regions.

Etiology and pathogeny. Main cause of altitude sickness originating is an acute oxygen deficiency. Oxygen deficiency development is predetermined by reduction of barometric pressure with obligatory fall pO2 in air or decrease of oxygen contain in air or in man-made gas medium of hermetically sealed rooms. The first situation can arise during high-altitude flights on flight vehicles with cabins of open type or after lesion of air-tightness of cabins of a closed type; the second one is owing to failure of systems that regenerate air in hermetically sealed cabins and rooms.

Adaptive reactions directed on improvements of oxygen transportation to cells, and pathological reactions conditioned by oxygen deficit, are closelyinterweaved in pathogenesis of altitude sickness. It can be considered in such sequence. Deficit of oxygen in environment results in decrease of partial pressure in alveolar air and arterial blood. Lowering of partial pressure in arterial blood causes in turn and irritation of chemoceptors of reflexogenic vascular zones (sinocarotid and aortal). Amplification of impulses of chemoceptors is the beginning of many reflex adaptive reactions that determine increase of a minute volume of blood, stimulation of hypophysis-adrenal system and above-vesiculate formations of brain, including cerebral cortex. Development of hyperventilation results in acapnia originating. It plays a certain part in pathogenesis of altitude sickness, and therefore it can be a cause of lesions of blood circulation and breathing regulations. Changes in activity of central nervous system that are showed in view of sensory and motor lesions arise due to considerable deficit of oxygen in arterial blood on the background of adaptation reactions. At those structures that are the most sensitive to oxygen deficiency in blood suffer first of all: photoceptors of eyes' cellular tissue, cortex of cerebral hemispheres, cerebellum.

Clinic. Two basic forms of altitude sickness are marked out: collapse and unconscious.

The collapse form of altitude sickness originates practically at 3 % of able-bodied people in 5 - 30 minutes after altitude-chamber ascent on an altitude of 5000 m. It originates in 25 % cases for persons with functional failure of cardiovascular system regulation, and at 10-15 % cases for a practically able-bodied people after ascent on the altitude of 6000 - 7000 m. At that a general weakness, feeling of fever in all body or only in a head occurs, vision changes, air deficiency is felt, giddiness and loss of consciousness come up. Exterior of ill person, his/her behavior changes: paleness of face dermal cover comes up, sweating increases, features of face are sharpened, and it takes a suffering view. Motion activity is increased at first, and then delayed; a pose becomes constrained, a look is long, fixing on separate subjects. Attitude to surroundings becomes indifferent. Consciousness remains saved for continuous time, but all instructions of doctor are performed slowly and as if reluctantly. If a sufferer will not be supplied with a normal oxygen feed, his/her) condition can sharply worsen - a loss of consciousness will be set in. Frequency of cardiac contractions becomes less often, arterial pressure is reduced, and that testifies a development of collapse form of altitude sickness.

Unconscious form often arises without any precursors. Ill person does not feel unpleasant sensations, loses feeling of adequate attitude to external situation and own condition, the loss of consciousness comes suddenly. In some cases attacks of clonic cramps precede to consciousness loss.

Loss of consciousness at this form of altitude sickness refers to group of homeostatic unconsciousnesses, as its cause is hypoxemia - considerable decrease of blood saturation with oxygen. At that a cerebral blood circulation in some time after loss of consciousness remains on a rather high level, therefore a renewal of normal supply of organism with oxygen results in recovery of consciousness and disappearance of all symptoms of altitude sickness within 10-20 seconds.

Treatment. It is necessary to transfer a sufferer with altitude sickness to breathing by oxygen or mixture of oxygen with 3-5 % contents of carbon dioxide; it is the only reliable method of this disease treatment. The oxygen therapy for fast and full recovery of health in light cases is sufficient.

Except for the oxygen therapy it is necessary to use a medicinal therapy at high-gravity forms of altitude sickness, if a sufferer is unconscious during continuous time or if a loss of consciousness arises multiply times and is accompanied by attacks of cramps, vomiting. Citramonum, caffeine, camphor, cordiaminum, strophanthin, lobeline or cytitonum are prescribed with this purpose. Drugs with dehydrational properties (mannitol, dextrane, and glucose) are recommended for a preventive measures and elimination of posthypoxic brain hypostasis.

Heat exchange. It is necessary to take into consideration nature of changes and feature of work at solution of problems, connected with capacity for work. Experiencing of light forms of altitude sickness that has not resulted a health condition in nonperishable negative changes later on is not contraindication to work on a profession.

Expressed and nonperishable changes result in disablement. Medical social commission of experts determine a degree of decrease of capacity for work, solve a problem concerning necessity of person transfer to disablement, give recommendations concerning a training for a new profession with allowance for degree of manifestations of occurred changes.

Preventive measures. The most effective way of preventive measures of altitude sickness is usage of oxygen equipment that supports normal entry of oxygen in organism. It is necessary to perform trainings in conditions of an altitude chamber: regular ascents up on the altitude, which increases step by step (from 3 000 up to 5 000 m), and also under high-level conditions for increase of resistance to altitude sickness. Preliminary and periodic medical examinations of aircrews are of great importance. Contraindications to ascent to altitude are any lesions in a central nervous system, hypophysis and endocrine disorders, cardiovascular diseases, organs of touch and alimentary glands.

Decompression sickness

Some technology processes are carried out under conditions of heightened atmospheric pressure. For example, a drifting of horizontal and vertical underground excavations through watered seams or fulfillment of work under water that is possible only under condition of water forcing out from an air working chamber using compressed air. Pneumatic work isperformed in special units named torsion boxes and they are most widespread at building bridges and dams, foundations under various facilities, tunnels, undergrounds, in coal and mining industry and so on. Influence of heightened atmospheric pressure is testing with help of divers and scuba diving.

The caisson disease is a pathological condition that develops owing to formation of gas bubbles in blood and tissues in case of decrease of external respiration (in a man on leaving caisson and emergence).

Etiology and pathogenesis. Caisson sickness is a consequence of transition of gases of blood and tissues from dissolved condition in free one -similar to gas - in case of decrease of environment atmospheric pressure. At that, gas bubbles are formed, they destroy normal blood circulation, stimulate nervous endings, deform and damage tissues of organism. Main part of general pressure of gases in lungs and consequently

blood and tissues falls on a portion of nitrogen, a physiologically inert gas that does not take participation in gaseous exchanges. High partial pressure of nitrogen in lungs, its physiology and non-reactivity predetermine its basic role in formation of gas bubbles in case of decompression development. Term of dynamic balance recovery for various tissues of organism is unequal at change of nitrogen partial pressure in external and alveolar air. Blood, lymph and tissues, which perfuse well, are saturated faster and destroy it.

Dynamic balance of gas becomes broken, tissues and liquid of organism become oversaturated with gases first of all by nitrogen at lowering of pressure of environment (when a worker leaves a caisson box, at ascent from depth onto surface). Process of excess nitrogen removal from tissues before arrangement of a new gas balance at sluggish decompression usually flows without formation of gas bubbles. Oversaturation of tissues with gases reaches a critical level in case of fast decompression. Conditions for bubbles formation in tissues and liquids are formed. There are two basic types of bubbles. The first on include bubbles located outside of vessels, formation and return development is determined by process of diffusion - exchange of gases between a bubble and medium that surrounds it. The bubbles located inside tissues, definitely refer to this type. They are capable to enlarge and press on tissues that surround them, causing their deformation, and that invokes sensation of pain in patients. Mechanism of development of sensations of muscular-articular decompression pain at patients has such characteristics.

The second type involves gas bubbles, evolution of which is conditioned not only by processes of diffusion, but also by junction of one bubble with another or, to the contrary, by its splitting into even finer bubbles. They join one another, being formed in venous channel, that gives possibility for acute aeroembolism development in circulatory system.

Pathologic and anatomic picture. The most expressed and specific morphological manifestations in case of fast death from a high-gravitydecompression disease are availability of numerous bubbles in venous system, a right half of heart overflowed and spread by gas bubbles, phenomenon of edema and emphysema of lungs, numerous zones of hemorrhages in various organs and tissues.

Clinic. Three degrees of gravity of a decompression sickness are marked out: mild, mean and high-gravity.

Itch of skin, eruption, non-acute pain in muscles, bones, joints and along nerve trunk is characteristic for the mild degree. More often, continuous pain arises in one or several joints of extremities, in particular in knees, shoulders, and also in radiocarpal, elbow joints and ankles. The pain has no concrete localization. Most of all it is felt around of joint, being diffused to all directions from it. The pain, as a rule, strengthens at palpation of joint and bending of extremities. Joints and muscles experiencing the greatest physical loadings are involved in the process most often.

Itch of skin is felt on a body or on proximal segments of extremities. It reminds itch of skin after a bite of an insect.

Some portions of skin have mottled pattern due to skin vascular embolism. Gas accumulation in hypodermic gives start to development of hypodermic emphysema.

The disease of the mean degree of gravity is characterized by disease of an internal ear, gastrointestinal tract and organ of sight. First of all syndrome Menyera is formed as a result of gas bubbles origination in labyrinth of internal ear. Acute weakness, gravity and headaches are watched in a clinical picture. These signs integrates with a loss of consciousness, vomiting, buzzing in the ears, and decrease of hearing. Strong paleness of dermal covers, heightened hidrosis appears. Patients complain that all subjects are revolved before eyes; a minor turn of head strengthens agonizing sensations. There is a possibility of consciousness loss. Gastrointestinal lesions are characterized with accumulation of gas in intestines, vessels of mesentery and are accompanied by arise of strong abdominal pain, often defecation. Palpation of abdomen is agonizing; it is strained. Visual acuity is reduced and accompanied by dilatation of pupils and oppression of their reaction on light.

The high-gravity degree of caisson sickness is met today seldom. It is characterized by formation of emboluses in vessels of central nervous system, heart and lungs. Patients complain on general weakness and weakness in legs, sharp coughs, strong pain in thorax, in particular at breathing, asphyxia. Clinical signs of oedema of lungs occur in due course. A significant amount of gas bubbles of different size that produce lesion of cardiovascular activity is accumulated in cavities of right heart and in vessels of lungs in case of originating of multiple aeroembolism. Thus paleness, strong weakness, often and surface breathing is marked in patients: arterial pressure drops. Pulse falls down, dermal covers gain cyanotic tint. Loss of consciousness can be set at expressed phenomena of hypoxia. Myocardial and lung infarction is probable. The cerebral lesions are conditioned by gas emboluses in brain. Weakness, headache arises after a short-lived latent period. Sensitiveness of one half of body disappears in light cases, and phenomena of paralysis arise in more gravity cases: speech is lost; signs of facial nerve paresis and paraparesis of lower extremities appear. It is accompanied by distress of urination and defecation.

The chronic decompression disease is determined. Two forms of it are marked out: primary and secondary. The primary chronic decompression sickness develops slowly. Deforming osteoarthrosis is the main clinical manifestation of this form.

The secondary chronic form represents a complex of pathological changes owing to experienced acute caisson sickness. Its main clinical symptom is aeropathic myelosis and Meniere's syndrome.

At chronic form of the disease, gas embolas are localized in different organs, mainly in bones. At first the clinical picture flows without symptoms both permanent pain symptom and lesion of function of extremities arise only at complication of the process by deforming osteoarthrosis. Head and proximal ending of diaphysis of thigh are violated at the first turn. After that, head and upper part of diaphysis of shoulder, then distal parts of thigh, proximal endings of shinbone, lower endings shoulder and radial bones are struck.

Diagnosis of caisson sickness is established on a basis of the characteristic complaints and clinical symptomatology that come up after decompression. Occurrence of dermal itch, pain sensations, Meniere's syndrome, paralyses, sudden development of collapse - all this with allowance for the preceding decompression is a direct evidence of caisson sickness.

Treatment. A radical method of a caisson sickness treatment is recompression that influences patient by heightened pressure in a recompression chamber. The method is based on the fact that gas bubbles located in patient's organism decrease their volume and solve at recompression. The recompression renders assistance for dissolution of the bubbles, that is it eliminates etiological factor of illness. The medical recompression is carried out under a special program. Symptomatic treatment is used depending on patient's condition: stimulation of cardiovascular system, warming, oxygen, means directed on struggle with pain, with a possible edema of lungs. Application of a hyperbaric oxygenation gives quite good outcomes.

Verification of the ability to work. The sick-leave is given for a period of treatment for 10 days at mild degree of illness. Patient can be temporarily given a work outside of heightened atmospheric pressure and other unfavorable factors operation with issue of a labor sick-leave in case if further treatment in out-patient conditions is necessary. Return the sufferer of caisson sickness of mean gravity to the same work is authorized after a period of temporary incapacity for work.

Availability of complications in the form of firm organic changes on the part of organ of sight and gastrointestinal tract leads to a steady disablement with a rather large list of counter indicative kinds of labor activity.

The labor forecast at a high-gravity degree of a caisson illness is always unfavorable. It is necessary to send patients on commissioning for disablement degree definition and rehabilitational measures elaboration.

Preventive measures. The warning of decompression disease is envisioned, first of all, by observance of rules of work in caisson-box. So, the maximal pressure during their realization should not exceed 3. 9 atm. A working day in a caisson box is divided on two parts with a rest between them not less

than 9-10 hours outside of a caisson box. General number of working hours during a day, including time of locking and unlocking, is ranged from 6 h till 2 h 40 minutes depending on pressure in a caisson box.

Breathing with oxygen, struggle against overcooling of workers is a preventive action against caisson sickness.

An ambulatory or a medicine post with a day-night duty of medical staff is organized for the well-timed and qualified health services on each site of construction where the caisson work is realized. Isolation ward on the occasion of a decompression sickness, medical airlock can be at a medical center.

Persons permitted to decompression and diving jobs should pass preliminary medical examination. Contraindications for admittance for these jobs is hypertensive disease, pulmonary tuberculosis, respiratory tract lesion of not tubercular etiology, peptic ulcer of ventricles and duodenum, illness of nephroses and urinary bladder, sugar Diabetes, and excessive stoutness.

All people working in a caisson box are subject to weekly medical examination with participation of doctor - therapeutist and otolaryngologist.

Theme №5. OCCUPATIONAL DISEASES, CONDITIONED BY THE IMPACT OF ELECTROMAGNETIC IRRADIATION OF RADIO-WAVES AND ULTRASOUND UPON A PERSON'S ORGANISM. ILLNESSES ARISING OWING TO INFLUENCE OF UNFAVORABLE FACTORS OF MANUFACTURE'S MICROCLIMATE.

OCCUPATIONAL DISEASES, CONDITIONED BY THE IMPACT OF ELECTROMAGNETIC IRRADIATION OF RADIO-WAVES

Electromagnetic irradiation as a totality of electric and magnetic fields is one of the main factors of the impact of factors of the impact of the environment. Electric and magnetic field do not exist separately from each other, and their mutual transformation condition appearing of a unified electromagnetic field, which expands in the surrounding environment in the form of electromagnetic waves with the speed of 3 • 108 m • c-1.

Electromagnetic irradiation can be characterized by the oscillation frequency and the length of waves. The frequency is measured in Hertz (Hz) (1 Hz is equal to one oscillation a second), and the unit to measure waives is a meter (m). Derivatives of these units are correspondingly kilohertz (1 kHz= 103 Hz), a megahertz (1 MHz = 106 Hz), and also a kilometer (km), a centimeter (cm), etc.

Based on biological action of various range of action of the irradiation, in hygienic and medical practice, it is recommended to use a simplified classification of main ranges of radiation.

The range of electromagnetic irradiation is very wide: from infrasound with the frequency of up to 3 Hz and the length of a wave - over 108 m and to ionizing (X-ray and y-irradiation) with the frequency over3 • 108 - 3 • 1011 Hz,the intensiveness of electromagnetic irradiation can be characterized by surface density of energy current, i. e. the amount of energy, which goes through the plane, the area of which is equal to one unit, located in perpendicular to the direction, where electromagnetic waves spread. A unit of measuring of the density of the energy current is a Watt per a square meter (W/m2). Boundary permitted level (BPL) of the density of the flow of energy of radiation in the range of low frequencies at irradiating throughout the whole workday - 0. 1/m2, at irradiation for not more than 2 years - 1 W/m2, and not more than 15 to 20 min - 10 W/m2 under condition of obligatory utilization of protective glasses.

Electromagnetic irradiation is utilized for thermal processing of metals, semiconductors and dielectrics. Induction heating up of metals and semiconductors is carried out mainly by magnetic field in the range of HF and UHF.

HF and UHF equipment is used to dry various materials (wood, paper and leather), to warm up plastics, welding of synthetic materials (production of book covers, folders, bags and toys), as well as sterilization of food.

Such rages of electromagnetic radiation as HF, UHF and SHF are most widely used in radio broadcasting and TV broadcasting, and SHF range - for microwave-link equipment, radiolocation, radio navigation and radiodefectoscopy. Active implementation of radiation in physiotherapy needs particular attention. Properties of radiation to warm up tissues in organism are used in such procedures as low frequency magnetic therapy (devices "Polus-1", and "Polus 101"), inductometering (devices DKV-2 and 1KV-4), microwave therapy with centimeter (such devices as "Luch-2", "Luch-3" and "Luch-58") and decimeter waves (devices "Volna-2" and "Ranet").

The main source for artificial electromagnetic radiation is radio and TV stations, radiolocators, and high-tension lines. It is necessary to remember that together with radiation. It is necessary to remember, that together with radiation, servicing personnel is often subject to other hazardous production factors. In the areas of induction heating and at processing of electronic schemes with utilization of soldering, in cabinets of intercity relay stations, it is possible toobserve contamination of air environment with aerosol of lead, tin, carbohydrate, and nitric oxide. In cabinets of intercity relay stations, premises of radio- and TV stations, at the areas of induction heating up, there is high temperature of air; the level of noise can be up to 75 - 99 dB. The work of operators of intercity relay stations, as well as personnel of radio and TV stations is accompanied by significant nervous, emotional and sight tension.

Pathogenesis. The mechanism of the action of radiation onto a human being is very complicated and is not comprehended in full. It has been stated that electromagnetic irradiation causes radio wave and heating impact onto biological objects. Heating action of microwaves is reduced to the fact that at every change of the direction of an electromagnetic field, there are relaxation oscillations and transition of ions in organism tissues, which electromagnetic irradiation is aimed at, and it is accompanied by the discharge of heat and increase of the tissue temperature. Blood, lymph glands, parenchymal organs, muscles and crystalline lens warm up most of all.

Thus, in the basis of a heating action of electromagnetic irradiation there are primary processes of interaction of electromagnetic waves with tissue molecules. Electromagnetic energy in a biological environment transforms engulfing molecules into kinetic energy, what causes tissue heating. The degree of increase of the temperature is determined by field strength, duration and frequency of irradiation, depends on the fact what part of body is subject to irradiation, as well as effectiveness of thermal regulation and some other indications.

The mechanism of the action of radio radiation of a small (less then heating) intensiveness is realized mostly through its reflector action onto the central nervous system. Hypothalamus is the most sensitive to the impact of radio waves, where highest vegetative centers are accumulated. It has been stated that parasympatic portion of vegetative nervous system is the most sensitive to the action of radio radiation, than a sympatic one.

The action of radio radiation onto the brain is realized by a complex group of biophysical, physical and chemical, and quantum-biological effects. On the cell and subcell levels, there are changes of potassium-sodium gradients in cells, polarization of biological membranes with the affection of their permeation, deformation of structures of water systems, change of activity of ferments, disorder of oxide processes, etc.

Conditionally, there are mechanisms of biological action of electromagnetic fields as follows: a) direct action onto tissues and organs, providing the change of functions of the central nervous system, as well as neurohumoral regulation connected with it; b) reflector changes of neuroumoral regulation and c) joining of main mechanisms of the pathogenesis of the action of electromagnetic irradiation with mostly disorder of the exchange of matters, and activity of ferments. Probably, all three mechanisms are rightful, and a role of each of them is determined by physical and biological changes. Clinics. Clinical symptoms of

the impact of radio irradiation depend on its range, intensiveness and duration, and possibly on the regime of irradiation.

Research show that the most biologically active ones are SHF- waves, then UHF range and the less active one is HF range.

A c u t e f o r m of pathological impact of electromagnetic irradiation is divided into three stages: mild, mean and severe. Acute form of the affection takes place during accidents or in case of gross violation of the safety measures, thus in this case, if the intensiveness of irradiation much exceeds the thermal threshold. People, who suffered from the impact of intensive SHF irradiation, the body temperature increases by 1 to 2 °C, general weakness, sickness, pain in extremities and muscles, headache, state of worrying, thirst, dyspnea, face reddening, hyperhidrosis, lability of pulse and arterial pressure and nose bleeding take place, and leucocytosis can be observed. Sometimes, there are simpatico-adrenal crises, and fits of paroxysmal tachycardia.

After acute affection with electromagnetic irradiation, there are functional disorders of the nervous system in the form of vegetative-vascular dystonia or asthenoneurotic syndrome. These stages last for 2 to 3 months and then pass.

C r o n i c f o r m of the affection takes place in the result of prolong action of electromagnetic irradiation, intensiveness of which exceeds BPL, however it is lower that the thermal threshold.

In this case, reaction - reply of the organism is both in adaptation of reconstruction of the nervous and cardio-vascular system, and in the development of cumulative effect, which is manifested through the intensification of pathological reactions in the organism with the period of work. In the first plan, there is disorder of functions of the nervous and cardio-vascular system. People, who worked under conditions of the action of electromagnetic field, adrenocorticotropic activity of the hypophysis, the activity of sex glands is inhibited, enzymopathy appears; neurocircular dystonia develops on hyper- or hypotensive type; immune and biological reaction of the organ is changes; and trophic disorders can be observed.

Under conditions of modern production, the development of symptom complex of chronic SHF affections is possible only at the period of work not less than 10 to 15 years. Women are more sensitive to the action of electromagnetic irradiation, than men; general health state, previous infections and overfatigue of a person are very important as well. In spite of general polymorphic clinical pattern of the disease, the main role of action of electromagnetic fields onto a human being is played by functional disorder of the central nervous system and the affection of cardio-vascular system.

Changes in the central nervous system can be characterized frost of all by the development of asthenoneurotic and vegetative symptom complex.

Asthenic syndrome develops in the initial stage of the disease. Patients complain to have increased fatigability, irritation, and headache without clearlocalization, as well as sleep disorder, unpleasant feelings in the heart area, and hyperhidrosis.

In case of growing of the expression of asthenic reactions and joining of vagotonicsigns, the so-called astehenovegtative syndrome develops, which clinically manifests itself through growing of general weakness, further decrease of the ability to work, memory and concentration. Dizziness and stable arterial blood hypotensia with bradycardia cause the development of consciousness losing.

Further in the clinical symptoms, the change of the direction of vegetative reactions with further transition to the arterial hypotensia and bradycardia into the state, which can be characterized by the lability of the arterial pressure and pulse, and into a neurocircular dystonia of the hypertonic type can take place. Increase of tonus of the sympatic part of a vegetative nervous system can be characterized by the presence of frequent and long headache, dizziness, pain in the heart area, sleep disorder, memory disorder and emotional lability. At examination, such patients have the increase of reflexes, finger and eyelid tremor, general hyperdrosis and bright red dermographism. In some cases, in case of prolong impact of SHF irradiation, when a patient has fit-like intensive headache, dizziness, general weakness, cardialgia,

tachycardia, arterial hypertensia, body temperature increases, and a patient has a fixed idea about the death threat. On this background, there can be present scattered organic symptoms, which prove the development of dysciruclatory encephalopathy.

On the background of the given above neurological syndromes, there are also functional disorders in the cardio-vascular system. This can be manifested through unpleasant feeling in the heart and pain in the heart area. There are also lability of pulse and arterial pressure, shift of the limits of heart to the left, and sometimes, there can be systolic noise to the top of the chest.

At electrocardiograph research, there is often a change of rhythm of heart contractions (sinus arrhythmia) and amplitude of T wave.

People, who suffer the action of electromagnetic irradiation, often have endocrine-metabolic disorders. First of all, it is manifested through the increase of the functional activity of a thyroid gland, disorder of the activity of sex glands, increase of the secretion of adrenalin and noradrenaline with urine, loss of weight, hair loss and nail fragility.

As to the peripheral blood, there is a tendency to lymphocytosis and thrombocytopenia. There is a big number of eosinophiles, monocytes, reticulocytes, the content of general protein at the expense of the level of globulin, histamine, cholesterol, as well as the decrease of albuminic-globulin and potassium-calcium ration, and the level of chlorides.

In addition, digestion system suffers from it. It is manifested through dyspeptic disorder, vaguely marked pain syndrome. There are insignificant changes in extractor and inserter function of pancreas. People, who have worked with the source of the SHF irradiation for a long time, might have the development of dystrophic changes of a crystalline lens - cataract, a characteristic peculiarity of which is the localization of a pathologic process in the area of a rear pole.

Depending on the expression of changes in various organs and systems, there are three stages of the disease.

Stage I - it is characterized by the development of asthenic syndrome, which is often combined with vaguely marked vagotonic symptoms. Functional activity of a thyroid gland increases. These changes have functional character and do not affect much work abilities of patients.

Stage II of the pathological process can be characterized by the development of asthenovegetative syndrome with a stable bradycardia and arterial hypotensia. However, there can be vegetative-vascular dystonia with the lability of pulse and arterial pressure. There are deeper dystrophic changes in myocardium, changes can be found in peripheral blood, and there are moderate endocrine-exchange disorders.

Stage III of the disease can be met very rarely. Hypothalamic syndrome develops; sympathico-adrenal crises become more stable. There is a fit-like headache, shivering, seizing pain in heart, acute weakness, and arterial hypotensia. At much tense of electromagnetic field, encephalopathy with psycho disorders can develop accompanied by memory worsening, depression and mitochondria state.

Diagnostics. To state a diagnosis of the occupational disease in the result of the impact of electromagnetic irradiation, it is necessary to conduct detailed sanitary and hygienic characteristics with defining frequency range of oscillations, intensiveness of irradiation, term of contact, period of work under harmful conditions of the production. It is also necessary to consider non-specifics of manifestations of the disease and on considering it, to eliminate other general diseases, which can condition the development of asthenia or cause neurocirculatory disorders.

Characteristic manifestations of the action of electromagnetic irradiation onto the organism of a human being is asthenic or asthenovegetative syndrome with a vagotnoic direction of disorders, which further are replaced by the syndrome of vegetative and sensor dystonia with prevailing sympathic reactions, appearing of endocrine-exchange disorders, changes of indicators of blood, presence of a cataract. Fast reverse progress, in particular, in initial stages, under the impact of treatment and in the result of normalization of work conditions are the proof of the diagnosis.

Treatment. Asthenic states are indicators to prescribe tranquillizers (trioxazin 0. 3 g, diazepam 5 mg two or three times a day); generally strengthening preparations (injections 5 ml of 5 % solution of ascorbic acid with 20 ml of 40 % solution of glucose intravenously once a day, and the course is 15 injections; 1 ml of 6 % solution of thiamine bromide; 1 ml of 5 % solution ofpyrodixine hydrochloride intramuscularly once a day, course - 15 to 20 injections); tonic preparations (saparal 0. 05 g two or three times a day, ginseng tincture 25 drops three times a day).

At parasympathic - toning direction of vegetative disorders, it is recommended to use anticholinergic drugs (ergotamine hydrotartrate - beloid, belataminal - 1 pill two or three times a day); antihistamine medical preparations (Dimedrol 0. 05 g and suprastin - 0. 25 g).

In case of the presence of hyperkinetic syndrome in cardio-vascular system (tachycardia, palpitation, tendency to increased arterial blood pressure), it is recommended to have small dosages ofP-blockers: propranolol 0. 02 g two or three times a day; preparations, which widen vessels and have hypotensive action (rhaunatine - 2 mg, cynarizin - 25 mg, caviton - 5 mg three times a day, no-shpa or papaverine hydrochloride - 2 ml 2 % solution intramuscularly once a day for 10 to 15 days).

Complex therapy includes curative gymnastics, reflexo- and psycho therapy, diet with small food value, but high content of proteins, rhodon and coniferous baths.

Verification of the ability to work. At the initial stage of the disease, the ability to work is not affected. After active treatment, patients should be transferred to work beyond the contact with electromagnetic irradiation for the term of one month and in case of positive progress of the disease, they can do usual work.

People who underwent diseases in moderately marked stage, it is necessary to conduct treatment under conditions of specialized hospital, after what to fix results of treatment and for dynamic work, they should be transferred to work, which is not connected with the action of electromagnetic irradiation, for the term of 1 to 2 months. Return to work can take place only under condition of complete return of functions.

In case of absence of a real treatment effect, as well as in case of presence of severe stage of affection, patients need rational employment beyond the action of electromagnetic oscillations. Acute decrease of qualification is the basis for assigning patients to the Expert Commission and to determine the stage of loss of the work ability for the period of re-training (one year). In case of a cataract, further work in contact with radio irradiation is prohibited.

Preventive measures. The following methods are recommended and methods of protection for the impact of electromagnetic field: organizational, technological, sanitary and technical, individual and treatment/preventive ones.

Main organizational measures, which allow improving the state of the environment in places of location of sources of electromagnetic irradiation, there is the reduction of the duration of the action and increase of the distance from the source to a worker. Technological measures envision the provision of mechanization and automation of production processes, utilization of manipulators and remote control.

Sanitary and technical measures involve screening of all sources of electromagnetic field. Among measures of individual protection, there are radioprotecting clothes and glasses.

Treatment and preventive measures include the conduct of preliminary and periodical medical examinations, during which it is important to do research of systems of an organism, which suffer the most from the action of electromagnetic irradiation.

OCCUPATIONAL DISEASES CAUSED BY INFLUENCE OF ULTRASOUND UPON A PERSON'S ORGANISM

Ultrasound is mechanical vibrations of elastic medium, which differ from sound with vibrations of higher frequency (above 20 kHz) and cannot be perceived with person's ear. Ultrasound vibrations like sound vibrations spread out in form of alternating thickening and discharging, and are characterized with

length of wave, frequency and speed of spread. The higher is frequency of ultrasound vibrations the more is degree of their absorption by medium and the smaller is depth of their penetration into person's tissue. Absorption of ultrasound is accompanied by heating of medium.

Range of ultrasonic frequency is divided on vibrations of low frequency (from 1. 12404 to 1. 0^ 105 Hz), which spread through air andcontact, and vibrations of high frequency (from 1. 0-105 to 1. 0-109 Hz), which spread only through contact.

Ultrasound is applied in different spheres of economy: metallurgy, machinery construction, apparatus building, radio engineering, chemical and light industry, medicine and so on. Wide application of ultrasound stipulates increase of quantity of workers, which are under its influence. Main occupational groups, which are under influence of ultrasound, are as follows: inspectors, fitters, supervisors of sewage disposal plants, welders, tinsmith, doctors and nurses, who operate therapeutic and diagnostic ultrasonic apparatus, surgery units and sterilization tools.

Pathogenesis. Three kinds of ultrasound are distinguished depending upon intensity of ultrasonic waves and their influence on live tissue:

1. Ultrasound of small intensity (below 1. 5 W/cm2), which is considered as physical catalyser. It causes some changes of physicochemical organism response, exchange process speeding up, tissue light heating, micro massage, and does not cause morphological changes at cells.

2. Ultrasound of medium intensity (1. 5 till 3. 0 W/cm2) causing reaction of oppression in nervous tissue. Speed of function renewal depends from ultrasound influence intensity and duration.

3. Ultrasound of high intensity can cause irreversible oppression even up to total tissue destruction.

Ultrasound biological action consists of violation of functional state of receptor apparatus and peripheral vegetative formations (excitation of temperature, tactile, and pain vibroreceptors); transition of mechanical vibrations energy to heating energy with dilatation of vessels those changes into spasm later. This is accompanied with reinforcement of oxygen absorption by cells and lowering of carbonic acid concentration, accumulation of dross, which influence large toxic action on central and peripheral nervous system, cause injury of cell membranes.

Besides, development of photo- and spectral chemical processes, which evolve in cavity, has meaning.

Clinic. Functional changes from side of nervous, cardiovascular and endocrine systems, auditory and vestibular analysers may be observed in case of systematically influence of ultrasound, which intensity and contact duration exceeds MPC.

Persons who long time serve ultrasonic equipment complain to have headache, blackout, general weakness, quick fatigability, sleep disorders, irritability, memory worsening, heightened sensibility to sounds, and fear against bright light; sometimes complaints can have dyspeptic nature.

Bradycardia and hypotonia are marked for the workers up to the end of a working day; bradysystolia, disorder of intracardiac and intrastomach conductivity are determined with the help of ECG. Monocytos, eosinophilia that later turn into eosinophinia are marked in blood. Quite often loweringof sugar content in blood, hyperproteinemia is determined. All this developments have non-persistent character.

Mentioned symptomatology is expressed strongly in those cases when ultrasound is transmitted not only though air (this concerns low-frequency sound), but through contact also (high-frequency sound).

Vegetative syndrome of asthenia is determined during clinical observance, sometimes diencephalitic abnormalities - loss of mass, subfebrile temperature, paroxysm of visceral attack type, increasing of muscles mechanical excitation, itch - are observed.

Disorders in the form of angiodystonic syndrome, vegetative polyneuritis, vegetative myofascitis of arms, and vegetative vessel dysfunction can develop in operators during a long-period operation with ultrasonic defectoscopes.

As a rule, general cerebral disorders unite with development of vegetative polyneuritis of arms in different extent of expression. This becomes apparent as acrocyanosis, intumescense, hyperhidrosis, lowing of all kinds of sensitivity of short and long gloves type.

Treatment. When asthenic syndrome is present in clinical picture, a prescription of tranquillisers- 0. 2 g meprobamat 1-2 times a day, 0. 3 g trioxazine 2 times a day - is recommended to patients. 0. 05 g ascorbic acid 3 times a day, warm shower, coniferous baths, one hour walks before sleep are recommended parallel with that.

Group B vitamin (B1 1 ml 6% solution intramuscularly, riboflavin 0. 005- 0. 01 g two-three times a day during 15 days, injections of cocarboxylase intramuscularly 0. 05 g a day during 20-25 days) is necessary to use in parallel with tranquilliser receptions (three times a day) for persons with more marked symptomatology: continuous complaints of asthenic kind, development of neurocircular dystonia.

Vegetative polyneuritis with sensible and trophic disorders needs longer treatment. Massage, ozokerite applications, rhodon baths and all this in complex with intravenous injections of 10 ml 0. 5 % Novocain solution, 15-20 injections in total, are recommended for such patients. Treatment in sanatoria and health resorts (Odessa, Khmelnik) gives good results.

Veification of the ability to work. Ability to work in the presence of early, acute marked appearance of asthenisation and vegetative-vessel changes remains on the assumption of regular observation and out-patient treatment. In individual cases a temporary (1 - 2 months) move of patient to work, which is not connected with influence of ultrasound, is recommended.

Reasonable placing in a job that excludes influence of working environment vibroacoustic factors is recommended in case of development of obvious stable neuro-dynamic and neuro-circular dysfunctions, disorders of hearing and vestibular apparatus parallel with adequate treatment in outpatient or hospital conditions. Preventive measures. It is necessary to check a permissible ultrasound level on working places (110 dB or 0. 1 W/cm2 at the most in the frequency range from 0. 1 till 10 MHz), to follow general requirements after methods of check-up and protection from ultrasound influence (designing of automated ultrasonic equipment and units with remote control; providing of ultrasonic equipment with casings and screens; use of individual protective means: noise shields, gloves; 10-15 minutes stoppages over every 1. 5-2 working years) for the purpose of prophylaxis of negative ultrasound influence on persons serving ultrasound units.

Persons older 18 years can be permitted to work with ultrasonic equipment. It is necessary to conduct preventive and periodical medical inspections when taking on; 1 time a year with participation of doctors-specialists: neuropathologist, physician, and otolaryngologist. Hearing organ (audiometry) and speech in a whisper are subject to obligatory study. Persons with an impaired hearing or with a threshold-volute organ dysfunction should be transferred to another work.

ILLNESSES ARISING OWING TO INFLUENCE OF UNFAVORABLE FACTORS OF MANUFACTURE'S MICROCLIMATE

Unification of temperature, humidity, air movement speed and infrared heat radiation in a working area is understood as manufacture's microclimate.

It largely depends on meteorological or climatic conditions of the given region, and at definite kinds of jobs (in open air) can be completely conditioned by them. Besides, the microclimate of manufacturing premises depends on nature of a engineering procedure, conditions of an air exchange and other factors. Overheat

Overheating of organism is a condition that is characterized by disturbance of a heat balance, increasing of quantity of heat in organism. Overheat arises under influence of heat of ambient manufacture medium, and also if there is the factors that preclude from withdrawal of heat in environment. Overheat is watched on works with high temperature of environment (blast furnaces, open-hearth furnaces, rolling

shops, coal mines with deep deposit of coal), and also in climatic regions with a hot climate. Increase of heat exchange that arises in case of fulfillment of hard physical work, in particular, in clothing impermeable for water evaporating, at high humidity and immovability of air assist overheating.

Pathogenesis. Mechanism of effect of various kinds of heat (convection, conduction, radiation) on an organism is not identical. Convection and conduction heat being transmitted from molecule to molecule produces heating of tissues surface and blood circulating in them. This blood transfers heat both in more deep tissues, and in organs.

Radiation (infrared) heat can permeate deeply into tissues of man.

Starting device of reactions of physical and chemical heat regulation in organism is a thermal irritation of dermal and vascular thermal receptors with subsequent corresponding reaction to it from a center of thermal control. Change of physiological functions at considerable overheat arises also owing to effect of heated blood on a central nervous system. Blood vessels of skin are dilated, their filling with blood is augmented, and temperature of skin increases. Heat emission through irradiation, convection, conduction decreases, and heat emission through evaporation of moisture from a body surface increases in case of environment temperature rise.

So, for example, it is considered that a portion of heat emission through convection and irradiation is reduced from 73 up to 8 % from summary heat emission, and a portion of heat emission through evaporation of moisture is augmented from 27 up to 92 % at environment temperature rise from 16 up to 30 °C and at work of a middle gravity degree for a dressed person. Emission of heat from the body surface through convection and irradiation is ended at environment temperature of about 33 °C.

Clinic. Four degrees of overheat are marked out. At the I degree, a general condition of ill is satisfactory, there are complaints on feeling of heat, listlessness and sleepiness, absence of desire to work and move. At that, body temperature can reach 37. 5 °C, arterial pressure (systolic and diastolic), volumes of lung ventilation, consumption of oxygen and excretion of carbonic acid are reduced. A minute volume of heart is increased, pulse becomes more frequent on 15-20 beatings a minute. Hyperemia and humidification of skin can take place. Overheating of the II degree of gravity is characterized by an acute hyperemia of skin, profuse emission of sweat, as well as sensation of fever. Body temperature can reach 38. 5 °C.

Systolic arterial pressure is increased by 5-15 mm of mercury column, diastolic one is reduced by 10-20 mm of mercury column; pulse becomes more frequent by 40-60 beatings a minute; minute and systolic volume of heart, lung ventilation, quantity of absorbed oxygen and discharged carbonic acid are augmented.

Overheat of the III degree can be observed at effect of temperature of above 60 °C. State of health deteriorates, what can be proved by the sensation by strong fever, palpitation, pulsation and pressure in temples, gravity and pain in head. Motion agitation is marked; skin has sharp hyperemia, and sweat flows off by drops; pulsation of carotid and temporal arteries are watched. Body temperature reaches 40 ° C, systolic arterial pressure is reduced by 30-40 mm of mercury column, pulse becomes twice and more times faster in comparison with the initial value, reaching 160 beatings a minute on average.

Overheat of the IV degree is characterized by lesion of activity of cardiovascular and central nervous systems that takes place at the so-called thermal shock.

A face has hyperemia from the beginning, and becomes pale-cyanotic later on. Skin is dry, hot or covered with sticky sweat; temperature increases up to 41-42 ° C; diuresis is decreased.

Breathing is shallow, fast, and irregular. Pulse is rapid, fine, and thread-like, cardiac sounds are toneless. Consciousness changes from mild degrees up to coma; cramps have tonic and clonic nature; psychomotor excitation; often dreaming and hallucinations develop. Clotting of blood with increasing of residual nitrogen and urea, and decreasing of chlorides takes place in it. Mortality at this stage reaches 20-30 %.

Peculiar form of overheat is a sunstroke. It is mostly caused by direct effect of solar radiation (for example, on builders, workers of open-cast mines and agriculture) and is a consequence of influence of infrared radiation on a central nervous system. Gravity of sunstroke flow is conditioned by degree of lesion of brain membrane and other structures of central nervous system. Infrared radiation permeating to considerable depth (2-3 cm) in tissues can cause diseases of meningitis and encephalitis. It is necessary to mark that such pathology in conditions of works does not develop even at high intensity of infrared radiation.

Clinic. General weakness, sensation of indisposition, headache, loss of consciousness, twinkling before eyes, tightening in a thorax, buzzing in the ears, sometimes nose bleedings, giddiness, vomiting, irregular feces come up at this form of overheat, skin gains red color, in particular on face, and excretion of sweat strengthens. Obviously expressed lesions on the part of acentral nervous system arise in high-gravity cases: aberration, sharp excitation, convulsions, hallucinations and visions. Body temperature of body, as a rule, does not increase.

Lesion of water-electrolytic exchange that is showed in the form of cramps is possible owing to overheat of organism and loss of a large quantity of liquid through sweat. In these cases, evident titanic cramps that are spread to various groups of muscles, in particular sural, thighs, shoulders, forearms, and their sharp morbidity during motion alongside with phenomena referenced for thermal shock, are watched. Patients are adynamic, with sharpened features of face. Skin obtains cyanotic tint, and gets dry and cold on touch. Distresses on the part of cardiovascular system take place: pulse becomes more frequent (up to 110-120 beatings a minute), arterial pressure is low, cardiac sounds are muffled. Diuresis makes 50-100 ml a day; content of chlorides in urine is sharply decreased. Signs of blood clotting can be found: quantities of erythrocytes and hemoglobin are augmented; and viscosity of blood is increased.

Attacks in the form of epilepsy, disorder of psychics can be in high-gravity cases.

Treatment. The treatment of overheat is directed first of all on struggle against of hyperthermia and for normalization of activity of cardiovascular system. For this purpose wet wrappings, chill on head at cramps are recommended. It is recommended to take much liquid, injection of glucose solution of Ringer parenterally together with vitamins. Oxygenotherapy and injections of adrenalin subcutaneously give a positive result at collapse; injections of cardiac glycosides, hyposensitizing means intravenously at acute cardiovascular failure is recommended. Milk and vegetative diet is recommended.

Help at thermal shock foresees transfer of a victim in a cool place, local cooling of head, sponging off a body with cold water. Oxygen inhalation gives quite good result. Intravenous injection of polyglucine, rheopolyglucine, 5 % glucose solution, isotonic solution of chloride natrium (up to 1. 0-1. 5 l) is recommended in case of arterial pressure decrease and in the presence of other signs of vascular failure. Seduxen/diazepam, Dimedrol, and Droperidol are prescribed at observation of arterial pressure level.

Dehydrational therapy with osmotic diuretics (Manitol 1 g/kg of body weight), lumbar puncture is carried out at phenomena of brain hypostasis. Medical hypothermia with a continuous forced ventilation of lungs and correction of disturbance of acid - alkaline and water-electrolytic balances is recommended in high-gravity cases. Reanimation measures are carried out in case of lesion of breathing and blood circulation termination.

Verification of the ability to work. Patients can be temporarily disabled for a term of treatment with light signs of overheat. They can return to their previous work after full convalescence. When residual functional distresses on the part of a nervous or cardiovascular system take place after experienced overheat, it is necessary to provide the patient temporarily a job, which is not connected with the influence of high temperatures and intensive heat radiation, until his/her full convalescence. Patient is given a sick-leave if necessary.

If the marked stable functional lesions after overheat remain, the further work in conditions of influence of high temperatures and intensive heat radiation is counter-indicative.

Patients in case of full invalidity or absence of possibility of a rational provision of employment are sent on commission for definition of a group of physical inability.

Preventive measures. Improvement of working conditions on works with intensive emission of heat is implements with application of technological, sanitary, hygienic and medical measures.

Application of perfect engineering, localization of heat emission, shielding and thermal insulation of sources of radiant and convection heat, rational ventilation, application of air douche, rational organization of operational and rest mode, observance of drinking and alimentary rations, which are directed on recovery of water-salt balance, render assistance in prevention of overheat unfavorable influence.

Receding and periodic medical examinations that are carried out once per 2 years including therapeutist, neuropathologist, ophthalmologist also have large value. It is necessary to take into consideration medical contraindications for work in conditions of high temperatures and intensive heat radiation presence at realization of the preceding medical examinations.

Cooling

The cooling is a distress of organism functions as a result of low temperature effect. Low temperatures of air are met in rooms where it is connected with an engineering process (refrigeration chambers, textile manufacturing etc.), or with cold season of year for want of heating.

Etiology and pathogeny. Cold as a parasitic factor of manufacture medium influences unfavorably on organism and production activity of a man. Speed and depth of cooling depend parallel with force and duration of chill factor effect as well on condition of organism and conditions, in which one is. Resistance of organism to cooling is decreased at physical tiredness of a person; overcooling comes faster in conditions of high air humidity or high wind.

Cooling is a consequence of disturbance of a heat balance and develops in those cases when heat output in organism exceeds heat production. Phases of compensation and decompensation of heat regulation are distinguished during development of cooling. Thermoregulator reactions of an organism in a phase of compensation of heat regulation have reflex, adjustment nature and are directed on prevention of decrease of body temperature by the way on the one hand decreasing of heat emission and on the other hand by increasing of heat production. Decreasing of heat emission is reached by discontinuance of hidrosis, spasm of skin and muscles vessels, decreasing of blood circulation in them. Production of heat strengthens at the expense of increase of metabolism. Balance in phase of decompensation of heat regulation between production of heat and a heat emission breaks, heat emission prevails, and consequently a condition of pathological hypothermia develops. Thus, hypoxia takes place as outcome of disorder of breathing and blood circulation. This condition is strengthened by break of microcirculation owing to a decrease of vessels tone, slowing down of blood circulation and deterioration of rheological characteristics of blood.

Pathologoanatomic picture. Morphological changes at cooling are showed with plethora of internals, main and spinal cords and their membranes, cerebral haemorrhages, and haemorrhages into mucosa of ventriculus, small-sized nuclear necrosises in internals, hypostasis of lungs.

Clinic. Four stages in clinical process of cooling are distinguished: compensator, adynamic, soporose and comatose.

In compensator stage suffereds are excited, they complain on chill. Lips are cyanotic, dermal covers are pale; cold, shivering of muscles, shortness of breath, tachycardia, increase of arterial pressure, more frequent urination are marked.

In adynamic stage consciousness is not lost, however sufferer is stopped, sometimes euphoric, the delicacy complains on a headache, loss of consciousness. Availability of adynamia, decrease of muscles

tonus, depressing of tendon jerks is marked. Pulse is normal or delayed up to 40 b/minute, arterial pressure does not change essentially, and cardiac sounds are damped down.

In soporous stage general dormancy, flaccidity, sleepiness down to condition of sopor, sometimes euphoria, disorder of memory, dysarthtia is marked. Pupils are dilated or are periodically narrowed down and extended, breathing is slowed down (till 8-10 in 1 minute), surface, sometimes with moans; pulse is infrequent (from 50 up to 30 b/min), arterial pressure is lowered. Incontience of urine and feces is possible.

In comatose stage consciousness in sufferers is missed, arbitrary motions with head and extremity, convulsive tonic contractions of muscles, predominantly flexors, can appear.

Motional excitation arises sometimes. Pupils are narrowed down; their reaction on light is absent. Breathing is surface, infrequent (up to 3-4 in 1 minute). Pulse is determined only on large arteries, infrequent (up to 20b/minute). Arterial pressure is lowered, cardiac sounds are muted and auscultated hardly.

Complications at cooling are caused by fall-off of an immunological reactivityof organism, because of what a threat of development of inflammatory diseases appears in the course of time: pneumonias, pleuritis, acute respiratory diseases. Besides, functions of other organs and systems can be upset, and that quite often results in asthenisation of psychosises, trophic lesionses.

Treatment. Nature of medical care at overcool depends on depth of hypothermia and degree of disorder of vital functions of organism. The Influence of cold factor on normalization of condition of patients in compensator stage of cooling needs only to be stopped. In adynamic stage temperature of a body also can be normalized independently under according conditions (stay in warm room, warm drink, application of a heater, warm bath).

Persons suffered in soporous and comatose stages of cooling are needed in the first aid. Main efforts should be concentrated on support of breathing and blood circulation, prevention of further cooling and warming of the organism. Sufferers are immediately transferred in a warm room, wet clothing should be removed off them, and they should be put around with heaters. Possibility of falling a tongue back is eliminated, slime from a mouth is pumped out, and oxygen inhalation is performed. If breathing and cardiac activity break has taken place, all complex of reanimation measures is executed. The main method of taking a sufferer from a critical condition is an intensive warming. During the warming a heated 5 % solution of glucoses is introduced dropwise, polyglucin. 40-50 ml 40 % glucose solutions with insulin and ascorbic acid are introduced with purpose of power stuff replenishment. Euphyllin, neuroleptics or ganglioblocators in doses that do not cause a decrease of arterial pressure is possible to use for improvement of cerebral circulation. For correction of acidosis 100-200 ml 5 % solution of hydrocarbonate natrium are poured in; antihistamine drugs with purpose of decreasing of a vascular wall permeability are applied also. Diuretic drugs (furosemid - 40-60 mg, manit - 1 g/kg) for preventive measures and treatment of post hypoxia edema of head brain and stimulation of uropoiesis during warming and after it are applied .

Local lesions in view of obliterating endarteritis and frosted parts of body are possible except for general cooling due to influence of cold also.

The obliterating endarteritis arises at continuous cooling of extremities and is met mainly at fishermen, workers on mining of peat, maintenance staff of refrigerating plant. Clinical signs of this disease are an albication of skin of fingers, decrease of dermal sensitivity, paraesthesies, difficult motion of extremities and weakening of pulsation on peripheral vessels. Four signs of the obliterating endarteritis are marked. On the I stage (spastic) functional angiospastic disorders are found out. Pain, sensation of a cooling-down and numbness in extremities, weakening of pulse on peripheric vessels appears at patients. Such phenomena can be watched rather continuous time, they periodically disappear and come up again. At the second stage (ischemic) the angiospastic syndrome becomes more constant and expressed owing to development of steady structural changes in walls of vessels. A threat of formation of thromboses in walls of vessels exists. Stage III (necrotic) is characterized by formation of ulcers on extremities, what is

conditioned by disorder of nutrition of tissues. On the IV stage (gangrenous) a development of dry or wet gangrene is observed.

Freezing as well as the obliterating endarteritis is characterized by local lesion of tissues of organism owing to low temperature effect.

Clinical signs of freezing occur after some period of time after termination of cold influencing. Three degrees of freezing are distinguished. Sensation of itch, pricking occurs at freezing of the I degree, as well as albication of affected sites of body, which transfer later in reddening, is observed. The indicated changes pass over quickly without any consequences. Decrease of skin sensitivity that is accompanied by its albication and edema of affected sites is characteristic for the II degree. Bladders charged with serous-bloody liquid occur. Gangrene develops at freezing of the III degree.

Freezing develops faster in case of joint action of factor of chill with heightened humidity, hypodynamia, local disorders in a blood supply of tissues, due to tight footwear or clothing.

Treatment is carried out depending on form and degree of disease gravity. Thermal physiotherapeutic procedures, means, designed for elimination of pain, vasodilatation are recommended ill with the I and II stages an obliterating endarteritis. A surgical operation is sometimes necessary at the III and IV stages.

General warming of body, rubbing with camphoric or boric alcohol (3 % solution) of lesion sites of skin with the subsequent imposing of dry bandages for patients with freezing of the I degree.

Surgical operation is recommended for patients with freezing of the III degree. It is necessary to remember that appearance of a secondary infection contamination is possible at these stages, therefore prescription of antibiotics should be included in a complex of medical measures.

Verification of the ability to work. Persons, who have experienced light forms of overcooling, can return to previous work after a conducted treatment and full convalescence. If residual phenomena with functional lesions that do not give capabilities to perform previous work take place after high-gravity forms of freezing experienced before, such patients with allowance for degrees of functional disorders should be rationally provided a job or sent on commission for definition of group of physical inability.

Preventive measures. The main measures on preventive measures of cooling of an organism are following:

• prevention of manufacturing rooms cooling down using thermal insulation of a floor, walls, windows etc. ;

• installation of local heating on fixed working places;

• installation of equipment for heating of rooms in large shops without fixed working places (refrigeration cabinets, preparation plants) and during work on open air in cold climatic zones; temperature of air in rooms can be supported in limits 21-24 ° C;

• application of means of individual protection from patients;

• implementation of measures on hardening an organism, his adaptation to staying in conditions of a cold climate;

• organization of preliminary and periodic medical examinations of persons, who work in conditions of low temperatures effect; the periodic medical examinations should be carried out once during two years including therapeutist, neuropathologist and surgeon.

It is necessary to take into consideration a list of contraindications for work under conditions of chill factor effect at preliminary examinations of persons, who are assigned to work.

Theme №6. OCCUPATIONAL DISEASES CONDITIONED BY FUNCTIONAL OVERSTRAIN

The pathology, which is referred to diseases of locomotor apparatus, takes a rather considerable place among occupational diseases. First of all these conditions are connected with functional overstrains and microtraumatism.

They are met among blacksmiths, loaders, operatives of rolling patients, mine workers, typists, line-operators, pianists, insulation workers, etc. Thus, the basic unfavorable factors in genesis of these diseases are both a high-gravity manual labor and a local loading and considerable static strain owing to motions of extremities that are multiply repeated, as well as continuous stay in an enforced pose.

Majority of this pathology syndromes are localized in the upper extremities, what is explained with large local loadings, significant amount of small stereotyped motions of hands and fingers of arms (in some trades they reach several tens thousand motions). So, gravers of crystal only during one shift do up to 40 thousand press efforts in various positions of hands with considerable pressure of hands and forearms muscles. Milkmaid fulfpatients about 2500 compressions with fingers when milking only one cow during a day. Typist does above 60 thousand strokes on keys of type machine by fingers per one shift, and operators of accounting machines fulfill from 100 up to 200-250 thousand strokes with an effort at each stroke from 0. 5 up to 1N.

These unfavorable factors are strengthened by enforced working positions, continuous static strain of back and extremities muscles, non-observance of operation and rest mode, irrational working methods (incorrect bearing of hands at musicians), microtraumatism of skin and its receptors, temperature drops, constant humidifying of hands by water, oils, emulsions and so on.

Estimation of gravity and strain of labor process is carried out in accordance with «Labor hygienic classifications. The I and II classes (they eliminate threat of development of occupational diseases) are considered as optimum and permissible. Thus, such characteristics are standardized:

• power of dynamic work (W) at loading of muscles of the upper extremities should not exceed 45W for men and 30. 5 W for women, and at load of muscles of the lower extremities and trunk 90 and 63W accordingly;

• weight of load that is lifted should not exceed 30 kg for men and 10 kg for women;

• quantity of hand and finger motions per one shift should not exceed 40000 small stereotype motions;static muscular loading should not exceed 430000 Nf per shift when the load is holding with one hand, and 970000 Nf when by two hands;

• working pose should be free; staying in an inclined position under angle less than 30°should not exceed 25 % of shift duration, and if it is necessary a quantity of trunk inclinations under angle more than 30° should not exceed 100 times.

There is a possibility of development of some occupational pathology in case of exceeding of these standards (the III class of labor conditions).

Classification. The following occupational diseases conditioned by a functional overstress are marked.

1. Occupational dyskinesia (coordinator neurosis).

2. Diseases of peripheral nervous system: neuropathy, cervical shoulder plexopathy, cervical and lumbar-sacral radiculopathy, and lumbalgia.

3. Diseases of apparatus of resistance and motion: a) illnesses of muscles (myalgia, myositis, myofascitis); б) illnesses of fibrous-tissue and synovial formations (shoulder scapula periarthritis, epicondylitis, tendovaginitis, stenosing ligamentitis, contraction of palmar aponeurosis, and

bursitis); в)osteochondropathy (deforming arthrosis, spondylarthrosis, and bone aseptic necrosis); г) combined syndromes.

Pathogenesis. Mechanism of development of diseases conditioned by a functional overstrain is very complex and is not yet clarified to the end. If a pathogenesis of occupational dyskinesia is evaluated, it is considered that a continuous work, which demands doing of considerable amount of small motions of high coordination with a very fast speed, can cause an overstrain of nervous processes, lesion of cortex neurodynamics with formation of an isolated "ill nucleus" in certain department of motion analyzer. It results in lesion of complex motion stereotype, which is situated in foundation of strictly differentiated motions.

Besides, such factors, as emotional overload, dissatisfaction with job, often conflict situations at work and in a household provoke the development of occupational dyskinesia.

High-gravity physical work, fulfillment of a great number of uniform motions is accompanied with traumatisation of peripheral receptors of a nervous pipe. Thus, lesions of blood circulation and metabolism, which lead to degenerative changes in peripheral nerves, arise. Such factors as strains, compressions, infringement of nervous pipes in marrow or in muscle-binding channels, in particular in a working time that is carried out with permanently bent extremities also assist to development of these processes. Lesion of blood circulation and metabolism develops also owing to accumulation in muscles of underoxidized products of exchange, changes of inflammatory nature in interstitial tissue and as a result of secondary degenerative lesions of muscle fibers, which develops later. The indicated processes as well as aseptic inflammation, which accompany them, can be developed in tendons,serous bags, periosteum, and cartilages of joints. It results in development of such pathologic conditions, as periarthritis, tendovaginitis, bursitis, arthrosis and so on.

OCCUPATIONAL DYSKINESIA

Occupational dyskinesia takes a special place among all diseases conditioned by functional overstrain, as it is a functional disease of the central nervous system. For the first time, this disease was described as "writing spasm" in the middle of XIX century. The cause of its originating was connected with an intensive written work, as the disease was fixed at office employees and copyists. Later it turned out, that these phenomena of discoordination arise not only during writing, but also at many other kinds of job, which demand fulfillment of hard coordinated motions with fast speed. Today occupational dyskinesia is described for gravers, knitters, graphic artists, draftsmen, fitters, milkers and milkmaids, musicians playing keyboard and string instruments.

Clinic. The disease arises gradually and develops slowly. Such clinical forms of this pathology are marked out: convulsive, paretic, atactic, shivering, neuralgic, and mixed. Increase of tone of hand small-sized muscles during occupational work is characteristic for the convulsive form.

So, persons, whose occupation involves writing, experience discomfort when writing of separate characters or digits, their handwriting becomes worse, and then cramps appear in one or several fingers during writing.

Sudden weakness in hands comes up at patients with paretic form of dyskinesia in time of working; fingers are relaxed involuntarily owing to what a worker can not hold in hands a pen, a pencil or other tool.

A version of the paretic form is so-called atactic form of dyskinesia, which develops predominantly during work on a keyboard (typists, lino operators and pianists). Characteristic symptom of such disease is that an ill cannot touch the key, which is necessary to him/her.

At the shivering form of dyskinesia a tremor comes up predominantly in that hand, which is more loaded during fulfillment of this or that activity.

For the neuralgic form an occurrence of intensive pain is characteristic in muscles of hand, forearm or all arm during work-time. Talk about the mixed form of occupational dyskinesia takes place in the event of presence of combination of several forms at ill.

Diagnosis. It is necessary to remember that the most typical symptoms of the occupational dyskinesia are selectivity with lesion only one function, which makes a basement of this or that occupational work: writing, typing on a printing machine, playing on a musical instrument and so on. Other motion functions of working hand can also suffer in the course of time. Differential diagnostics. The differential diagnosis of occupational dyskinesia is carried out with hysterical paresis of hand, organic diseases of central nervous system, in particular with those that are accompanied by extrapyramidal and cerebellar lesions. Paresis of hand at hysteria develops usually quickly and is spread to all its functions.

The organic diseases of central nervous system are accompanied with intermediate symptoms and never are limited only with lesions of occupational functions of arm.

DISEASES OF PERIPHERAL NERVOUS SYSTEM

The given group of diseases includes lesions of separate nerves of spinal cord (ulnar, middle, small shank), polyneuropathy of the upper extremities, cervical - shoulder (plexalgy and plexopathy, lumbalgy, neck also lumbosacral radiculopathy). A special place is taken by the so-called tunnel syndromes (compressive neuropathy), which are developed owing to squeezing of meddle, ulnar, radial and other nerves with tissues, which surround them, and which have changed pathologically.

Neuropathy of an ulnar nerve is met most often owing to traumatism of the nerve during fulfillment of work, which demands supporting of elbow with table (carver, graver, engraver, and glassblower). Patients complain of pain and paresthesia in a region of the fourth - fifth fingers. Strength and endurance of hand muscles are reduced owing to what they cannot hold in hands tool or detail that should be processed continuous time. Hypesthesia is met on the fourth - fifth fingers and on ulnar edge of hand; hypotrophy of the fifth finger's muscles arises. Patients cannot bring the fifth finger together with the fourth one.

Lesion of the middle nerve is characterized by that at patients early occurs paresthesia and pain in the second - third or in all fingers, in hand and forearm. The pain is considerably strengthened at night, during cooling, at change of atmospheric pressure. Hands are cyanotic, wet. Hypostasis of fingers, hypoesthesia on palms, in particular on the first - third fingers and on back side of distal and middle phalanxes of the second - fourth fingers can be observed. In the course of time atrophy of muscles of the first finger's rise develops, strength of hand's compression is reduced.

The neuropathy of nerve of small shank develops at workers, which stand on knees or squat down during continuous time (parquet floor layers, roofers, workers of agriculture).

Squeezing of nerve in popliteal fossa or near a small shank bonehead takes place because of features of occupational work. Patients complain on a hindered rotation of foot outside; hypesthesia of external surface of shank and back of shank appears. The so-called "cock step" arises at patients.

Vegetative-sensory polyneuropathy. Complains appear on an incipient stage of the disease: ache in bones and forearms, paresthesia in them (feeling of numbness, "crawling of insects"). These feelings are strengthened during a change of atmospheric pressure, at cooling. In the course of time pain and paresthesias strengthen, become constants, disturb ill after work, at night, upsetting sleep. Peripheral vegetative - vascular lesions come up: hyperhidrosis, puffiness of distal phalanxes, change of skin coloration and hands temperature. Cyanotic color and invariable or heightened temperature, and paleness or marmoreal skin and hypothermia of hands at predominance of spastic phenomena are marked in case of paretic phenomena predominance in capillary tubes and vessels of small caliber. Sensory lesions at vegetative polyneuropathy decrease or hyperesthesia takes place.

Surface sensitivity in process of development of a vegetative - sensory polyneuropathy is reduced according to polyneuritic type. Expressiveness of peripheral vegetative - vascular lesions increases: edemas

and hard movable properties of fingers, which are over or considerably decrease after beginning of work come up in the mornings. Trophic lesions are often observed: hyperkeratosis of palms, change of form and frailness of nails, deformation of interphalanx joints. Complain on weakness, fatigability, decrease of force in the upper extremities are joined in case of motive fibres retraction in pathological process. Decrease of hands pressure force, hypotrophy of separate muscle groups of the upper extremities, decrease of tendon jerks are determined objectively.

Lumbosacral radiculopathy is met at persons, whose work is connected which a considerable static-dynamic loading on lumbosacral area of vertebral column owing to frequent fulfillments, in particular to forced tilts and trunk turns (operatives of rolling-mill, and lumberers), systematic lifting and transportation of hard loads (loaders, dockers, and carvers), continuous maintenance of forced pose (drifters, fettlers, miners), influence of general jerk similar vibrations (drivers of large trucks, tractor and combine operators). Overfreezing and temperature differences strengthen an effect of these factors.

Steady pain in transversal - sacral region, in particular during trunk tilts and turns is marked at patients. Hypotrophy of gastrocnemius muscle, decreasing of reflex of Achilles' tendon develops. Weakness and hypotrophy of muscles of the front regions of shank develops gradually, in particular in a long extensor of large finger. Knee reflex is reduced or disappears quite often.

DISEASES OF MUSCLES

These diseases are ones of the most widespread forms of occupational pathology conditioned with the functional overstrain. They include two clinical syndromes: myalgia and myositis. These conditions are met at workers of the most different trades, whose jobs are connected with considered static-dynamic loading. First of all, builders, workers of brickworks and bread-baking plants, milkmaids, typist, pianists are among them. Muscles of forearms are subjected to the affliction most often, muscle of shoulder less often.

Myalgia starts from ache, sensation of discomfort, gravity, and strain in the according muscles. Speed of work is reduced, mechanical excitement of muscles and their bioelectric activity is increased. Strength and, in particular, their endurance to static loads is reduced.

Myalgia is a functional stage of the disease. Condition normalization is already marked in some days of rest. At the same time a relapse of the disease with tendency to further progress down to development of degenerative - dystrophic also of secondary inflammatory changes in muscles is observed rather often at resumption of work.

Myositis is characterized with strengthening of pain and paresthesia in hands that cause patients to complain not only in working time, but also in periods of rest, especially at night. The pain strengthens sharply during active motions. Speed of motions, productivity of work is reduced. The affected muscles become hardelastic, flabby, and atrophic. Very painful dense knots from 3-4 mm up to 2-3 cm are palpated in thickness of a muscle tissue.

DISEASES OF FIBROUS AND SYNOVIAL FORMATIONS

Such diseases as periarthritis, ligamentitis, epicondylitis, tendovaginitis, bursitis belong to diseases of peripheral ligaments or muscle fastenings.

Humeroscapular periarthritis as an occupational disease develops during fulfillment of work, which demands repetition of motions in a shoulder joint, in particular on a background of a considerable static-dynamic loading (bricklayers, plasterers, painters). The disease starts with pain in joints of shoulder at motion, raise, and rotation of shoulder. With the course of time, the pain strengthens, becomes constant, crunching appears in joints at motion. Limitation of motion in the joint increases gradually, especially at rotation and taking the shoulder aside. Taking an arm behind own back is considerably limited, and often it is impossible. Development of small mobility in the shoulder joint is possible later on. Pain comes up at

palpation of a large knob of a shoulder bone, deltoid muscle less often, in particular in a place of its fixation to the shoulder bone.

X-ray research at periarthritis of shoulder joint demonstrates sclerotic change of a knob surface of a shoulder bone and availability of shades of calcareous deposits of different sizes, forms and density.

Multiple edge resorptions, which are surrounded by sclerotic process, and destruction of a large knob of a shoulder bone, saline deposits in a synovial bag are observed on a roentgenogram at this pathology most often. Epicondylitis. This disease is diagnosed at localization of an aseptic chronic inflammation and degenerative-dystrophic changes in a region of place of a node fixation of tendon of protractor-arch supporter group of muscles of forearms (lateral epicondylitis) or in a region of a medial node of fixation place of tendon of flexors of hand and fingers (medial epicondylitis). The disease develops predominantly at persons occupied with high-gravity physical activity, and in case of fulfillment by them often protractor-bending and rotating motions in an ulnar joint (workers of agriculture, construction, blacksmiths, and operators of accounting machines).

Literal epicondylitis is characterized by appearance of ache in the region of a lateral node of shoulder bone during forced motions of a forearm and strained extension of a hand. In the course of time, the pain strengthens and arises at any motions in an ulnar joint.

Soreness at palpation of the lateral node is a constant symptom. A small swell occurs sometimes in this region. Unbending of forearm up to 160-170° for majority of patients is limited. Decrease of hand pressure strength is marked at all patients.

Tendovaginitis. This disease is characterized by lesion of a synovial membrane of tendinous vaginas of degenerative-dystrophic nature with concomitant an aseptic (serose-haemorrhagic) inflammation. Tendovaginitis as an occupational disease develops mainly on the upper extremities, in particular as a result of fulfillment of a great number of motions with fingers, hands (insulator workers, bricklayers, winders, typist, and tailors). A tendon of flexors and extensors of hand fingers are usually struck, tendon of a long head of shoulder biceps muscle, heel tendon is struck much less often.

Acute and chronic forms of the disease are marked according to their clinical developments. Aseptic inflammatory process at acute tendovaginitis is accompanied by a fibrin deposit on a tendon surface and can be spread on cellular tissues around the tendon. The process, which flows chronically, is characterized by accumulation of exudation in synovial room. The exudation contains a lot of fibrin. At first from it the so-called rice corpuscles are formed, and then fibrinous adnations between parietal and visceral leaves of incrassate and loosened synovial membrane come in.

Synovial vaginas, which are placed on back surface of hand mainly in a region of a short extensor tendon of the large finger and long muscle, which brings the large finger of a hand aside, are struck at acute tendovaginitis more often. Start of disease usually is connected with a considerable physical loading of according muscles, immediately after which an ache, feeling of fever, pricking, and gravity in the forearm, feeling of tiredness, weakness in hand appears. Dense, quite often painful swelling of 8-12cm length and 3-4 cm width comes up on back-radial surface of the lower third of a forearm in some hours or 1-2 days. Crepitations (crepitating tendovaginitis) is revealed at palpation of swelling during several hours or 2-3 days. Pathological process in case of chronic flow of the disease is developed most often in region of fingers flexors synovial vagina, which is arranged in a wrist canal.

The corresponding tendons are painful during a palpation, incrassated. Swelling can be determined on a palmar surface of a radial carpal joint and distal part of the forearm. Pain and fluctuation takes place during its palpation. Force of this hand soon decreases.

Chronic tendovaginitis also develops gradually or is a consequent of a relapse of the crepitating tendovaginitis. Inflammatory reaction at this version of clinical flow of tendovaginitis is not expressed sharply, though it is necessary to have in view that increasing phenomena of sclerotic process are resulted

in a thickening of a synovial membrane, narrowing synovial room and lesion of tendons motions (so-called a stenosis form of tendovaginitis).

Stenosis tendovaginitis. Under conditions of production, this disease is formed mostly in a region of a radial carpal joint. Inflammatory process is spread to ligamentary structures of one of the hand osteofibrous canals. Or the cicatricial corrugation of bundles, which are formed the canal and its stenosis with infringement of veins, which passes through it, owing to primary development in it degenerative-dystrophic changes, is observed. Stenosis ligamentitis of a ring-type part of a finger fibrosis vagina (stenosis tendovaginitis of fingers flexors) can be an example of such lesion. Continuous traumatism of a palm at the level of calcaneum-phalanx articulations (for fettlers and stamp operators) can result in cicatrical changes of a ring-type part of a fibrous vagina of fingers flexors (usually the first -third) with a subsequent narrowing of a tendon vagina and deformation of tendon bundles. Pain arises in sites of a corresponding calcaneum-phalanx joint in case of compression of tool, processed detail and at motions of a finger. An intermittent contraction (jam of a finger, which is fixed in a position of bending, and it is necessary to apply much effort for its unbending) appears at some period of time. It is accompanied with pain. The jam becomes frequent in the course of time, extraction the finger from a pathological position becomes more and more difficult and demands help of the second hand. Sometimes, it becomes impossible, even if such help is rendered.

Occupational bursitis is a chronic inflammation synovial vaginas, which develops as a result of systematic pressure, overstrain and traumatism at mine workers, roofers, bricklayers, pavers, parquet floor layers, branchers, who are representatives of those trades, where continuous leaning on an elbow or a knee takes place with development of vagina aseptic inflammation and formation of serose-haemorrhagic exudation in it.

An inconvenient position, roughness of ground, imperfection of protective clothing, continuous cooling, abrupt change of ambient temperature, and vibrations assist the development of the disease. Pathogenesis. Dystrophic changes in hollow of affected synovial bags with formation of numerous free bodies, which have chondroid density, arise under influence of prolonged pressure and rubbing near working surfaces of joints. Sites affected with a sclerotic process, as well as liquid contents of the bags also experience a calcification. Loss of elasticity and thickening of the bags, fibrosis in the hypodermic basis, hyperplasia of walls of the synovial bags is an outcome of these processes.

Clinic. Bursitis acute (serous and purulent) and chronic (serous and proliferative) is distinguished. The acute bursitis is diagnosis less often, than chronic.

At acute serous bursitis patients complain of morbidity in a joint, availability of intumescences of soft consistence and spherical shape. The skin in area of the intumescences has signs of hyperemia, local rise of skin temperature in this area is determined at contact, and limitation of mobility in the joint takes place. Content of the bag has serous nature.

Body temperature at the purulent bursitis increases up to 39-40 °C. Punctata of the bursa is purulent with large contents of leucocytes.

Chronic serous bursitis is met seldom. It is characterized by duration of flow, moderately expressed morbidity, availability of restricted intumescences of ovoid shape. Proliferate form is a widespread form of the chronic bursitis. Flow of the chronic bursitis of such form is lingering with periods of remissions and exacerbations; an infection contamination can be affixed often. A fluctuation is determined locally, walls of the bags are incrassate, and an articular slot is reduced.

The Dupuitren's syndrome is a fibrosis-cicatricle degeneration of aponevrosis with formation of tendogenous contraction of the third - fifth fingers in a site of the finger - phalange and proximal interphalangeal joints. It arises at fulfillment of work that is accompanied with traumatism of a palm. Clinically as a rule, painful feelings arise in case of fulfilment of hard physical work. Hardening and cicatrisation of a palmar aponeurosis close to foundation of the third - fifth fingers, hardening and traction of their tendons, formation of these fingers contraction is marked.

OSTEOCHONDROPATHIES

The types of osteochondropathies are arthrosis, spondylosis arthrosis, as well as aseptic neurosis of a bone.

Arthrosis. The occupational deforming arthrosis is developed at considerable dynamic and static loading on a joint. Concomitant vibration, and also a plenty of motions, jerks make assistance to this pathology development. Representatives of such trades as bricklayers, loaders, blacksmiths, plasterers, painters, typists, pavers, parquet floor layers and so on are affected more often. An elbow joint on the upper extremities is struck more often, and a knee-joint on the lower extremities.

Sensation of discomfort, availability of crunching in the joint, pain of uncertain nature disturbs patients. Pain is strengthened gradually, becomes continual. Intumescences, deformation of the joint is observed. Amplitude of motions and mobility in the joint is restricted.

Aseptic necrosis of a bone. It is an occupational disease, which is characterized by development of degenerative-dystrophic changes in supporting bones of a hand and foot, and also in bones of large joints of extremities, of the elbow one first of all. A cause of their originating is an occupational chronic overload and micro traumatism of osteal structures during hard physical work, and influence of intensive local vibrations, in which spectrum a low- and mid-frequency vibration prevails. The pathological process develops more often in a fortnightly bone, which experiences the greatest loading from all bones of a radiocarpal joint.

Aseptic necrosis of the fortnight bone (Keenback's disease). This disease is developed at a considerable systematic static and dynamic load of hand at drift miners, fettlers, workers of building trades, joiners. The process is characterized by development of a subchondral aseptic necrosis, which is accompanied with fractures, deformation and fragmentation of the bone.

The disease is characterized with pain in the radiocarpal joint, which arises during motion. With the course of time the pain is strengthened, becomes permanent. The intumescence of 1-1. 5 cm in diameter, which is excruciating, can be seen in a region of a fortnight bone projection on the backside of the wrist at palpation.

The amplitude of motions in this joint is restricted.

Diagnostics of occupational diseases caused by a functional overstrain has some complexities that are conditioned, first of all, by absence of specific clinical manifestations of these conditions. Therefore, it is necessary to analyze working conditions, nature and intensity of work of an ill person, presence of concomitant unfavorable factors of working environment, duration of the occupational service, feature of originating, flow and clinical manifestations of the occupational pathology, presence or absence of other etiological factors for diagnosing the disease in each concrete case.

The occupational diseases caused by functional overstrains are usually arisen gradually, developed gradually during of rather continuous time. A considerable improvement of the condition due to rather continuous interruption in work, and in incipient stages and in several days of rest is also characteristic for occupational diseases. The absence of other etiological factors, first of all, infection diseases and traumas, has essential value. The majority of these conditions are developed at persons, whose duration of an occupational record makes not less than 10-12 years. Localization of thedisease, which depends on a degree of loading for this or that hand, matters also.

Treatment. Main medical practice at the diseases caused by functional overstrains should include medicaments drugs, physical and reflex methods.

Total and rather continuous termination of occupational work is by an obligatory condition for treatment of dyskinesia. Sedative drugs, tranquilizers, method of autogenic training, psychotherapy, electrosleep and balneotherapy are applied in the treatment. The medical measures are rather effective only on incipient states of disease.

Drugs of antispasmodic effect and those influencing on condition of microcirculation: Xantinoli nicotinas0. 15 g three times a day or "Nicoverinum" 1 pill and Nicospanum three times a day after meals are applied when treating the vegetative - sensory polyneuropathy. Vitamins of the B group (bromine thiamine, hydrochloride pyrodoxine, cobalamin cyanide), cocarboxylase, adenosine triphosphate, and biostimulants (aloe, FIBS) are prescribed with the purpose of metabolic process normalization. Ganglionic blockers: benzohexonium, Pachykarpin, spasmolytin (0. 05-0. 1 g three times a day), intravenous injections of 5 ml of 0. 5 % Novocain solutions (10 injections per a course) are applied at presence of more expressed and steady changes.

Method of reflexotherapy and physiotherapy - electrophoresis 5 % of Novocain solution on hands, and ultrasound provides positive results. Massage of a cervical - collar region, balneotherapy-radon and hydrosulphuric baths are recommended.

Treatment of the occupational radiculopathy is carried out with analgesics, non-steroid antiphlogistic drugs (Analginum 0. 5 g, Butadion 0. 25 g, Indomethacine 0. 25 mg), neuroleptic drugs (Haloperidolum1. 5 mg triply per day), myorelaxants (Mydocalm 0. 05 g, and Sibazone 0. 5 mg during a day), injections of vitamins of the B group, biostimulants. Ultra-violet exposure, diathermy, radon and hydrosulphuric baths, massage, medical gymnastics are widely used.

The treatment of diseases of muscles is the most effective in the incipient stage. Application of ultrasonic sound, short-wave diathermy and Bernar's currents gives a positive result. Medical gymnastics and massage have large value in treatment of myofascites. Intramuscular introducing of 5 ml of 2 % Novocain solution per day during 10 days, vitamin B1 (30 mg per day during 15 days intramuscular) is recommended at presence of muscles sharp morbidity. Warm baths with a subsequent massage of arms and shoulder girdle muscles are prescribed also.

Therapeutic measures at a periarthritis of the shoulder joint consist of mobilization, novocainic anesthesia, physiotherapy, including roentgenotherapy and treatment by ultrasound, punctures and washing of a subacromial bag. Rheopyrinum, Analgin with Amidopyrin, electrophoresiswith Novocain are applied in the period of worsening. Continuous immobilization of the shoulder joint is not recommended, as it can result in development of hard mobility of the joint.

Treatment of epicondylitis of a shoulder in an incipient stage starts from immobilization of hand and forearm with a plaster bandage. Hidrocortizon (from 5 up to 25 mg) every other day during 6 - 8 days inject in a region of the node. The novocainic blockade of the region of the node gives positive results. The course of the treatment consists of 3 to 4 blockades with an interval of 5 days with a simultaneous immobilization of an extremity. Dyadynamic therapy and paraffin applications on the region of the elbow joint (in 3-4 weeks after the immobilization) are effective physiotherapeutic procedures.

Treatment of tendovaginitis foresees a dismissal of the ill from work, a prescription of Novocain blockades in the forearm region (8-10 ml of 0. 5 % solution) and imposing of a plaster bandage. Warm, UHF therapy, paraffinic applications (4-6 performances) are prescribed from the 3 - 4-th day. The plaster bandage is removed on the 7 - 8-th day, the volume of motions with the hand and fingers is increased.

The treatment at the acute bursitis is conservative: rest, antiphlogistic substances (antibiotics, sulfanilamide drugs, blockade with Hidrocortizon or introducing of 25 mg (1 ml) suspension of Hidrocortizon in emptiness of the bag after washing it with 0. 5 % Novocain solution, 2-3 injections in 3-4 days), physioprocedures (UHF, paraffin application, dyadynamic therapy with 10 % potassium iodide solution during 15 minutes, only 10-20 sessions).

Surgical treatment is applied at a relapse or transition of the process in chronic.

Injections of Hidrocortizon, Novocain, Rumalon, Lydas (per 1 ml) under cicatricially changed tissues each day or in a day (10-15 injections), and also phonophoresis of Hidrocortizonum, electrophoresis of Iodine, lithium, and Lydasum are prescribed in the incipient stage of palmar aponeuvrosis contraction; a surgical treatment is recommended later on.

It is necessary to ensure a relative rest for the affected joint at arthrosis of a knee joint. Analgesics, non-steroid antiphlogistic drugs (Analgin, Rheopyrin, and Indomethacin), and vitamins are prescribed. Injections of Rumalon of 2 ml intramuscular a day for 5 or 6 days are prescribed. Electrophoresis with Novocain, Iodine, Chlorine, phonophoresis of Hidrocortizon, UHF therapy, paraffin applications, balneotherapy, and massage are recommended from physiotherapeutic procedures.

Verification of the ability to work. It is possible to conduct the treatment of the ill with presence of a myalgia, vegetative polyneuropathy, serous bursitis ambulantly with a shift of the patient on an easier work for the period of two weeks according to a medical board recommendation. It is necessary to dismiss the patient from work at other forms of the pathology:for 5-6 days at myalgia with a pain syndrome and lumbalgia, for 2-3 weeks if there is polyneuropathy, myositis, tendovaginitis with crepitation, epicondylitis, for 3-4 weeks at a humeroscapular periarthritis, arthrosis, aseptic necrosis of a bone.

Then, patients are transferred to easy work under conditions, which eliminate traumatic effect, for the term from 10 till 14 days at myalgia, tendovaginitis, and from 4 till 6 weeks at other conditions of this pathology for strengthening of the treatment results. After that a sanatorium treatment is recommended.

Development of dystrophic changes, often relapses, development of a course of pathological process, presence of the steady pain syndrome, steady lesion of the function are a basis for a training for a new profession of ill and its rational job placement that is not connected with effect of the functional overstrain. Patients are sent to medical board for a solution of the problem of the degree of disablement (1-2 years) in case of proficiency lowering due to change of the working place. And the group of physical inability can be established at a steady decrease of functional capacities of ill (aseptic necrosis of wrist bones).

Preventive measures. Complex mechanization of the most hard works and those operations, which demand fulfillment of a huge number of stereotyped motions with hands and fingers, is the most effective mean among preventive measures for warning of unfavorable effect of functional overstrain. Correctly organized operating mode and rest, gymnastics on a working place, massage and warm baths for hands, prophylactic vitaminization and general ultra-violet lighting, which are carried out in autumn and spring, have also important value.

Particular value in preventive measures of these diseases alongside with occupational selection is given to periodic medical examinations, which are carried out once every two years with participation of a doctor-neuropathologist, surgeon, gynecologist, and therapeutist.

Theme №7. INTOXICATION WITH AROMATIC HYDROCARBON

One of the simplest representatives **of aromatic hydrocarbons is benzol.** It is a colorless liquid with peculiar pleasant smell. It evaporates well at the room temperature. It is badly dissolved in water, but well in alcohol, ether and chloroform.

Benzol is widely used in various spheres of the industry: in rubber, chemical, pharmaceutical, polygraphic, paintwork, in the production of synthetic caoutchouc, explosive and medicinal matters. It is used as a solvent for fats and caoutchouc. The allowed concentration of benzol in the air of the work zone is 5 mg/m3.

Under production conditions, benzol gets into the body mostly in the form of vapors through respiratory organs and undamaged skin.

Benzol is discharged partially in the unchanged state through lungs, partially, it oxidizes to hydroxyl compounds - phenol and dihydroxybenzene, which are discharged via urine together with sulphuric acid or gluconic acid. Benzol belongs to poisons with general toxic polytropic action; but its well-known action is

the action onto the haematogenous system. This conditions listing of benzol with the group of "Blood poisons".

In the result of the action of benzol, both acute and chronic intoxications can be observed.

Pathogenesis. With acute poisoning, the action of benzol is mostly obvious in the central nervous system and it progresses according to the type of poisoning with narcotic poisoning.

Mostly, pathogenesis of chronic poisoning is in the inhibition of haemopoiesis - affection of proliferation of progenitor cells on haemopoiesis. Obviously, from the intensiveness (concentration of benzol vapors in the air of production territories) and the duration (number of work years in contact with benzol) impact, as well as from individual properties of the organism and its haematogenous organs (inherited inclination and previous diseases, which influence the blood system) depends the depth and the stage of affection of the marrow.

With the great intensiveness of toxic impact, the deepest affection of haematogenous organs is possible. In such cases, total inhibition of haemapoesis, disorder in proliferation of stem haematogenous cells and partially - predecessor of haemapoesis take place. Also, ability of these cells to differentiate can be affected. The result of such deep disorder of haemapoesis is progressing pancytopenia.

Less intensive toxic impact onto the marrow is accompanied by the inhibition of proliferation of differentiated blood cells (myeloblasts, erythroblasts and megacaryoblasts). Prevalent affection of granulocytopoiesis is possible here (progressing leukopenia) or thrombopoiesis (thrombocytopenia or hemorrhagic syndrome). Affection of germ of haemapoiesis can be assisted by pathologic changes or the impact ontpo a corresponding germ of haemapoisesis (fibromyoma, prolonged and excessive menses, gastric achylia, toxic impact onto the leucopoiesis of some medicinal drugs).

It has been stated that the toxic impact onto haematogenous cells are caused by not only benzol, as its transformations (phenols), which are created in the marrow, where benzol is accumulated. Thus, mutation in the chromosomal apparatus of haematogenous cells and the disorder of mitosis are conditioned by toxic impact of phenols.

Pathologic and anatomic pattern. Phenomenon of asphyxia is characteristic for acute intoxication with aromatic carbohydrates. Plethora of internal organs and spot hemorrhages in lungs, pleura, epicardium and mucous tunic of gastrointestinal tract; swelling, plethora of brain and its membranes as well as fine hemorrhages are observed.

At toxic intoxication with benzol, changes mostly take place in the haemopoiesis system. Hemorrhages into skin, mucous and serous tunics, internal organs, soft brain membranes, matter of cerebral hemispheres, and its ventricles. The permeability of walls of blood vessels increases; perivascular intermediate sclerosis. Marrow has mucus-like consistency and it is pink-yellow. On the microscopic level, hypoplasia of marrow is noticeable; more rarely, atrophy and aplasia pf the panmyelophthisis take place (Fig. 3).

All the shoots of marrow are affected. Sometimes, some areas of haemopoiesis stay. Together with the inhibition of haemopoiesis processes, there are cases with marked hyperplasia of marrow, up to leukemic pattern. Spleen is reduced, with phenomena of hemosiderosis. Liver is increased, and has signs of fat degeneration, hemosiderosis, intermediate sclerosis, and infiltrates with lymphoid and plasmatic cells. There are regenerated changes of epithelium of twisted channels in kidneys; ulcer on the mucous tunic, also much fat deposits in the hypodermic cellular tissue can be observed.

A c u t e i n t o x i c a t i o n s under production conditions can be observed rather rarely. They belong to accidental situations due to violation of safety rules. In the clinical pattern, changes in the central nervous system can be observed.

With light poisoning, general weakness, headache, dizziness, nausea, vomiting, noise in ears and jiggling when walking are observed. However, other transformations from the side of other organs and

systems are not observed. Sometimes, it is possible to notice some leukocitosis with stab shift, which passes fast.

With more marked intoxications, conditioned by the impact of significant concentrations of benzol, loss of consciousness and pupil change reaction can be observed. Intense respiration is slowed down, pulse is increased, and weak filling and arterial pressure are decreased. At the same time, marked leukocitosis can be often observed.

In case of provision of the corresponding assistance, recovery takes place comparatively fast. There are cases of sudden death from theparalysis of higher nervous centers with the action of high concentration of benzol (cleaning of tanks where benzol was).

C h r o n i c i n t o x i c a t i o n develops fast with prolonged impact of subtoxic concentrations of benzol. The severity and character of phenomena, which develop with poisoning, depend on work conditions (character of production processes, the temperature of the environment, concentration of benzol in the air, as well as duration of staying under these conditions), and from the organism state.

There are mild, mean and sever forms of chronic poisoning with benzol.

In mild cases of chronic poisoning, the most characteristic sign is the decrease of the number of leukocytes. At first, transitory leucopoenia is observed, then the decrease of the number of leukocytes is stable enough, and reaches the level 4. 0-109 per liter and lower. At this background, the decrease of the share of neutrophilic granulocytes with toxigenic grain, hyper segmentation of nuclears, and increase of the number of stab neutrophilic granulocytes can be observed.

From the central nervous system, neurasthenic syndrome prevails. Signs of hypertension are observed.

Chronic benzol poisoning of the mean degree of severity is characterized by further decrease of the number of leukocytes (up to 2. 8 -2. 0 - 10 9 per liter). It is also accompanied by thrombocytopenia, the number of thrombocytes decreases to 120 - 80-109 per liter. From the side of the central nervous system, asthenovegetative syndrome can be observed, which is manifested through headache, dizziness, general weakness, adynamy, increased irritability, sleep disturbance and hyperhidrosis. There can be changes in the peripheral nervous system according to the type of vegetative multiple neuritis, especially among those who has contact with benzol through arm skin.

Changes of the state of the cardiovascular system are characterized by hypertensia, liability of cardiac activity, hypertension, and moderately marked myocardium degeneration.

Patient's functions of stomach towards hyposecretion are in disorder, digestion process gets worse and the acidity of digestive juices reduces. The liver is moderately increased, and its function is in disorder. The number of y-globulin and sometimes, P-globulins is a little increased. Albuminoglobulin ratio is decreased.

Hemorrhagic syndrome (skin hemorrhages, epistaxis and menamenorrhagias), as well as positive symptoms of tourniquet and cuff takes place. The duration of hemorrhage increases; thromboplastic activity decreases; fibrinolisis is intensified, and the retraction of blood clot is in disorder. In the marrow, various manifestations of a mild form of hypoplastic state with the intensification of proliferate activity of myelocariocytes.

With the severe form of poisoning, marked anemia is observed; the number of erythrocytes decreases down to 1. 0-1012 in one liter; thrombocytopenia achieves the level of 30 - 35-109 per liter; the duration of bleeding increases to 15 - 20 min, and resistance to infections decreases. The following is characteristic: intense general weakness, drowse, noise in ears, dizziness, blackout, paleness of coverlets, and systolic noise in the upper part of the heart. Profuse hemorrhage appears (nosal, gastrointestinal and uterine hemorrhages). They are often accompanied by infectious complications (pneumonia, necrotic tonsillitis and septicopyemia). The latter can cause the death of patients.

The forecast in case of presence of a severe form of chronic intoxication with benzol can be rather unpleasant.

The described pattern of the chronic intoxication with benzol and the progress of hematological changes are rather conditioned. Thus, if in the air of the work zone where a worker is, the concentration of benzol is rather small, the pattern of poisoning is different. At first, anemia can take place, which later on is accompanied by leucopoenia and thromobpenia. In some cases, blood transformation starts with thromobopenia, and then the number of leukocytes and erythrocytes decreases.

Prolonged action of benzol can cause the development of chronic leucosis, which does not differ much according to its clinical pattern from the non-occupational one. Mostly it is chronic myeloid leucosis, sometimes - lymphoblastic leucosis and arethmia.

Treatment. With acute poisoning, it is necessary to terminate contact with benzol and stay outside as much as possible. In case of irritation of the mucous tunic of eyes, it is necessary to rinse them with 2 % of sodium bicarbonate, if respiratory tracts are irritated, then dionine is prescribed(0. 015 g) or codeine phosphate (0. 03 g two or three times a day). Good effect is provided by inhalation with the solution of baking soda; more severe forms of acute poisoning are the indication to prescribe oxygen. Together with it, hypodermic injections of 20 % of the solution of sodium caffeine-benzoate are prescribed in the amount o 1 to 2 ml or cordiamine of 2 ml. If in the future, changes in the liver take place, confinement to bed is prescribed; 15 to 20 intravenous injection 20 ml each of 40 %solution of glucose, 1 to 2 ml of 5 % solution of ascorbic acid. It is also possible to recommend a course (10 to 15) of intravenous injections of 10 % solution of calcium gluconate 10 ml each, which alternate with glucose injections (every other day). In addition, 20 pills three times a day are prescribed (total 15 to 20 days), vitamins B1 and B6. With chronic intoxications with benzol, curing measures are determined by the character of main clinical manifestations of the poisoning and the degree of their definition.

With the presence of neurological symptoms, sedative drugs are to be prescribed (sodium bromide - 0. 05 - 0. 1 g two to three times a day; tincture of valerian - 6. 0:200 ml - 1 table spoon three times a day; good result is provided by mild tranquilizers - meprobamate - 0. 2 g or tazepam - 0. 01 g two to three times a day). The duration of the course is two to three times. Together with it, it is recommended to administer 6 % solution of the vitamin B1 with 1 ml intramuscularly during 15 to 25 days; 5 % solution of vitamin B6 - 2 ml intramuscularly. The duration of the course is 1 to 1 V months.

In the clinical pattern of the blood affection, treatment should be conducted in a differentiated way with the consideration of current transformations. Hypoplastic state is the indication to prescribe pentoxile, which stimulates leucopoiesis and the production of antibodies. It is taken in after meals for 0. 2 to 0. 4 g three or four times a day, the course duration is 15 to 20 days. Leucopoiesis can be also stimulated by leucogen for 0. 2 three to four times a day during a week.

Administering of vitamins B1, B2, B6 and B12, often shows good results. Sometimes, together with listed medicinal drugs, glucocortocoids, iron-containing drugs and transfusion of blood of the same type are prescribed.

In case of anemic syndrome, administering of hemostimuline is prescribed (0. 5 g three times a day during meals).

Patients with toxic hepatitis are prescribed to take syrepar (intramuscularly - 2 ml during 30 days) and essentiale (2 capsules three times a day during meals).

Expertise of the ability to work. In case of acute poisoning with benzol of the mild degree, phenomena of intoxication pass fast (if further contact of the patient with benzol is terminated) and are not accompanied by the loss of the ability of the patient to work.

Recovery comes slower (on the fifth or seventh day) with the ointoxication of the medium degree, and a patient should receive a sick leave for this period. In the future, such patients are considered able to work on their occupation.

After a severe acute intoxication, stable outcomes of acute intoxication might stay after the recovery in the form of some syndromes of affection of the nervous system. Such patients are considered to have limited ability to work and should be assigned to face the Expert Commission to receive the invalidism group in the result of the occupational disease. Expertise of patients with chronic intoxication is conducted with the consideration of the character and the degree of the expression of hematological changes and the presence of other syndromes. With vaguely marked leucopoenia (4. 0-3. 0-109 per liter) and the absence of other transformations of blood and normalization of the pattern of blood, temporary promotion to another position with easier conditions and beyond the action of benzol for the period of two months and a sick leave should be provided for this period. Later, it is possible to continue work according to the occupation under conditions of strict keeping to sanitary and hygienic norms.

With more marked hematological transformations (II phase), patients should terminate the contact with benzol and its homologues completely. Patients should be assigned to face the expert commission to receive III group of invalidism and temporary pension for the period of re-qualification due to the occupational disease.

With the severe form of intoxication, patients should be considered unable to work (II group of invalidism) due to their occupational disease.

Preventive measures. To prevent poisoning with aromatic hydrocarbons, it is necessary to carry out sanitary and hygienic supervision of technological processes (sealing-in of the equipment, and effectiveness of ventilation), connected with the utilization of benzol and its homologues.

When carrying out some types of work, it is necessary to use individual means of self-protection to protect respiratory organs. With this purpose, filtering respirator is used, and in some cases, it is possible to use isolating respirator.

Considering the possibility of the permeation of benzol through undamaged skin, it is necessary to consider inexpedient to wash hands and uniform with paint spots with benzol, as well as the contact of bare parts of the body with it.

Besides benzol, its homologues are widely used in industries (toluol and xylol) and chloral derivatives (chlorbenzene, dichlorobenzene, etc).

Xylols - liquids, used as solvents to receive phthalic acids. Toxic action of xylols is characterized with the affection of the central nervous system and irritation of mucous tunic of eyes and respiratory tracts. Chronic intoxications are accompanied by the decrease of the level of erythrocytes, poikilocytosis, anisocytosis and moderate leucopoenia with relative lymphocytosis. Sometimes, some thrombocytopenia can take place. In case of getting to skin, the development of dermatitis is possible.

Dichlorobenzenes - are liquids, and paradichlorobenzene is a solid matter. Dichlorobenzenes are used as solvent, intermediate products to receive some dyes, as well as insecto-fungicides. In the clinical pattern of the intoxication with dichlorobenzenes are observed in the following cases:irritation of mucous tunic of eyes and respiratory tracts; in the blood the level of hemoglobin is decreased, the decrease of the number of erythrocytes and thrombocytes, as well as the appearance of methemoglobin.

Theme №8. INTOXICATION WITH NITROCOMPOUNDS AND CARBON OXIDE

Nitrocompounds and oilsperses of Benzol line are rather widely used in carious spheres of the industry, first in the chemical industry (productionof organic dyes, pharmaceutical preparations, explosive matters, reagents and some pesticides).

Aromatic nitrocompounds of the benzol line, most frequently met, **include nitrobenzene, dinitrobenzene, dinitrotoluene and trinitrotoluen**e. Most frequently used in the industry **oilspereses include aniline, benzidine and aminobenzene.**

Aniline is a colorless oleaginous liquid with mild aromatic smell; it darkens fast in the air; it dissolves well in ether, alcohol, and it dissolves badly in water. It can be used to produce dyes, artificial raisings, and color pencils, in pharmaceutical and rubber industries, as well as to produce explosive matters.

в - Naphthylamine is used in the production of azo dyes.

Nitrobenzene is a slightly oleaginous colorless liquid, which has the smell and taste of bitter almond. It dissolves well in ether and alcohol. It is practically non-dissolved in water and fats. It is used in the chemical industry to receive aniline, dyes, in perfumery, to produce soap and explosive matters.

Dinitrobenzene is a solid crystal matter of yellow color. Its vapors are 5. 8 times heavier that the air. It is used in chemical industry as an outcome product to receive dyes and nitroaniline.

Nitrotoluene is used in chemical industry to produce synthetic dyes, and in pharmaceutical industry.

In the organism of people, these compounds get with inhaled vapors or dust, through undamaged skin as well as in case of swallowing dust thought esophagus. Particular danger is in poisoning though the skin, as oilsperse and nitrocompounds of benzol can dissolve well in fats and lipoids. Favorable factors in this are hot seasons and skin dampness.

Aromatic oilsperses and nitrocompounds of the benzol line transform into aminophenol in the organism. In liver, they are neutralized by attachment with glucuronic acid and sulphuric acid with the creation of non-toxic vapor compounds, which are discharged by kidneys with urine.

Pathogenesis. Nitrocompounds and oilsperses of the benzol line are methemoglobin forming. Under the impact of these matters, blood pigmenthemoglobin is transformed into methemoglobin, which has dark brown color. Methemoglobin differs from the normal hemoglobinby the fact that bivalent iron hema is reverse and under the impact of oxidants, it transforms into a trivalent form, losing its ability to attach and transport molecular oxygen to tissues. Hypoxemia and hemic hypoxia develop. Nowadays, it is considered that pathological pigment is not created by the very aromatic nitrocompounds and oilsperes, but products of their transformation in the organism (phenyl-hydroxyl and nitrobenzene).

When poisoning with various aromatic nitrocompunds and oilspereses, methemoglobin disappears from blood after the termination ofthe impact of these matters within 2 to 6 days, renewing into an active form of heboglobin. Under appropriate conditions, oxidation of a-methyl group of porthyrin with further attachment of sulfur atom to pyrol rings progresses. Along with this, a pathological derivate of hemoglobin is created - sulfhemoglobin, which can also attach molecular oxygen. However, comparatively with methemoglobin, it is an irreversible pathological pigment, which is saved in erythrocytes until the end of their life cycle. Thus, presence of sulfhemoglobin in the blood is an important diagnostic criterion of the toxic influence of aromatic nitrocompounds and oilspereses.

Toxic impact of aromatic nitrocompound and oilsperses onto blood is accompanied by not only the creation of methemoglobin and sulfhemoglobin, but also with the appearance of inclusions in the form of oval grain, which are called bodies of Geins - Erlikh. Protein granulas, created from denaturated protein after dissociation of hemoproteid onto haems and globins are believed to take place. The number of bodies depends on the intensiveness of toxic impact. They appear more often just in several days after the beginning of intoxication. It is always a sign of a very deep damage of erythrocytes. Thus, in the basis of the creation of bodies of Geins - Erlich, there is denaturation and sedimentation of the pigment. The duration of life of erythrocytes with bodies of Geins - Erlich is reduced; they undergo mass ruining. Acute hemolytic anemia takes place, signs of which are the decrease of osmotic resistance of erythrocytes and the level of hemoglobin, ruining of erythrocytes and the increase of the content of indirect bilirubin, and marked urobilirubinuria. In the blood, there is a big amount of reticulocytes and even normoblasts. Changes of leucopoiesis are characterized by moderately marked neutrophilic with shifting to the left.

Thus, for poisoning with aromatic nitrocompounds and oilspereses of the benzol line, specific changes in blood are characteristic, like methemoglobinemia, sulfhemoglobinemia and presence of bodies of Geins and Erlich with further secondary hemolisis and the development of hemolytic anemia.

Besides, these compounds influence nervous system (like strong narcotic poisons they impact vitally important centers - respiratory, vasomotor and termoregulatory centers); liver (directoly on hepatocytes, assisting the accumulation of free fatty acids, triglycerides, lipids with the development of fat infiltration of cells, which is accompanied by the formation of strong cell poisons - alcohols, ketones, aldehydes and causes disorganization of cell metabolism and denaturation of proteins); cardiovascular system (directly onto the heart muscle - hypoxemic effect); skin and urinary tracts. Pathologic and anatomic pattern. Skin, mucous tunic of conjunctiva, tongue, and gums become pale gray, or pale yellow. Dot hemorrhages, and sometimes dingle ulcers are possible.

Blood becomes chocolate brown. Internal organs are plethoric. Marked fatty degeneration of liver can be observed, epithelium of intertwined channels are changed in a degenerated way and it contains hemosiderin. Spleen is increased, and is marked with deposits of hemosiderin.

Acute poisoning is characterized by the change of marrow. Yellow bone marrow is replaced with functioning red one. For chronic intoxication, sclerotic changes in all internal organs are characteristic.

A c u t e p o i s o n i n g. According to the clinical patter of acute poisoning with aromatic nitrocompounds and oilspereses of the benzol line, there are the following types of poisoning: mild, mean and severe.

In case of mild poisoning, presence of cyanosis of lips in some areas, nail plates on fingers. Face skin is pale with grayish shade. Patients complain to have weakness, drowse, and headache. The content of methemoglobin does not exceed 15 % of the general content of hemoglobin. In 1 to 2 hours after the termination of work, these phenomena disappear.

With poisoning of mean phase, the symptoms of intoxication are marked more. Well-marked skin and mucous tunic cyanosis takes place. Unusual coloration of skin and mucous tunic is conditioned by the increase methemoglobin in blood; creation of sulfgemoglobin and reduced hemoglobin. Patients complain to have acute headache, drowsiness, noise in ears, blinking in eyes, the state of shock, and sometimes loss of consciousness and decrease of muscle tone. Heart tones are dull and the pulse is accelerated. The content of methemoglobin achieves 30 to 40 % of the general content; the blood gets chocolate brown color; viscosity is increased and ESR is slowed down.

When poisoning with nitrobenzene and dinitrochlorbenzene, high level of methemoglobin is for 4 to 6 days, and when poisoning with aniline it is for 2 to 3 days.

In 3 to 4 days, pathological phenomena of the central nervous system weaken, but some changes in the peripheral nervous system can take place (pain in limbs and feeling of shivering.

In case of a sever form of acute poisoning with aromatic nitrocompounds and oilsperses, changes of blood go through the following three stages:

The first one: formation of methemoglobin and bodies of Geins-Erlich, which appear in blood on the 2nd - 5th day after intoxication.

The second one: hemolisis of degeneratively changed erythrocytes. The number of erythrocytes decreases sharply; the content of hemoglobindecreases; the content of indirect bilirubin increases. The term of progressing of this stage is from 5 to 8 days.

The third one: renewal of the blood content. Sharp increase of the number of reticulocytes takes place; basophile - grainy erythrocytes emerge; a general number of erythrocytes and the content of hemoglobin increase.

The renewal of the blood content takes place without utilization of specialized treatment methods. Complete renewal of blood is carried out for 3 to 4 weeks.

With acute poisoning, in particular, with nitrobenzene, the development of toxic hepatitis is possible. Slight increase of the liver is observed; dyspeptic phenomena increase, like nausea, vomiting, and loss of appetite. On the 3rd - 5th day, icteritiousness of sclera and mucous tunics appear. Skin becomes pale with yellowish shade of coloration. In blood, there is direct slowed down reaction of bilirubin; and bile pigments

- in urine are observed. The progress of toxic hepatitis is non-malignant. By the end of 2nd or 3rd week of hepatitis is subject to a reverse development. Severe acute poisoning with aromatic nitrocompounds and oilsperses can be met very rarely in industrial conditions. In the clinical pattern, there are mostly general brain phenomena, loss of consciousness and coma. In case of getting out of coma, retrograde amnesia, disorientation, obnubilation, headache and drowse can be observed. Pulse is from 120 to 150 beatings a minute; and the arterial pressure is lowered down. Sometimes, the content of methemoglobin is from 60 to 70 % in blood.

Phenomena of toxic hepatitis can be observed, and there is a threat of affection of kidneys (toxic nephropathy), especially in those cases, when intravessel hemolisis of erythrocytes and hemoglobinuria (hemolytic kidney).

C h r o n i c p o i s o n i n g. Chronic poisoning with aromatic nitrocompounds and oilsperses of the benzol line should include more or less stable diseases, which appear in case of prolonged impact of small dosages of these production poisons. Under their chronic impacts, biggest disorders can be observed from the side of such systems like blood, nervous system, liver and esophagus.

Toxic anemia is characterized by the development of stable anemia, for which it is characteristic to have the decrease of the coloration indicator and reticulocytosis, and the presence of 5 - 7 % methemoglobin is observed, and up to 1. 5 % sulfhemoglobin and bodies of Geihns-Erlich.

Changes leucopoiesis cause leukocitosis, and then leukopenia develops. Oxygen capacity of blood decreases significantly due to the decrease of the ability of hemoglobin to attach oxygen.

Affection of the nervous system can be characterized by complaints of patients to have headache, drowsiness, general weakness, worsening ofmemory, increased fatigability, sleep disorder, listlessness, and apathy. From the side of vegetative nervous system, hyperhydrosis and labiality of the arterial pressure are observed. Further, functional changes on the side of the central nervous system progress and transit into the microorganic symptoms system with the involvement of hypothalamus, as well as the development of hypothalamic syndrome.

Toxic hepatitis is shown through complaints of the sick to have pain, heaviness in the right hypochondrium, bitterness in the mouth, and non-perception of fat food. When examining a patient, moderate increase and pain in the liver, and in some cases, positive symptoms in gall bladder can be observed. The research of the functional state of the liver, as a rule, shows affections of hydrocarbon, anti-toxic, pigment, protein forming, excretory and other functions of this organ.

Disorder of pigment exchange at toxic hepatitis is rather specific. Light hyperbilirubinemia (without a clinical pattern of jaundice), which is characteristic for these patients, conditioned by the decrease of the fraction of free bilirubin, which appears due to low activity of the ferment of P-glucoronidase. In case of moderately marked chronic toxic hepatitis, the activity of such organ specific ferments for the liver, like ornithine-carbamyl-transferase, fructose bisphosphate-aldolase, and D-sorbitol-6-phosphate-dehydrogenase does not increase. There is also no disorder of fat exchange - the content of lipoproteins and cholesterol do not leave the boundaries of physiological waves.

Duodenal probing shows the absence or the weakening of cystic reflex, changes of the chemical composition of the B bile, in particular the decrease of the concentration of cholesterol and bilirubin. At cholecystorrhaphy, there are disorders of the movement function of the gall bladder.

Thus, chronic toxic hepatitis, which develops due to the action of aromatic nitrocompound and oilsperses, is characterized by the moderate expression (persisting hepatitis), mild disorder of main functions of the liver and dyskinesia of the gall bladder.

At chronic intoxications of with aromatic nitrocompounds, pathological process involves the esophagus as well.

Patients complain to have scattered pain in the suprastomach area after eating, nausea, belching, sometimes with "rotten egg" sensation, constipation, abdominal distension, and instability of defecation.

Functional and morphological research of the stomach enables to diagnose functional secretory and motor disorders, and more seldom, gastritis with mostly saved acidity. From the side of pancreas, decrease of the activity of pancreatic ferments in the duodenal content and the increase of their concentration in blood can be observed, but these changes bear functional character. As to the mechanism of such changes, it is necessary to consider both direct impact of aromatic nitrocompounds and their metabolites, and mediated impact due to the disorder of its neurohormonal regulation.

As to the heart and vessel system, patients suffer from the pain in heart, heart beating, and dyspnea at physical strain. Research of cardiovascular system enables to find the weakening of tones, inconstant systolic noise on the top of the heart and the inclination to hypertensia.

Changes of sight are characteristic only for some representatives of aromatic nitrocompounds - nitro derivative of toluene (trinitrotoluol). Bilateral toxic cataract of I - II degrees develops.

In case of prolonged action of some aromatic oilspereses (benzidine and P-naphthylamine), chronic irritation of the mucous tunic of the urinary bladder with products of metabolism of toxic matters (hemorrhagic cystitis, papilloma and malignant tumor of the urinary bladder) takes place. This disease can develop without symptoms for a long time and can be diagnosed only during cystoscopy.

At the action of such aromatic nitrocompounds and oilspereses, like Ursol, dinitrochlorbenzene, there is a threat of developing of allergic disease - bronchial asthma, dermatitis and eczema. There are cases of the change of the skin and hair coloration among those who work in contact with nitrocompounds and oilsperes of benzol for a long time. These matters are rather closely connected with proteins of skin and hair and dye them into brown when contacting with aniline, or in yellow - when contacting with trinitrotoluene.

Treatment. In case of development of acute intoxication, it is necessary to take the patient out of the contaminated zone. If toxic matters got to the skin, it is necessary to take off the contaminated clothes and to rinse the skin thoroughly with warm water and soap or a weak solution of potassium permanganate. Periodical inhaling of oxygen (from 20 to 30 minutes) is carried out for 5 to 10 hours. It is possible to alternate oxygen inhaling with carbogenes (5 to 7 minutes), which excites the respiratory center, improves the ventilation of lungs and assists the dissociation of oxyhemoglobin.

Good results are observed when prescribing cystamine, lipoid acid and ascorbic acid, intravenously, 10 to 30 ml of the preparation of "Chromosmone" (1 % solution of methylene blue in 25 % solution of glucose), 30 to 50 ml of 40 % solution of glucose, 20 to 30 ml of 30 % solution of sodium thiosulfate. At severe poisoning, article replacement of blood is used (3 to 4 liters) with the purpose to reduce the concentration of toxic matters and their metabolites, dilatation and reduction of blood viscosity. With this purpose, it is possible to conduct a forced diuresis. Incase of appearance of acute hepatitis, it is recommended to administer vitamins of Group B, tocopherol acetate, as well as lipotropins.

Treatment of patients with chronic intoxication should be complex with the consideration of the general state of a patient, as well as the major affection of corresponding systems and organs.

If functional disorders of the central nervous system have been found, it is recommended to administer sedative means, and in case of hypothalamic syndrome, intranasal iontophoresis or medical ionization is prescribed: the solution of 10 % calcium chloride, 2 % Dimedrol, and 5 % sodium bromide' with the inclination to arterial hypotonia - a complex of vitamins of the group B, eleuterokok, Chinese Schizandra and beloid are prescribed.

To get rid of hypoxemia and hypoxia, oxygen therapy is carried out. At toxic hepatitis, Diet No. 5, as well as lipotropic preparations (choline chloride), essentiale and legalon are recommended. Positive results are given by repeated duodenal probing, prescription of cholagogue preparations (alcohol, cholenism and hips). Patients with marked dyskinesia, bile-excreting tracts are prescribed to administer papaverine, no-shpa, platyphyllin and atropine.

Treatment for disorders of functions of stomach and kidneys should be individual with the consideration of the phase of progressing of the disease, character of changes of the secretory and movement

functions, the degree of morphological changes of the mucous tunic. Diet, vitamins and physiotherapy, as well as therapeutic exercises, small dosages of insulin (6 to 8 units) in combination with glucose (20 ml of 40 % solution) intravenously are prescribed.

Verification of the ability to work. At mild poisoning, patients are not able to work for a short period of time (for several days).

At acute intoxication of mean and severe degrees, temporary inability to work is 3 to 4 days. Then with the purpose to ensure the results of the treatment of patients, they are transferred to lighter work beyond the impact of toxic matters with the provision of a sick leave on occupational inability to work for 1 to 2 months. Further, they are considered capable to work according to their speciality.

In cases of mild chronic intoxication to ensure the treatment effect, patients are recommended to be transferred to another temporary position outside the impact of toxic matters for the period of 2 months with the additional payment if needed to provide average monthly payment according to the sick leave on the occupational inability to work. Further, they are permitted to work according to their occupation, but only under condition of keeping to sanitary and hygienic norms of labor. If the disease is a relapse, patients should be reemployed rationally (without the loss of qualification) at another place, which is more favorable in industrial meaning. In case of impossibility of such employment, a decision is made on temporary provision of invalidism group (for 1 to 2 years) due to the occupational disease until a new profession is not acquired.

At the moderately marked form of intoxication, further working contact with toxic matters is not recommended, and patients are subjects to rational employment; and in case of the reduction of the qualification -they should be sent to the Expert Commission to acquire an invalidism group.

Preventive measures. The basis of preventive measures is further limitation of the contact with toxic matters. It can be achieved due to mechanization of production processes, sealing-in the equipment and reconstruction of ventilation. Wet cleaning should be done in premises. All those who work in possible contact with these matters, should use individual protection and should have an opportunity to take a shower at work. Those, who are being employed or employees who contact with oilspereses and nitrocompounds of benzol, should go through preliminary and periodical medical examinations.

Intoxication with carbon oxide

Carbon oxide is a colorless gas, and in pure form, it has no smell or taste. It is lighter in weight than air; it does not dissolve in water and is well dissolved in the liquid ammonia.

Carbon oxide is a constituent part of a number of gases, which are used or created in industries. Producer gas contains from 9 to 29 % of carbon oxide, and blast-furnace gas contains up to30 %.

Under unfavorable sanitary and hygienic conditions, as well as if not to keep to rules of safety measures, occupational poisoning with carbon oxide at the industry can take place. Poisoning is possible in boilers, producer gas, blast furnace, open-hearth and foundry shops, as well as during testing of engines. Significant discharge of carbon oxide is possible during gunnery, bombardment and machine-gun fire, as well as in tank, armored cars and cartridge towers. The increased content of carbon oxide can be observed in the air of some shops of ceramic, brick, cement, construction industry, as well as engine-rooms of diesel locomotives, cockpits, garages, auto machines, motor boats and in chemical industry during synthesis of some matters, output material for which is carbon oxide. Due to the fact that the main part of carbon monoxide is carbon oxide, it is necessary to consider the possibility of domestic poisoning.

The main way for carbon oxide to get into a human organism is via respiratory organs. It can be discharged with air exhaling in an unchangeable state. Partial oxidation in the organism into carbon dioxide is possible.

Pathogenesis. According to toxic properties, carbon oxide is a strong poison, which impacts blood. High likeness of carbon oxide with bivalent iron to hemoglobin, which is almost 300 times higher than the

likeness of hemoglobin to oxygen and it conditions its toxic action onto the body. Carbon oxide, squeezing oxygen from its compounds with hemoglobin, creates carboxyhemoglobin. Whereas a part of hemoglobin is inactive, what infringes the transportation of oxygen to tissues and leads to the development of hypoxia.

The number of created carboxyhemoglobin is proportional to partial pressure of carbon oxide and is inverse to the pressure of oxygen in the exhaled air. At the increased content of carbon, oxide in the exhaled air initiates the process of dissociation of carboxyhemoglobin, which is mainly over after 7to 9 hours after single impact of carbon oxide. Such existing dependence is given in Table 8. Formation of carboxyhemoglobin is accompanied by the decrease of content of oxygen in the arterial blood with 20 to 12 %, arterial-venous difference up to 4-2 % (6 - 7 % in the norm), content of carbonic gas from 45 to 35 % (data in percentages are characterized with volumes of matters).

When poisoning with carbon oxide together with hypoxia, reduction of transportation form of iron in the blood takes place. Besides, at bigger concentration in blood carbon oxide has direct impact onto the cells of tissues, inhibits tissue respiration in the blood of brain, and carries out inhibitory impact onto the cytochrome-enzyme system.

Hypoxia and carboxyhemoglobin excites reflexes with carotid glomerules, have marked impact onto metabolism and the state of endocrine and vegetative system.

The boundary permitted concentration (BPC) of carbon oxide in the air of the industrial zone is20 mg/m3. If working for not more than one hour, BPC can be up to 50 mg/m3; if working for not more that 30 minutes

- it can be up to 100 mg/m3; and if working for not longer than 15 minutes

- up to 200 mg/m3. Maximal single BPC in the atmosphere air is six mg/m3, and average daily one - one mg/m3. For residential facilities of BPC is two mg/m3.

Pathologic and anatomic pattern. In the pathologic and anatomic pattern of acute intoxication with carbon oxide are observed in the dissemination of vascular changes. In many organs and systems (skin, muscles and brain), plethora, small and large hemorrhages, as well as degenerated changes and necroses can take place.

A characteristic sign is a relative coloration of skin and mucous tunic, which gains pink coloration.

Clinics. The clinical pattern of the acute poisoning with carbon oxide is diverse and is characterized by mainly changes of blood systems, disorder of the activity of cardiovascular and central nervous systems. The coloration of mucous tunic and skin is bright pink and intensiveness usually corresponds to the degree of the severity of intoxication. One of the syndromes, which have a decisive meaning in the diagnostics of acute intoxication with carbon oxide, is the change in the nervous system. At the action of some concentrations of carbon oxide take place passing symptoms of the disorder of the central nervous system, which are accompanied by a headache of pulsing character, mostly in the area of temples, nausea, vomiting, dizziness, general fatigability, weakness in legs, fast heart beating, and heart weakness.

At physical activity of a patient from the area, gas-laden with carbon oxide, and the provision of a corresponding assistance, all the listed above phenomena disappear gradually.

In case of prolonged action of significant concentration of carbon oxide, a severe form of poisoning develops, which is accompanied by the loss of consciousness and comatose state with complete inhibition of reflexes. When inhaling much carbon oxide, coma can take place immediately. During the coma, trismus or lockjaw can be defined, significant rigidity of muscles of the torso and limbs, dot cramps, pathological reflexes, disorder of cardiac activity and respiration. Depending on the severity of the intoxication, state of coma can last from several hours to several days. During growing disorders of cardiac activity and respiration (it becomes very rare and superficial), death can be caused by the respiratory center paralysis.

If the progress of toxic process is more favorable, then coma is replaced by a short-term period of movement excitement, in the basis of which there is the disorder of corticosubcortical activity, which appears on the background of external boundary dormancy, which is kept in the cortex. Patients jump, intend

to run, become aggressive, and cannot orient in time or space. Excitement goes away, after what they gradually lose consciousness. However, complete renewal of the psycho activity does not take place immediately. For a long time, patients are in spellbound state, which can be characterized by dormancy of psycho processes, indifference to the environment, and disturbance of memory.

In the distant period after severe forms of poisoning, in particular after prolonged coma, stable affection of nervous system can be observed. They include phenomena of Parkinsonism, which can be clinically defined in several months after poisoning. Obviously, changes, which take place on the height of intoxication in extrapyramidal system, and for some time they can develop clinically compensated. At the progressing of the process, a corresponding clinical symptomatology develop: anemia, movement stiffness and rigidity of muscular system. Peripheral sectors of the nervous system at acute poisoning with carbon oxide suffer much more rarely. Cases of the progressing of neuritis and polyneuritis are described.

If the form of intoxication if severe, swelling of retina can take place; in the fundus of eye sudden expansion of veins can be observed,small hemorrhages along vessels, which can later cause atrophy of optic nerves. In some cases, complete lose of sight is possible, caused by the affection of central sectors of sight analyzer.

Severe acute poisoning with carbon oxide can be accompanied by trophic changes of skin and other organs. Patients, who have been under the impact of carbon oxide for a long time, often have affected skin. In the initial period, these changes of skin are more or less well-outlined erythem, which is pigmented further. In a number of cases, on the background of erythema, blisters of different sizes, filled with transudation of yellow color, which remind burns. Blisters are localized on the skin of chest, hips and limbs. They burst easily; in case of infecting, it can be complicated with purulent process.

Main pathologic processes, which are observed at acute poisoning with carbon oxide, include changes in the peripheral blood. Thus, at the light degree of intoxication, polycythemia, increase of the content of hemoglobin, sometimes, neutrophilic leukocitosis, increase of the blood viscosity and slowing down of ESR can take place. On the height of intoxication, determine carboxyhemoglobin is determined in blood.

Patients with acute intoxication with carbon oxide have changes in the cardiovascular system. They are characterized with the appearance of tachycardia, widening of heart boundaries, and tone dullness. Often, there are various types of arrhythmia can be met. On the height of intoxication, arterial blood pressure is increased.

The possibility of c h r o n i c p o i s o n i n g with carbon oxide are denied by some researchers, but others consider them the result of numerous mild acute poisonings. Patients complain to have a headache, buzzing in the head, dizziness, increased fatigability, irritability, poor sleep, worsening of memory, short-term disorder of orientation, heart beating, dyspnea, states of unconsciousness, disorders of skin sensitivity, hearing and sight. Functional disorders of the central nervous system can be observed, like asthenia, vegetative dysfunction with angiodystonic syndrome, inclination to vessel spasms, and hypertension with further development of a hypertonic disease.

Chronic poisoning causes the development of arteriosclerosis. Possible disorders of a menstrual cycle, generative function among women, as well as unfavorable progress of pregnancy, and weakening of male sex functions.

The amount of hemoglobin and erythrocytes increase in the blood, and moderate anemia and reticulocytosis can be observed.

First aid and treatment. A sick person should be immediately taken outside in the fresh air, and stay in calm state and be warmed up. Oxygen inhaling should be started as soon as possible. At severe intoxications, urgent hyperbaric oxygen therapy is recommended for 1 to 1. 5 hours, and in case of the necessity, this procedure should be repeated.

On the background of oxygen therapy, the rest of therapeutic measures should be taken. In mild cases, alcohol, tea, coffee can be used; in case of nausea - 0. 5 % solution of Novocain can be used. Cordiamin and camphor can be administered hypodermically. During the first hours, 10 to 50 ml of chromosome, 20 ml of 5 % solution of ascorbic acid, 50 ml of 2 % solution of Novocain with 500 ml of 5 % solution of glucose and 1 or 2 ml of 5 % solution of pyridoxine can be administered intravenously. In case of brain swelling, the following lytic cocktail can be introduced intramuscularly: 2ml of 2. 5 % solution of aminazine, 2 ml of 2. 5 % solution of Dimedrol, 2 ml of 2. 5 % solution of promethazine, 1 ml of 2 % solution of promedol; 200 ml of 40 % solution of glucose (by drops intravenously) simultaneously with insulin- 10 units hypodermically can be administered. In case of cramps- enema with the solution of chloral hydrate (2 %, 100 ml) or barbamyl (10 %, 5 - 10 ml), with disorders of respiration - 2. 4 % solution of aminophylline 10 ml intravenously repeatedly, lobeline (1 %, 0. 3 - 0. 5 ml), and artificial respiration.

Verification of work ability. After treating of patients with acute poisoning of mean form in hospital, they are provided with an occupational sick leave and they stay under observation. Depending on the presence of severity of complications, their work ability can be limited, what conditions the invalidism of the occupational character.

Patients with initial signs of chronic intoxication are promoted to another job with the provision of an occupational sick leave for two months. In case of little effectiveness of the conducted treatment and preventive measures or marked symptoms, it is recommended to promote the patient to another job permanently with possible invalidism group on the occupational disease.

Preventive measures. Sealing-in of equipment and pipelines, where carbon oxide is possible to be emitted, full-time control over the concentration of carbon oxide in the air of facilities and fast withdrawal of the gas accumulated there, and automated alarm on unsafe concentration of carbon oxide.

Individual protection: if necessary, work in gas masks and respirators.

Theme №9. INTOXICATION WITH LEAD AND HYDROGEN ARSENATE

Intoxication with Lead

Lead is a soft, silvery-white or grayish metal. In the nature, it is mostly met in the form of sulphuric lead.

Poisoning with non-organic compounds of lead are more real in the mining and metallurgic industries, as well as in the production of lead paints and pigments, accumulators, during hardening of metal items in lead baths, in production of crystal, when soldering (utilization of lead solders), when cutting metal items, which are painted (red lead), as well as in polygraph enterprises.

Under production conditions, lead gets to the organism in the form of vapors and aerosol mostly via respiratory tracts. A danger of the lead getting through a gastrointestinal tract only exists in case of failure to keep to sanitary and hygienic rules (contamination of hands and eating and smoking at work places). A low content of protein, calcium and iron in the meals of those, who work under conditions of the contact with lead, can assist tot the increase of adsorption of lead in gastrointestinal tract. In the life, lead gets to the organism mostly though gastrointestinal tract with water, meals and in the result of contamination of hands.

The permitted concentration of lead in the air of the production zone is 0. 01 mg/m3.

Lead circulates in blood in the form of highly dispersed colloidal phosphate or albuminate of lead. It is mostly extracted via a large intestineand kidney. It can be detected in all the secretions (saliva, digestive juices, bile-excreting and breast milk). In urine in the norm - from 0. 04 to 0. 05 mg/l of lead; in faeces - twice or three times as much. Lead mostly deposits in bones, then in the liver and kidneys. At the disorder of acid-base balance lead can leave in the form of readily soluble lead phosphate and circulate in blood again. Such unfavorable impact can cause trauma, infection and alcoholism.

Lead is an anitplasmatic poison with a wide spectrum of the action. It causes mostly changes in the nervous system and cardio-vascular systems; disorder of ferment reactions, which participates in the synthesis of hemoglobin and a vitamin exchange; and decrease of immunobiological reactivity of the organism.

Pathogenesis. Lead interacts in the organism with active groups of proteins - sulfhydryl, amine and carboxyl. In the result, the activity of much ferment is affected; first, they participate in porphyrinic exchange including dehydratase 8-aminolevulinic acid and pherochelatese. The process of the transformation of tryptophan is affected. These changes slow down the formation of heme, and in the result of this and cytochrome, complicate the synthesis of pyridin nucleotides. In the result, energetic processes in cells are affected. Besides, under the impact of low concentrations of lead, the synthesis of RNA and DNA changes, and thus, plastic processes in cells are affected as well. It decreases adaptation opportunities of the organism and causes the increase of general sickness rate increases. The considered mechanisms are in the bases of syndromes, characteristic for the intoxication. The clinics of lead intoxication are given thought the connection merging of several syndromes.

Pathologic and anatomic pattern. At the lead intoxication in pathomorphological pattern, changes of nervous cells of anterior horns (cornu ventrale), where vacuolization, pigmentation, nuclear pycnosis, and dissolving of chromatophilic substance. Dystrophic changes in peripheral nodules can take place of the sympatic part of the vegetative nervous system and in general in periphery nerves. In cases of a severe affection in the brain and bone marrow, there are portions of hemorrhages and stases.

Clinics. Cardinal used to be characteristic for chronic intoxication with lead - lead border (dark gray, and sometimes, violet-flaky narrow line along the end of jaws) and the lead coloration (sallow gray color of a face) - now due to the improvement of the environment at the production, connected with lead; they lost their diagnostic meaning. Chronic intoxication with lead can be characterized with mostly affection in the blood system, affection of the nervous system and gastrointestinal tract.

Changes of biochemical indications in the blood, caused by the intoxication with lead, comprise disorders of порфириновый porphyrinic exchange; first of all aminolevulate- dehydrase reacts when an increasedamount of lead gets into the organism, the activity of which in erythrocytes decreases; the content of aminolevulinic acid, protoporphyrin and coproporphyrin increase in erythrocytes, which are considered the most reliable and specific sings of poisoning. The detected dependence of the expression of changes of porphyrinic exchange from the degree of the impact of lead. its content in blood and the severity of poisoning. Changes in the morphological pattern of blood - reticulocytosis, increase of the amount of basophile-grainy erythrocytes - refer to non-specific signs of saturnism, their diagnostic value is insignificant. Anemia at saturnism belongs to the group of hypochromic anemia, as its characteristic sign is hypochromia of erythrocytes at the increased content of iron in the blood serum (the so-called sideroachrestic anemia). In its development, a significant part is played by the direct impact of lead to erythrocytes, what leads to the reduction of the long term of their life. In the clinical pattern of the chronic lead intoxication, three stages can be distinguished:

Initial form of the chronic lead intoxication can be characterized by the absence of clinical signs and is determined based on the so-called laboratory symptoms of the intoxication. The content of aminolevulinic acid in the urine achieves 15 mg per one gram of creatine and coproporphyrin- 300 mkg per one gram of creatine. The level of lead in blood does not usually exceed 500 mkg/l, and in the urine - 100 mkg/l; reticulocytosis - up to 20 - 25 %, the amount of basophile-grainy erythrocytes increases up to 35 %.

Mild form of chronic lead intoxication is characterized by the joining of clinical symptoms. At this form of intoxication, the initial form of polyneuropathy can be diagnosed. Here, vegetative-trophic disorders can be diagnosed: pain, parasthesia, the feeling of numbness in limbs, especially at night at rest. Objectively at the neurological examination, the change of coloration of the skin on fingers can be observed (light cyanosis or paleness of the skin), hyperhydrosis, hypothermia, symmetrical distant disorders of the

sensibility, first in the form of hyperstesia, and then -hyperstesia, muscular hypotonia, dormancy of dermatographism, labiality of arterial pressure, and tendency to bradycardia. The decrease of the excitement of olfactory, gustatory and visual analyzers can be observed.

Changes in gastrointestinal tract at the mild form of chronic lead intoxication are expressed through the affection of stomach secretion (increase or decrease), processes of adsorption into the intestines, intestinal mobility with the development of dyskinetic syndrome. Functional disorders of the liver are possible.

Disorders of biochemical indicators at this form of intoxication of the lead are more marked: the content of aminolevulinic acid and coproporphyrin in urine can increase up to 25 mg and up to 500 mkg per 1 g of creatine correspondingly, the content of lead in blood, as a rule, doesnot increase 800 mkg/l, and in urine it reaches up to 150 mkg/l; reticulocytoosis - up to 40 %0, and the number of erythrocytes with basophile grains - up to 60 %0. Some decrease of the content of hemoglobin is possible.

Marked form of chronic intoxication with lead is characterized by the development of marked polyneuropathy, at this with sensitive disorders, movement disorders can be observed, and asthenovegetative disorders can develop.

The classical form of polyneuritis at the lead impact onto the body of a worker is the so-called antebrachial type of the paralysis. The syndrome is characterized by the major affection of extensors of hands and fingers. The process starts with the affection of bending extensor of fingers, and later it is accompanied by paresis of other finger extensors and hands, which stays in the position at right angle in a semi prone position. Fingers are bent; a thumb bends towards the palm (the so-called "hanging hand").

At the marked form of chromic intoxication with lead, the following can be observed very often: the so-called lead colic, which is expressed with fit-like pain in the abdomen, persistent constipation (the duration can be up to 10 - 14 days), which cannot be cued by laxative preparations; increase of arterial blood pressure, often with bradycardia, increase of the body temperature, as well as moderate leukocitosis and dark red color of the urine (due to the excretion of a big number of porphyrin). Sometimes, lead colic is accompanied by the affection of urinary tracts, and it develops as kidney colic. It is necessary to take into the consideration the possibility of the development of atypical vague forms of lead colic, progressing of which takes place during a long period of time in a wave-like form (from 3 to 4 months) and which are characterized by less marked clinic pattern and laboratory symptoms.

Recently, new data have been collected as to the mechanism of the development and progressing of lead colic. It is considered that at the action of lead onto the organism, autoantibodies are created, which, evenbefore the appearance of clinical indications of the lead intoxication assist to the development of immune complexes. Autoantibodies appear in the result of changes of antigenicproperties of erythrocytes due to metabolic disorders at the formation of heme or at the expense of creation of metal protein. These immune complexes, as well as erythrocytes with antigenic properties circulate in the peripheral blood, and first they affect normal blood provision in organs (at the expense of "plugging in" capillaries). It is caused by the disorder of microcirculation of organs and conditions a pain syndrome.

Nowadays under production conditions, lead colic starts gradually, with prodrome: increased fatigability in the end of a work day; general indisposition; pain in cortical bones, muscles and in the waist zone; loss of appetite, inclination to delaying of bladder emptying, irritability and sleep disorder. Sometimes, these phenomena appear together with pain in the stomach, which increase much and get cutting character.

For the marked form of chronic lead intoxication, the development of the anemic syndrome with the decrease of the level of hemoglobin lower than 130 g/l in men and 120 g/l in women is characteristic.

At the prologuned contact with lead, affection of the determined portions of bones and limbs can be noted: appearance of homogeneous levelly darkened intensive shadows in the metaphases in long cortical bones, which are much separated from the diaphyses of bones. Changes in the bone tissue at the intoxication

with lead are not accompanied by the destructive processes, changes in periosteum are absent. Mostly, large and small cannon-bones, hip, shoulder, elbow and spoke bones, as well as ribs are affected.

Biochemical disorders at the marked intoxication with lead are the most expressed. The content of the aminolevulinic acid and coproporphyrin in the urine is over 25 mg and over 500 mkg per 1 g of creatinine correspondingly. The concentration of lead in blood achieves 800 mkg/l and higher, and in the urine 0 over 150 mkg/l; reticulocytosis is higher than 40 %o; and the number of basophile-grain erythrocytes is over 60 %o.

Treatment. The most effective therapeutic means at chronic intoxication with lead is complexing agents, which create strong non-dissociating small toxic complexes together with lead, which can be easily taken out of the organism though kidneys. Mostly, 10 % solution of titacin calcium, which is administered intravenously once or twice a day for two to three days (20 ml in 500 ml of 5 % solution of glucose). Pentacin (especially at lead colic). Both preparations have high extracting activity regarding lead and are capable to terminate one of the most complex manifestation of saturnism - lead colic. Preparations are administered intravenously. Pentacin is administered in isotonic solution of sodium chloride or in 5 % solution of glucose - 200 ml once or twice a day, a dailydosage 2. 0 to 4. 0 g. The course of the treatment comprises three stages from 3 to 5 days of breaks between cycles. To treat intoxication with lead, D-penicillin is used in the daily dosage of 600 to 900 mg; it should be administered in 30 minutes after meals.

When treating patients with chronic intoxication with lead, which is accompanied by the neurological disorders, the following can be used: vitamins of group B; ascorbic acid; preparations, which have spasmolitic action, ganglionic blocker, and physiotherapeutic methods of treating.

If anemic syndrome is defined, the following is recommended: preparations of iron with hydrochloric acid, hemostimulin (hemostimulating agent, three times a day throughout a week); vitamin therapy - vitamin B12 (100 mkg) every other day intravenously (15 injections), vitamin B6intramuscularly (10 injections).

In case of kidney syndrome, the following is recommended: diathermy of the area of the liver; intravenous injections of 20 ml of 40 % glucose solution, hypodermic insulin injection (5 units); 300 mkg of vitamin B12 (10 to 12 injections); as well as vitamin K: 1 pill for five days.

Verification of the ability to work. The issue on verification of the ability to work at saturnism is solved depending on the expression of poisoning. At the initial form of intoxication, it is necessary to promote a person to another temporary workplace beyond the contact with lead for 1 to 2 months. In future, such patients can return to the same workplace (under condition of complete normalization of indicators of porphyrin exchange). In case of relapses of the intoxication, the worker has to terminate the contact with lead completely.

At the expressed form of intoxication, patients should be released from work with lead completely, even when complete disappearing of signs of saturnism can be observed in the result of treatment.

Preventive measures. The most effective preventive measure is, certainly, replacing lead and its compounds with other non-toxic matters at corresponding productions.

Maximum mechanization of operations of processing of materials which contain lead; sealing-in of sources of dust discharge; equipping of production zones with rational ventilation, mechanical purification of work premises from dust. In premises with much dust, people should work in respirators or industrial filtering gas masks.

When working with lead and its compounds, it is necessary to keep closely to the rules of personal hygiene, prohibit eating at work places; smoking should be permitted only on specially equipped rooms. Significant role in prevention of intoxication with lead is on preventive eating products with pectin matters (fruit non-clarified juices and apples), as well as preliminary and periodic medical examinations. Intoxication with Hydrogen Arsenate

Hydrogen arsenate is a heavy colorless gas. Under industrial conditions, it is a by-product, which is created under the action of technical acids onto metals (tin, copper and bismuth) and compounds with arsenic. Hydrogen Arsenate is extracted when soldering and treatment of metal products with acids, filled in accumulator batteries on submarines and galvanization. The permitted concentration is 0. 3 mg/m3.

Hydrogen arsenate permeates into the organism through respiratory organs, and less often through undamaged skin and gastrointestinal tract.

Pathogenesis. Arsenic compounds block sulfahydril groups of ferments and create stable toxic complexes. Arsenic disturbs carbohydrate and lipid exchange, as well as tissue respiration. In the pathogenesis of intoxication with arsenic, capillary toxic and hemolytic effects, as well as irritating action onto the skin and mucous tunic.

Hydrogen arsenate is a strong hemolytic poison and methemoglobin creator. Mechanism of hemolytic action of hydrogen arsenate, which has not been clarified yet. Probably, hemolysis is conditioned by a number of factors:

1) Inhibition of catalase, which is contained in erythrocytes, in the result of which a big number of hydrogen peroxide, which causes hemolysis;

2) formation of the following products inside erythrocytes, first of all, which have the hemolytic property, first of all, hydrolases; together with this hydrogen arsenate destroys glutathione, which can inhibit hemolysis;

3) accumulation of metal arsenic in erythrocytes, which ruin the structure of the latter, what causes their dissociation.

The result of hemolysis is anemia of parenthymatosic organs, nervous and cardiovascular systems. Changes of intermediary metabolism, which is characteristic for hypoxemic states, can be observed: increase of the content of sugar and lactic acid in the blood, and decrease of alkaline reserves in blood.

Thus, anoxia, which develops at intoxication of hemolythic character, conditions the general hypoxia with accompanying manifestations. Serious disorders of functions of kidneys and liver, which can be observed at the action of hydrogen arsenic, cannot be explained but by hemolysis. An important part is played by direct action of arsenic, which is formed in the process of oxidation of hydrogen arsenate.

At the action of hydrogen arsenate in people can be observed in blood (hemolytic anemia, a big number of retyculocytes, basophile-grain erythrocytes appear, and the number of SOE is increased), of kidneys (coloration of urine change - the color of "meat slops", proteinuria, high specific gravity and uremia can be observed), of liver (toxic hepatitisphenomena), of cardiovascular system (increase of heart sizes, tachycardia, and arterial hypotonia), of nervous system (headache, dizziness, drowse or sudden excitement).

Clinics. A c u t e p o i s o n i n g appears suddenly. On the clinical progress, three forms of acute poisoning with hydrogen arsenate can be distinguished.

Mild poisoning can be characterized by general weakness, illness, headache, nausea, desire to vomit, and pain in limbs. Sclera and skin coverlets are weakly icteric. Sometimes, blood urine can take place. The number of erythrocytes and hemoglobin in blood gradually decreases. The aforementioned phenomena disappear fast and recovery takes place in several days.

With poisoning of mean severity besides the given above complaints, patients are chilling, they get much ache in the waist, and temperature rises. On the first day, jaundice develops with specific bronze coloration of skin coverlets. In the coverlet, the content of indirect bilirubin increases; the number of erythrocytes and hemoglobin decreases; retyculocytes and basophile-grain erythrocytes appear. Hemoglobinuria, albuminuria and detritus occur. Often, on the third or fourth day, increased liver, moderate tachycardia can be observed; systolitic noise can be heard on the upper part of the heart; and blood pressure decreases. When treating, improvement of the health state occurs gradually, and recovery takes place in four to six weeks.

Severe poisoning. After the latent period, which lasts for 2 to 3 years, first indications of intoxication take place: intense headaches, feeling of cold, increase of temperature up to 38 - 39 0C and vomiting.

Urine gets brown coloration and contains a great number of renewed hemoglobin and protein can be found in it. Then, sudden fall of the number of hemoglobin and erythrocytes can be observed; the content of bilirubin increases. At the end of two to three days, there are symptoms of affection of livers, and direct bilirubin can be determined.

If the disease progressing, kidney decompensation can develop, which is observed on the fourth or fifth day and is characterized by the presence of uremia, and marked hemolytic of jaundice. Headache and drowse increase, nausea and vomiting appear. Further, urine is terminated and phenomena of uremia take place. Death occurs mostly at the end of the first week.

At relatively favorable progressing of the disease, the recovery period can be accented. The state of the patient gradually improves, jaundice disappears, the number of erythrocytes and hemoglobin increases. Complete recovery can be stated only in 2 to 2 V months.

Distant consequences of poisoning of hydrogen arsenate can involve the following: severe anemia and obesity of parenchymal organ, as well asthe decrease of resistance of organism as to the infection; secondary nonspecific pneumonia, changes in kidneys in the form of glomerulonephritis; chronic hepatitis and disorder of functions of bile-excreting tracts.

C h r o n i c i n t o x i c a t i o n. It occurs very rarely. For it, secondary anemia is characteristic, which develops without marked clinical manifestation of hemolysis.

Patients complain to have headache, sleep disturbance, bad appetites, pain in epigastric area, indigestion disorder, fast fatigability, loss of weight, and sometimes - petechial hemorrhage on the skin and general asthenia. In the blood, anemia, basophile grain of erythrocytes, and moderate leukocitosis can be found.

Treatment. Confinement to bed, complete tranquility. Warmth is recommended. During the first hours of the development of hemolysis -bloodletting in the amount of 300 ml of blood with its further transfusion, and intravenous administering of glucose for 500 ml of 10 % solution of ascorbic acid (300 - 500 mg) by drops. Hypodermic injections of isotopic solution of sodium chloride - up to one liter. Rectal drips of 5 % solution of glucose (500 ml), insulin (5 - 10 units) hypodermically. Administering of alkaline. Diathermy of spots of kidneys. Cordiamin, Corazol and adrenalin. With strong vomiting - injections of morphine with magnesium sulfate. Intravenous administering of 20 to 30 ml of 30 % solution of sodium thiosulfate. Oxygen inhalations. Immediate administering of mecaptid hypodermically or intramuscularly in the dosage of 1 ml of 40 % oil solution: the first day - three injections every 4 to 5 hours, and on the second and third days - two injections in 8 to 12 hours.

At stable anuria, usage of peritoneal dialysis, hemodialysis with the utilization of artificial kidney apparatus and generative restorative treatment.

Verification of the ability to work. After severe acute intoxication, as well as in case of marked chronic obligatory release from the contact with hydrogen arsenate and other toxic matters.

Preventive measures. General sanitary and hygienic means. Chemical control on the state of air environment. Previous and periodical medical examinations.

Intoxication with Hydrogen Arsenate

Hydrogen arsenate is a heavy colorless gas. Under industrial conditions, it is a by-product, which is created under the action of technical acids onto metals (tin, copper and bismuth) and compounds with arsenic. Hydrogen Arsenate is extracted when soldering and treatment of metal products with acids, filled in accumulator batteries on submarines and galvanization. The permitted concentration is 0. 3 mg/m3.

Hydrogen arsenate permeates into the organism through respiratory organs, and less often through undamaged skin and gastrointestinal tract.

Pathogenesis. Arsenic compounds block sulfahydril groups of ferments and create stable toxic complexes. Arsenic disturbs carbohydrate and lipid exchange, as well as tissue respiration. In the pathogenesis of intoxication with arsenic, capillary toxic and hemolytic effects, as well as irritating action onto the skin and mucous tunic.

Hydrogen arsenate is a strong hemolytic poison and methemoglobin creator. Mechanism of hemolytic action of hydrogen arsenate, which has not been clarified yet. Probably, hemolysis is conditioned by a number of factors:

1) Inhibition of catalase, which is contained in erythrocytes, in the result of which a big number of hydrogen peroxide, which causes hemolysis;

2) formation of the following products inside erythrocytes, first of all, which have the hemolytic property, first of all, hydrolases; together with this hydrogen arsenate destroys glutathione, which can inhibit hemolysis;

3) accumulation of metal arsenic in erythrocytes, which ruin the structure of the latter, what causes their dissociation.

The result of hemolysis is anemia of parenthymatosic organs, nervous and cardiovascular systems. Changes of intermediary metabolism, which is characteristic for hypoxemic states, can be observed: increase of the content of sugar and lactic acid in the blood, and decrease of alkaline reserves in blood.

Thus, anoxia, which develops at intoxication of hemolythic character, conditions the general hypoxia with accompanying manifestations. Serious disorders of functions of kidneys and liver, which can be observed at the action of hydrogen arsenic, cannot be explained but by hemolysis. An important part is played by direct action of arsenic, which is formed in the process of oxidation of hydrogen arsenate.

At the action of hydrogen arsenate in people can be observed in blood (hemolytic anemia, a big number of retyculocytes, basophile-grain erythrocytes appear, and the number of SOE is increased), of kidneys (coloration of urine change - the color of "meat slops", proteinuria, high specific gravity and uremia can be observed), of liver (toxic hepatitisphenomena), of cardiovascular system (increase of heart sizes, tachycardia, and arterial hypotonia), of nervous system (headache, dizziness, drowse or sudden excitement).

Clinics. A c u t e p o i s o n i n g appears suddenly. On the clinical progress, three forms of acute poisoning with hydrogen arsenate can be distinguished.

Mild poisoning can be characterized by general weakness, illness, headache, nausea, desire to vomit, and pain in limbs. Sclera and skin coverlets are weakly icteric. Sometimes, blood urine can take place. The number of erythrocytes and hemoglobin in blood gradually decreases. The aforementioned phenomena disappear fast and recovery takes place in several days.

With poisoning of mean severity besides the given above complaints, patients are chilling, they get much ache in the waist, and temperature rises. On the first day, jaundice develops with specific bronze coloration of skin coverlets. In the coverlet, the content of indirect bilirubin increases; the number of erythrocytes and hemoglobin decreases; retyculocytes and basophile-grain erythrocytes appear. Hemoglobinuria, albuminuria and detritus occur. Often, on the third or fourth day, increased liver, moderate tachycardia can be observed; systolitic noise can be heard on the upper part of the heart; and blood pressure decreases. When treating, improvement of the health state occurs gradually, and recovery takes place in four to six weeks.

Severe poisoning. After the latent period, which lasts for 2 to 3 years, first indications of intoxication take place: intense headaches, feeling of cold, increase of temperature up to 38 - 39 0C and vomiting.

Urine gets brown coloration and contains a great number of renewed hemoglobin and protein can be found in it. Then, sudden fall of the number of hemoglobin and erythrocytes can be observed; the content of bilirubin increases. At the end of two to three days, there are symptoms of affection of livers, and direct bilirubin can be determined.

If the disease progressing, kidney decompensation can develop, which is observed on the fourth or fifth day and is characterized by the presence of uremia, and marked hemolytic of jaundice. Headache and drowse increase, nausea and vomiting appear. Further, urine is terminated and phenomena of uremia take place. Death occurs mostly at the end of the first week.

At relatively favorable progressing of the disease, the recovery period can be accented. The state of the patient gradually improves, jaundice disappears, the number of erythrocytes and hemoglobin increases. Complete recovery can be stated only in 2 to 2 V months.

Distant consequences of poisoning of hydrogen arsenate can involve the following: severe anemia and obesity of parenchymal organ, as well asthe decrease of resistance of organism as to the infection; secondary nonspecific pneumonia, changes in kidneys in the form of glomerulonephritis; chronic hepatitis and disorder of functions of bile-excreting tracts.

C h r o n i c i n t o x i c a t i o n. It occurs very rarely. For it, secondary anemia is characteristic, which develops without marked clinical manifestation of hemolysis.

Patients complain to have headache, sleep disturbance, bad appetites, pain in epigastric area, indigestion disorder, fast fatigability, loss of weight, and sometimes - petechial hemorrhage on the skin and general asthenia. In the blood, anemia, basophile grain of erythrocytes, and moderate leukocitosis can be found.

Treatment. Confinement to bed, complete tranquility. Warmth is recommended. During the first hours of the development of hemolysis -bloodletting in the amount of 300 ml of blood with its further transfusion, and intravenous administering of glucose for 500 ml of 10 % solution of ascorbic acid (300 - 500 mg) by drops. Hypodermic injections of isotopic solution of sodium chloride - up to one liter. Rectal drips of 5 % solution of glucose (500 ml), insulin (5 - 10 units) hypodermically. Administering of alkaline. Diathermy of spots of kidneys. Cordiamin, Corazol and adrenalin. With strong vomiting - injections of morphine with magnesium sulfate. Intravenous administering of 20 to 30 ml of 30 % solution of sodium thiosulfate. Oxygen inhalations. Immediate administering of mecaptid hypodermically or intramuscularly in the dosage of 1 ml of 40 % oil solution: the first day - three injections every 4 to 5 hours, and on the second and third days - two injections in 8 to 12 hours.

At stable anuria, usage of peritoneal dialysis, hemodialysis with the utilization of artificial kidney apparatus and generative restorative treatment.

Verification of the ability to work. After severe acute intoxication, as well as in case of marked chronic obligatory release from the contact with hydrogen arsenate and other toxic matters.

Preventive measures. General sanitary and hygienic means. Chemical control on the state of air environment. Previous and periodical medical examinations.

Theme №10. DISEASES OF BRONCHOPULMONARY APPARATUS OF TOXIC-CHOMICAL ETIOLOGY

In various spheres of industry - metallurgic, chemical, oil-processing, pulp and paper, pharmaceutical, and mechanical engineering - mix of chloral, sulfur and nitrogen have become rather popular. They all can be in the air of the work zone in the form of irritating gases: chloral, chlorohydrogen, sulphur trioxide, hydrogen sulphide, nitrogen oxide and ammonia.

Chloral is a gas of green and yellowish color with strong odor. It is 2 V times heavier than air and possesses good oxidation power. It has irritating and reflexive action onto the body. In case of irritation of interoreceptors of mucous tunic of upper respiratory tracks, spastic phenomena can take place in bronchi, the heart activity changes, and phenomena of irritation of respiratory and vessel centers are observed.

Poisoning with chloral is possible in pulp and paper, and textile industries, where chloral is used for bleaching; as well as in pharmaceutical industry to make chloride of lime or bleaching powder.

Sulphur trioxide is colorless gas with strong irritating smell. It can be well dissolved in water, ethyl or methyl alcohol. Mostly, sulphur trioxide is in the industrial atmosphere of metallurgic workshops, workshops where sodium sulfite is produced, as well as in refrigerators. The main way of its introduction to the human body is through respiratory organs. In the organism, it can be found in blood. It acts as an irritant of mucous tunic of eyes and upper respiratory tracks and later it can affect lungs. It also has resorptive properties as it affects metabolism processes.

Sulphuretted hydrogen is colorless gas with characteristic smell of a bad egg. It is a bit heavier than air and thus it is accumulated in hollows, like pits and trenches.

Sulphuretted hydrogen is emitted into the air when producing viscose fiber, as well as when using dues at textile enterprises, and during mining and processing of polysulfide oil. It can be contained in sewage water in canalization pipes. Main way of getting to a human body is via respiratory organs. During accidents, such a big amount of sulphuretted hydrogen can get into the body, which caused acute poisoning.

Nitric oxides are gases of yellow-brown color. They look like a mix of the most spread nitrogen dioxide. These gases are created under production conditions when producing sulphuric acid, chromic acid, nitric acid, aromatic nitrocompounds, aniline dyes, as well as during oxy-acetylene welding, flame cutoff and electric welding.

Acute toxic affection of bronchopulmonary apparatus. Main clinical forms of acute affection of bronchopulmonary apparatus of toxico-chemical etiology are as follows: acute toxic laryngitis-pharyngitis-tracheitis, acute toxic bronchitis, acute toxic bronciolitis, acute toxic swelling of lungs, as well as acute toxic pneumonia.

Under industrial conditions, they can appear in accidental situations, in case of inhaling toxic matters of significant concentrations. Appearance of intoxication is caused by the following: high concentration of the chemical matter in the air; duration of its action; general reactivity of the organism; as well as physical and chemical properties of poisoning matters.

Pathogenesis. Irritating matters when having impact onto the body of the worker, get in contact with the moisture of respiratory tracks and creation comounds, which have burning properties (hydrochloric acid with the action of chloral, salt cake - sulphuretted hydrogen, etc). These compounds cause disorders of respiratory functions due to local irritating action, as well as the disorder of the reflector character (impact onto the interoreceptors of bronchis). The result is spastic contraction of heart muscles, respiratory and cardio-movement centers. Spreading and heaviness of affections depend on the degree of dissolving of toxic matters in water.

Matters, which are easily dissolved in water (chloral, chloropicrin, ammonia, and sulphuretted hydrogen), have impact mostly onto the mucous tunic of tracheas and large bronchi. Clinically, it is manifested by an acute laryngotracheitis and acute toxic bronchitis.

Matters, which are difficult to be dissolved in water (nitrogen oxide, phosgene, chloropicrin and dimethyl sulphate), they deeply permeate into the bronchopulmonary system, and affect small bronchi and bronchioles. And clinically, this can be conditioned by the appearance of acute bronchitis and broncholitis.

Acute toxic laryngotracheitis. In the clinical development, there are three phases of severity.

Mild phase is characterized by hyperemia of mucous tunic of upper respiratory tracts; and in some places small hemorrhages are observed.

Mean phase: swelling of mucous tunic, coarse voice and sometimes its complete loss are observed.

Severe phase is characterized by the necrosis of mucous tunic with creation of ulcer in it. Possible development of acute swelling of lungs with further asphyxia and death of the patient are observed.

Acute toxic bronchitis. There are three phases of the severity of acute toxic bronchitis.

Mild phase: (superficial or catarrhal toxic bronchitis) is characterized by the following: painful coughing, pain and "scratchy" throat, squeezing and scorching throat, as well as obstructed breathing. Patients have epiphora andlight phobia. During the percussion, it is possible box sound can be heard, mostly in lower side portion of lungs, during auscultative examination, scattered dry rales can be heard on the background of coarse breathing. Duration of this phase of bronchitis is from 3 to 7 days; complications are absent.

Mean phase: patients complain to have rhinorrhea, epiphora, obstructed breathing via a nose, intensive pain in the chest, fit-like coughing with phlegm discharge. Cyanosis and dyspnea can be well heard. Above lungs, signs of emphysema are observed; during auscultative examination - dry rales and sometimes some moist rales can be heard. As to the cardio-vascular system - tachycardia is observed. As a rule, the temperature of body is increased to low grade, moderate neutrophilic leukocitosis, as well as increased ESR. The duration of this phase of bronchitis is from 7 to 10 days.

Severe phase is characterized by the presence of marked cyanosis, and dyspnea at rest. Objective signs of emphysema include dry and moist rales all over the surface of lungs. This phase lasts for 2 to 6 weeks, and in case of adequate treatment, complete recovery is possible. Further progress of acute toxic bronchitis is possible under conditions of joining infection, its transition into a chronic form with the development of pneumosclerosis.

Acute toxic bronchiolitis. It develops during the impact of toxic matters of irritating action, first of all those, like dimethyl sulfate, joining of beryl.

In mild cases, patients complain to have coughing, small amount of phlegm, moderate dyspnea, and low grade fever. Above the lungs, basal emphysema can be observed; small moist crepitations in small amount can be heard.

From the point of peripheral blood, small leukocytosis and increased ESR can be observed. This stage lasts for from several days to 2-3 weeks.

In more marked phases, dyspnea is increased, coughing becomes unbearable, and sometimes it is fit-like, and it is accompanied by pain in the chest, and thick mucoid sputum. Patients complain to have headache, loss of appetite, increase of temperature to 38 - 39 °C, and general weakness. Marked cyanosis takes place, and tachypnea (30 - 40 a minute). Objectively, there are sings of emphysema, ends of lungs are lowered; their movement is reduced. Above all the surface of lungs and especially in lower portions, big number of medium and small bubbling moist capitations can be heard. As to the cardio-vascular system, tachycardia can be observed; decrease of arterial pressure and muffled heart sounds can be heard. Liver increases, it becomes more painful during palpitation.

In the peripheral blood, increase of hemoglobin, erythrocytes, leukocytosis with stab neutrophil disorders, relative lymphopenia, and sometimes, eosinophilia can be observed, and ESR increases up to50 mm/h. Proteinuria and cylindruria can be observed in urine. On the radiogram of chest organism, decrease of transparency of lung field in mean and lower portions, and there are also fine formations, which merge in some places. Lung roots are expanded.

Patients with acute toxic bronchiolitis either recover or receive various complications: bronchopneumonia, transition to the chronic form with obliteration of the lumen bonchioles.

Acute toxic pneumonia. It appears in the result of short-term impact of the toxic matters and is characterized of acute beginning, short-term progress, and absence of the inclination to the appearing of new centers.

Pneumonia in the result of the action of gas appears in an acute form, within several hours after accidental aspiration of petroleum or its permeating into the respiratory ways. Cyanosis, dyspnea and intensive pain in the half, which is the location of pneumonia infiltration, are observed, as well as coughing with prune-juice sputum. The temperature increases to 40 °C.

During percussion, atrophy of percussion sound is observed on the side of affection; and during auscultative examination: bronchial breathing and crepitations are observed. In the peripheral blood, there are sings of the inflammatory process (leukocytosis, lymphopenia and the increase of ESR).

On the radiological picture, an area of pneumonic infiltration in the form of homogenous shadowing, which is localized mostly in the front portion of lungs.

Treatment. Oil and alkaline inhalations are recommended, as well as antitussive drugs (tussuprex and libexin), and antibiotics.

Acute toxic swelling of lungs is the most serious and dangerous form of acute toxic affection of bronchopulmonary apparatus. Etiological indications of its appearing can be nitrogen oxides and chloropicrin.

Pathogenesis. In the mechanism of development of toxic swelling of lungs, an important place is taken by the impact of toxic matter onto the activity of ferment systems, which contain SH groups. They are accompanied by high increase of the permeability of alveolar membranes with the disorder of metabolism. In the interstitial tissue, high protein liquid is accumulated in alveoli.

Besides an immediate action of toxic matters onto the ferment systems, the mechanism of toxic swelling of lungs is impacted, which is proved by the decrease and often prevention of the development of lung swelling due to elimination of some sections of the nervous system (vagosympathetic blockade and cutting of the vagus nerve on the neck).

In the development of toxic swelling of lungs, five periods can be named:

The first one or the period of irritation. Clinical manifestation: epiphora, coryza, coughing, "scratchy" throat, and squeezed chest; duration: 15 to 20 minutes. The second one or the period of imaginary well-being. Clinical manifestation: insignificant dyspnea, pulse instability, and signs of moderate emphysema; duration from 3 to 8 hours.

The third period or the period of the increase of the swelling. Patients complain to have squeezed chest, dyspnea, coughing with phlegm discharge. During the percussion, box sound above the lungs; during the auscultative examination: fine moist rales. The number of rales increases fast.

On the radiological picture of lungs, the pattern is vague and roots are expanded.

As tot eh peripheral blood, increase of ESR is observed, as well as leukocytosis with the motion of the formula to the left and lymphopenia.

The fourth period or the period of the end of swelling. Clinical manifestation: dyspnea and coughing increases, blood phlegm is discharged, and breathing is bubbling. During auscultative examination, significant number of heterogeneous moist rales.

Radiologically, it is possible to observe spotty shadows, which are conditioned by the accumulation of swelling liquid in alveoli. These shadows create big merged areas in some places.

When researching blood, its clotting is observed, which is manifested through the increased of hemoglobin, eretrocytes and leukocytes. Blood viscosity is observed.

The amount of oxygen in the arterial blood decreases sharply, but the amount of carbon dioxide increases; the so-called hypercapnic (blue) type of hypoxemia develops. The arterial pressure is normal or a little increased. Blue asphyxia can develop into the gray one.

The gray type of hypoxemia is characterized by low contain of blood of carbon dioxide, merging of the swelling of lungs with the decrease of cardiovascular activity (collapse). The patient's face gets gray pale hint and is covered with cold sweating. Mucous tunics are of dirty gray color. Limbs are cold and damp when touched. The pulse is frequent and thready; the arterial pressure decreases fast.

The fifth period or the period of reverse development of the toxic swelling. It is characterized by the decrease of dyspnea, cyanosis, coughing, and the amount of discharged phlegm, as well as crepitations in lungs. The pattern of peripheral blood is normalized. In 2 to 4 days, a patient recovers. The temperature stays increased (37. 5 to 38. 0 °C) during the week.

Complications: pneumonia and the development of pneumosclerosis.

Treatment. Patients are recommended to take tea, coffee, and put a hot-water bottle. They are recommended to take oxygen (oxygen inhalations are compiled with vapors of 30 % alcohol with the purpose to prevent the creation of foam). Eyes are washed with 2 % solution of sodium chloride and instill with albucid (30 % solution), Novocain 1 - 2 drops of 1 % solution. A nose and throat are rinsed with the solution of sodium bicarbonate. In case of the development of spasms of glottis, 1. 0 ml of 0. 1 % solution of atropine hypodermically.

People, who had contacts with damps, need to be examined in hospital. With the purpose to decrease the permeability of vessels, 5 to 10 ml of 10 % solution of calcium chloride are introduced intravenously.

With strong hypoxemia, the following is recommended: oxygen therapy (oxygen with alcohol vapors); bloodletting (from 300 to 500 ml of blood) under the control of the arterial blood.

With the gray type of hypoxemia are recommended: inhalations with oxygen (60 % with the addition of 5 % of carbonic acid); means which stimulate the respiratory center (caffeine, Corazol, and ephedrine); antibiotics and sulfanilamides medicines with the purpose of infection prevention (development of bronchopneumonia).

Verification of the ability to work. In cases of mild affection of bronchopulmonary apparatus with matters of toxic and chemical action, patients return to their work.

For patients with mean or severe phases of affection, it is necessary to make sure that results of the treatment stay for long; these patients need temporary termination of work in the areas with the contact with chemical matters of irritating nature. In case of the decrease of qualification for the term of more than two months, they can receive a sick leave or receive an invalidism group due to their occupation disease.

Preventive measures. Preventive measures envision the following: sealing-in of the equipment, utilization of individual means of protection of respiratory organs, as well as conduct of preliminary and periodical medical examination of workers.

Chronic toxic diseases of bronchopulmonary apparatus develop among workers of chemical enterprises and are the result of long-term impact regarding small concentrations of toxic matters of irritating action. They can also be the result if one (or several) acute affection of bronchopulmonary apparatus.

Chronic toxic diseases are more often had in such forms as affection of upper respiratory tracts (chronic rhinitis, pharyngitis, laryngitis and tracheitis), as well as chronic toxic bronchitis.

Affection of upper respiratory tracts (nose, throat and larynx) has, at first, catarrhal and then atrophic character. A patient has the feeling of dryness in the nose; heartburn, disorder of nose breathing, scratchy throat, sometimes nose bleeding as well as coarse voice. After the examination, hyperemia of mucous tunic is observed, as well as its thickening. For the action of chromium and fluorine (especially, in high concentrations), it is characteristic to have necrotic tonsillitis affection in the area of nose septum. At dynamical examination, it is possible to find some pattern in the development of deep erosion, which is finished with the formation of connective tissue scar or the perforation of the septum.

Chronic toxic bronchitis is a diffusive affection of the bronchial tree, which is characterized by the recurrent and progressing development of the disease.

Pathogenesis. In case if the organism of a worker is impact by irritating matters, hyperplasia of cup-shaped cells of bronchial glands with the hypersecretion of mucus and the change of their properties. Secretory, cleaning and protection functions of the mucous tunic of bronchi are affected, what assists fast to the development of inflammatory process. Chronic toxic-infectious inflammation, metaplasia and atrophy of epithelium are formed. They are manifested with swelling, collapse of small bronchi, and scar changes, which finally form broncho-obstructive syndrome.

Pathologic and anatomic patter. When chronic diseases of toxic and chemical ethiology develop (e. g. chronic toxic bronchitis), peribronchial tissue is affected. Interstitial tissue can be also affected with

further development of pneumosclerotic secondary bronchitis. The result of the action of toxic matters on the alveolar septums is the development of emphysema.

Morphological peculiarities of toxic bronchitis are as follows: 1) severe affection of bronchi with the involvement of peribronchial tissue of lungs with vessel and lymphatic systems into the process; 2) joining of chronic bronchitis with obliterating bronchiolitis, which develops very slowly with further access to spot carnification; 3) stable progressing pneumonia is characterized by a marked proliferative process in alveolar septums and its development next to brochiosclerosis of the diffusive pneumocyrosis of lungs.

The result of toxic bronchitis and interstitial pneumonia is the diffusive pneumosclerosis with its unchangeable components, like lung emphysema, pneumosclerosis, bronchiectasis and appearance of chronic pneumonia and bronchiectatic disease.

Clinic. There are three phases of chronic toxic bronchitis: mild, mean and severe.

Mild phase. Patients suffer from dry coughing, sometimes with small amount of purulent phlegm or mucopurulent sputum character. In lungs, some capitations can be heard on the background of coarse breathing.

Acute conditions are rare in this stage. Usually they appear under the condition of unfavorable microclimatic factors or joining of an intercurrent infection.

Deviations on the radiological picture at this phase are not found. When researching functions of external respiration, some small deviations on fast indications are found. Mean stage. It is characterized by the intensification of coughing; amount of phlegm discharge increases (it is coming closer to purulent one); dyspnea appears during physical exercises. Fit-like coughing can develop. Above lungs, percussion sound gets box sound on the background of coarse (sometimes, weak) respiration; scattered dry and moist crepitations can be observed. Acute conditions are more frequent and longer. On the radiological picture, intensification and deformation of lung pattern can be observed, mostly in lower portions; lung fields become more transparent. When researching functions of external breathing are determined by its stable disorder on obstructive type.

Severe stage. As a rule, it is a complicated bronchitis. More often, marked bronchospasmodic syndrome with the transfer to the secondary bronchial asthma or with appearance of bronchiectasis take place.

Manifestations of bronchospasmodic syndrome (complicated breathing with fits of suffocating coughing) remind fits f bronchial asthma, but eosinophilia and the change of phlegm. However, there are cases of the progressing of infection - depending bronchial asthma with progressing clinical pattern, presence of corresponding changes in the phlegm and blood, development in further progressing respiratory insufficiency.

In some cases, clinical pattern reminds the progress of bronchoectatic disease (patients discharge from 300 to 50 ml of purulent sputum, and frequent hemoptysis is observed).

Coughing becomes permanent, much amount of purulent sputum, often with blood and unpleasant odor, are observed. Obstructed respiration, which transits into fits of dyspnea are observed. Patients get cyanosis, frequent respiration; nails get the form of clock glass, and phalanx of fingers look like drum sticks.

During auscultative examination, in lungs, it is possible to hear scattered dry and moist rales, mostly in lower portions of lungs. From the side of cardiovascular system, there is tachycardia; tones of the heart are dull; the accent of III tone can be heard above the lung stem; stagnant phenomena can develop, fist in small and then in the large blood circulation circle.

In the blood, there is compensatory polycythemia (increased amount of hemoglobin and erythrocytes).

During radiological examination, it is possible to note deforming diffusive pneumosclerosis and lung emphysema. Independently from the overbalance of some clinical form, the severity of the state of such

patients is conditioned by the increasing decompensation of the function of external respiration and decompensation of cor pulmonale.

Treatment. To treat chronic affection of upper respiratory tracts, alkaline and oil solution inhalations are used; if there is erosion - ulcer defects of mucous tunic, it is recommended to use synthomycin emulsion or the solution of retinal. The treatment of chronic toxic bronchitis envisages means, which dissolve sputum and facilitate its discharge: inhalation with proteolytic ferments (trypsin and pancreatin), mucolytic and expectoration medicinal drugs. Significant place in the treatment of is given to drugs, which renew the bronchial permeability. First of all, these are sympathomimetic agents (asthmopent and salbutamol), derivatives of theophyllin (euphyllin). The effect of these medicinal drugs is increased by antihistamines, which together with the elimination of spasms of smooth muscles of the bronchi show anti-swelling action.

Significant place in the treatment of chronic toxic bronchitis is played by oxygenerotherapy, curing respiratory gymnastics, massage of the chest, and physiotherapeutic procedures (inductotherapy, electropharesis of Novocain or Calcium chloride).

In case of infection development, antibacterial means are recommended (antibiotics and sulfanamides); with cardiac decompensation - cardiac glycosidea (coglucon and strofantin), diuretics (furocemid, triampur and on-potassium-sparing diuretics).

Verification of the ability to work. In case of chronic affection of upper respiratory tracts, patients are able to work according to their occupation under condition of dispensary observation and normalization of work conditions. Presence of ulcerous-inflammatory processes is the condition to provide a medical leave (temporary work).

Patients with mild and mean phases of chronic toxic bronchitis need rational work beyond the area with dust action, matters of irritating action and physical overstrain. In case of the impossibility to get such a job and the decrease of qualification, they are sent to the Verification Commission to get the invalidism group.

Patients with complicated forms of bronchitis are unable to work as a rule; and sometimes need external help (II or I group of invalidism).

Preventive measures. Preventive measures are in the implementation of progressive technologies into the production process (sealing-in of the equipment, its further mechanization, effective ventilation and keeping to safety rules), as well as utilization of individual protection means and medical examination of workers.

Theme №11. OCCUPATIONAL DISEASES WITH PREDOMINANT AFFECTION OF HEPATOBILIARY SYSTEM (TOXIC HEPATITES) AND KIDNEYS

OCCUPATIONAL DISEASES WITH PREDOMINANT AFFECTION OF HEPATOBILIARY SYSTEM (TOXIC HEPATITES)

Wide development of chemical industry, organic synthesis, and utilization of pesticides caused the increase of the number of cases of hepatitis. This group of diseases includes affection of liver, caused by chemical agents, which are used at the production. The most systemized classifications the classification of hepatoxic matters with the consideration of chemical structure:

- chlorinated hydrocarbon;
- chlorinated naphthalene and biphenyls;
- benzol, its homologues and derivatives;
- some metals and metalloids.

1. Chlorinated hydrocarbon (methyl chloride, chloroform, carbon tetrachloride, ethyl chloride, dichloretan, and tetrachloritan) is widely used in machine engineering, airplane and vehicle manufacturing, as well as in production of shores, to clean clothes, during worming, disinfection and disinfection.

2. Chlorinated naphthalene and biphenyls are hard wax-like mass of yellow or brown coloration. Under the name of "galowax", and "savol" to cover electric wires, filling in condensators; they can be used as replacements of resins, wax, and caoutchouc.

3. Due to the development of industrial chemistry, benzol, its homologues and derivatives (nitrobenzene, toluol and aniline) are wider used to produce aromatic compounds, organic dues and explosive materials.

4. Metals and metalloids (lead, mercury, gold, manganese and phosphorus) are gradually replaced by less harmful compounds in the industry.

Liver completes an antitoxic function, independently of the ways of permeating and the place of the action of poison in the organism. At the same time, different on their construction, matters can cause affection of liver. Some of them, the so-called hepatobiliary poison, have particular similarity with tissues of the liver and cause specific hepatobiliary effect, getting to the organism in small dosages. Selection of a group of hepatobiliary poisons, without having bad tropism to the liver tissues, but still damage the latter, without affecting some functions.

Pathogenesis. Due to the action of the majority of hepatoxic matters, direct affection of parenchyma and disturbance of exchange ferment processes in its tissues take place. Depending on the chemical nature and the dosage of poisoning change the mechanism of its action. Necrosis of hepatocytes at poisoning with tetrachloride carbon is the result of the disturbance of fermentative systems of endoplasm reticulum. In other cases, degenerated changes of hepatocytes under the impact of tetrachloride carbon and allyl alcohol are connected with the intensification of re-oxidation of unsaturated fatty acids of membrane lipids. In case of the action of heavy metals, pathogenesis of the damage is caused by the blockade of sulfhydril of ferment groups.

Affections and other ferment systems of the liver - cholinesterase (mercury) and phosphatase (fluorine) are possible. Particular attention ispaid to the disorder of intralobar changes of corresponding areas (chlorinated hydrocarbons). At the action of trinitrotoluene in the organism, deficit of cystine due to its connection with nitrogroups takes place.

Besides, main (hepatoxic) actions, allergic affection of the liver (ursol) can take place. Some poisons cause both direct toxic and sensibilizing action (gold salts, some compounds of arsenic, and sulfanilamide substances).

Among syndromes, which characterize the affection of the liver, conditioned with the impact of occupational indicators, the syndrome of cytolisis and cholestasis prevail (excretory- biliary). The syndrome of cytolisis develops in the result of functional inferiority of hepatocytes at the change of permeability of their membranes with further hyperferremia. The latter, which develops with the increase of the activity of ferments, is characteristic for acute intoxication with hepatotropic poisons. Hyperbilirubinemia with increased content of fraction of free bilirubin in the blood serum at chronic toxic hepatitis can be explained by fermentative disorders - decrease of the activity of glucuronid - transferase system of hepatocytes.

Under the impact of low concentrations of chemical matters, which have sensibilizing action, for example, formaldehyde, toxic and allergic reaction of the liver is manifested through signs of cholestasis (increase of the activity of alkaline phosphatase of the blood serum) and initial indicators of the affection of intermediate tissue (increase of thymol indicator).

It is necessary to accentuate that preliminary affections of the liver with alcohol and viral infection increase the sensibility to hepatotropic poisons. Toxic effect is intensified at the bacterial infection and deficit of proteins in the meals.

Pathologic and anatomic pattern. Toxic affection is manifested with various morphologic changes in the liver: massive and submassive necrosis of parenchyma, fatty degeneration or ballooning degeneration. The term "hepatitis" in the majority of cases does not correspond to the morphological pattern of acute toxic damages; some researchers consider changes in connective tissue at toxic affection as a secondary inflammatory reaction onto degenerated affection of cells of the liver, and it is possible to indicate it as "reactive hepatitis". In case of appearance of acute poisoning with matters of hepatotropic action, the picture of zonal affection of the liver can be observed (fatty degeneration, necrosis of cells in the central part of the lobe), what as a rule is over with complete renewal of the normal structure of the liver. At acute intoxications, massive liver necrosis can develop, which is sometimes transformed into a large-nodule postnecrotic cirrhosis. For patients with chronic intoxication, the most characteristic morphological indication of the affection of the liver is some stage of fatty degeneration - from the diffusive to the nuclear ones, which is often united with albuminous degeneration.

Clinics. According to the clinical pattern, the following types of toxic hepatitis can be distinguished: acute and chronic ones.

Acute occupational hepatitis is one of the clinical syndromes of the acute poisoning at short-term impact of the high concentration of toxic matters. They appear during accidents, disorders of safety techniques and rules of keeping of toxic matters. These matters permeate through lungs.

The clinic of toxic hepatitis reminds viral hepatitis, however it has no preicteric period. Acute affection develops on the second - fifth day after the impact of toxic matters in comparison with high concentrations. The disease is manifested through the increase of the liver size, pain at palpation and increasing jaundice. Increase of the activity of intercellular ferments in the blood serum can be observed (alanine aminotransferase, aspartate aminotransferase, fructosemonophosphataldolase, lactate dehydrogenase, hyperbilirubinemia, urobilinuria, and bile ferments). Besides, hypoalbuminemia, decrease of P- lipoprotein and phospholipins of blood. The system, which are responsible for blood coagulation and anti-coagulation of the blood, are affected; hypocoagulation takes place; fibrinolytic activity increases; the content of heparin increases and hemorrhagic syndrome develops.

Patients with mild and mean forms of the disease have blurred passing jaundice, and with severe forms, it is intensive, with hemorrhagic syndrome. Severe forms of toxic hepatitis can cause acute liver decompensation. Significant meaning is possessed by the accompanying affection of kidneys.

If to follow the development of acute poisoning, then sings of the affection of the liver have cyclic character.

The first period is characterized by symptoms of the affection of the nervous system. Headache, dizziness, nausea, vomiting, and disorder of coordination take place. In severe cases, a patient can lose consciousness, with the development of the narcotic coma with severe affection of the respiratory and vasomotor centers. Main danger of this period is in the possibility of the development of coma and complications, conditioned by narcosis (aspiration of vomiting masses and asphyxia due to sunken tongue).

The second period of intoxication starts with the end of the first day, when symptoms appear, which prove the liver affection. Phenomena of acute toxic hepatitis depend on the stage of the expression of inflammatory and necrotic processes in the liver tissues. In case of light poisoning, jaundice can be absent. In more severe cases, there is pain in the right hypochondrium, and jaundice develops gradually. The characteristic fever, caused by resorption of necrotically changed liver proteins. On the second - fourth day of the intoxication, signs of functional decompensation of liver take place: phenomena of hemorrhagic diathesis increase (shadows under the eyes and epitasis); jaundice increases; and sleep is in disorder. Liver increases. Biochemical research show the decrease of prothrombin index, cholesterol, sudden increase of the level of the activity of ferments of the "necrosis" (aminotransferase and aldolase), the amount of bilirubin in blood increases, mostly by its direct fraction.

In some cases, toxic hepatitis can cause acute atrophy of liver. Liver decompensation is shown through inhibition, change of the sleep rhythm, tremor, increase of the content of ammonia in blood and significant hemorrhagic syndrome.

Acute intoxication can be ended with the development of hepatic coma. Significant meaning is possessed by accompanying toxic hepatitis of kidney affection. When researching the urine, it is possible to define protein and formed elements. However, only on the 5th to 7th day of the poisoning, more marked symptoms of acute kidney decompensation start showing.

The third period of intoxication is characterized by the expressed decompensation of the kidney function, in the base of which there is acute toxic necroso-nephrosis. Oliguresis is replaced by anuria. In the blood, the concentration of nitrogen scora increases (residual nitrogen, urea and creatinine).

Thus, the peculiarity of the acute poisoning with poisons of hepatotropic action is recurrence of the clinical pattern, conditioned by marked disorder of the functional state of the liver and kidneys, until the development of sings of their decompensation.

Together with general clinical manifestations, which are characteristic to all the toxic hepatitis, some patients have specific affections, characteristic toe some poisons. In case of massive impact, chlorinated hydrocarbon cause narcotic action. Neurological disorders prevail in the form of dizziness, ataxy, dormancy, psychomotor agitation, and in more seldom cases, state of coma. It is also joined by toxic hepato-and nephropathy. At acute poisoning with tetrachloride carbon or chloride naphthalene, jaundice appears on the first or second day after poisoning, and is accompanied by acute increase of aminotransferases of the serum at the unchangeable proteingram with severe affection of kidneys. The lethal end takes place within the first two weeks of the disease.

For intoxication with trinitrotoluene, methemoglobinia and appearance of Geins body in blood are characteristic. Liver affection at theaction of aniline develops on the background of the nervous system and blood (due to formation of methemoglobin), which are characteristic for the later.

In the majority of cases of toxic hepatitis after the termination of the contact with hepatotropic poisons, there is dyskinesia of the gall bladder and biliary tracts. After severe poisoning with massive necroses of parenchyma, macronodular cyrosis of the liver can develop.

In correspondence to the current existing classification of chronic diseases of the liver, toxic hepatitis with its clinical and morphological indications and the progressing, it is closest to chronic persisting hepatitis and of the so-called "non-specific" reactive hepatitis.

The disease appears in case of prolonged impact of subtoxic dosages of hepatotropic matters. The affection of workers' livers, who are in contact with small concentrations of toxic matters, is manifested in the form of hepatobiliary syndrome and functional decompensation of biliary cells and are characterized by dyspeptic and pain phenomena.

Patients complain to have bitterness in their mouth, decrease of appetites, unstable emptying, dull pain in the right hypochondrium, which is intensified after spicy or fat food. During the time of objective examination, often weak icteritiousness of scleras, more seldom, the jaundice of skin coverlet, increase of liver sizes, pain when palpating, positive symptoms, and irritability of gall bladder (Orthner, Merphy and phrenicus-symptoms). "Vascular stars" and "palmar erythema", and increase of spleen can be rarely met.

Pain syndrome can be explained by dyskinesia of bile-excreting tracts. It takes place even in the initial stages of the impact of toxic matters. On the background of dyskinesia of the gall bladder, secondary infection can develop, and signs of cholesterol can be observed.

Chronic toxic hepatitis develops for a long time. Its progress is non-malignant, without the inclination to progressing. Mild forms of the disease have the tendency to reverse development. Stabilization of the process with patients both with mild forms of the disease and with forms that are more marked is possible. Severe progressing of toxic hepatitis can be rarely observed. Usually, it can be met in case of the

affection of the liver, which has mixed character; for example, on the background of the past viral hepatitis or alcohol abuse.

More severe progressing of toxic hepatitis can be observed among workers with long period of work, among elderly people, and in case of the presence of dyskinetic syndrome.

The clinical picture of chronic affections of the liver, in case of the action of various poisons, can have its peculiar progressing. For instance, toxic affection of the liver among patients with chronic intoxication with benzol takes place on the background of the affection of the haematogenicsystem; with intoxication with trinitrotoluene - the development of the professional cataract, functional disorders of the nervous system, and unstable anemia; with chronic intoxication with lead - changes in the blood and nervous system. Summing up the given above, it is possible to make the conclusion that in the clinical pattern of intoxication, liver affection can be a prevailing syndrome (poisoning with dichlorethane, tetrachlorated carbon, or trinitrotoluene). At some intoxication, the liver affection takes place in parallel with the development of other symptoms or fades into the background (lead, benzol or fluorine).

Thus, the main peculiarity of toxic and chemical affection of the liver is the following:

1. Toxic hepatitis, no matter how little it is marked, it never develops in isolation, but always on the background of general phenomena of acute or chronic intoxication. At acute intoxications, this pattern is more marked.

2. Acute toxic hepatitis appears fast without syndromes on the background of general toxic action of the poison. Jaundice does not always take place. Diagnostic meaning is possessed also by the simultaneous affection of other parenchymatous organs, mostly kidneys, in particular in case of peroral intake of the poison (dichlorethane and tetrachlorated carbon).

3. Chronic toxic hepatitis is characterized by the lack of symptoms. Its progressing is rather favorable with long remissions. Functional disorders of the liver and dyskinesia of bile-excreting tracts can be saved for a long period of time. Cyrosis can be very rarely observed.

At chronic intoxications, fatty hepatitis can be formed; chronic persisting hepatitis takes place, and in some cases, latent forming of liver cyrosis is possible.

Treatment. At acute poisoning with matters of hepatropic action, first, it is necessary to terminate its intake into the organism. Further actions should be targeted at the neutralization of the poison and its excretion from the body. It is recommended to conduct gastric lavage with 10 to 15 liters of water with further administering of 100 to 200 ml of liquid paraffin or 30 to 50 g of salt laxative in case of peroral administering of the poison; even in case of minimal signs of intoxication, artificial diuresis with the utilization of diuretic means (urea, mannitol, or furosemide), as well as peritoneal dialysis and hemodialysis.

Particular place is taken by antidotal therapy. At acute poisoning with heavy metals (lead or mercury), it is recommended to administer sodium thiosulfate intravenously; and unithiol - intramuscularly or hypodermically. At acute poisoning with iron salts, it is recommended to administer deferral: internally, 5 to 10 g of the preparation dissolved indrinking water and intramuscularly - 1 to 2 g of the preparation every 3 to 12 hours.

Pathogenic therapy includes lipotropic preparations - 30 ml of 20 % solution of chlorine chloride together with 600 ml of 5 % solution of glucose is administered intravenously in drops; vitamin of the group B; antioxidants - vitamin C intramuscularly 1 ml 4 to 6 times a day; antiprotease preparations - trasilol and contrical 500 000 units a day intravenously by drops in 5 % solution of glucose or isotonic solution of sodium chloride. It is recommended to administer cocarboxylases intramuscularly 150 to 200mg a day; glutamine acid (up to 8 g a day) intramuscularly and antibiotics. In case of indications, symptom therapy is recommended (tranquillizers, and cardio substances). In cases of severe poisoning, it is recommended to administer intravenously glucocorticosteroid hormones.

Treatment of chronic toxic affection of the liver includes curing methods, which are conducted in case of presence if some intoxication, as well as the treatment of the liver affection itself.

At the mild form of the disease, curing diet, vitamin therapy (especially, vitamins of the group B), cholagogic means, and duodenal intubations are recommended. Antibiotics are recommended in the cases, when toxic hepatitis is complicated with inflammatory processes in biliary tracts.

With more marked forms and acute chronic toxic hepatitis, it is recommended to undergo hospital treatment. In case of the presence of toxic hepatitis, which develops for a long time, it is recommended to prescribe sirepar or other preparations of the cattle liver.

Treatment with steroid hormones and cytostatics are prescribed in case of sings of high activity of the process in the liver, what is rarely met in the clinics of occupational hepatitis.

Sanatoria and health resort treatment is recommended at moderate disorders of the functional state of the liver and the presence of dyskinesia of the biliary system, beyond the phase of the acute stage. Balneology resort centers are recommended, like Berizivsky Mineral Waters and Truskavets.

Verification of the ability to work. At acute intoxications with hepatotropic poisons, the ability to work is determined by the severity of intoxication and the possibility of reversibility of the pathological process. Mild stage of the acute intoxication envisages the possibility to return to work under the condition of dynamic follow-up and keeping to sanitary standards.

In case of severe diseases, presence of jaundice, high hyperfermentis, decrease of functional tests, and increase of the liver, the patient should undergo treatment in hospital conditions with the following sanatoria andhealth resort treatment. In the future, this person should be transferred to the job beyond the contact with toxic matters for the period of two months, with the payment in compliance with the occupational list of inability to work.

In case of stable residual phenomena of intoxication or in case of the transfer of the disease into a chronic form, the patient should be rationally employed beyond the contact with toxic matters.

If the patient has toxic hepatitis, it is recommended to transfer him/her to work beyond the contact with toxic matters (rarely temporary -up to two months, in case of favorable outcome). In case of stable disorders of the functions of the liver, the patient should be transferred to the work beyond the action of toxic matters for a long period of time with further re-qualification and provision of the invalidism group for the period of re-qualification on the occupational disease.

Preventive measures. Prevention of occupational hepatitis is in keeping to safety rules, correct keeping of toxic matters, and general and individual hygiene. An important place is taken by a correct occupational selection of workers at the manufacturing, where contact with hepatotropic poisons takes place, as well as full-value meals with sufficient quantity of protein, and vitamins; and exclusion of alcohol abuse. It is important to have periodical medical examinations of people, who work in contact with matters of hepatotropic actions, with the purpose to find early and most reversible forms of the disease.

OCCUPATIONAL DISEASES WITH MAJOR AFFECTION OF KIDNEYS AND EXCRETORY TRACTS

Etiology and pathogenesis. Pathology of kidneys and excretory tracts is taken by relatively small spread among the expanded group of occupational diseases.

The contact with the kidney parenchyma and excretory tracts with toxic matters, accumulation of these matters and their transformation in kidney structures is determined by the possibility of the affection of kidneys and excretory ways. The character of the affection of excretory system depends on the chemical composition of the compounds, concentration, tracts of their permeating to the organism, health state, and especially the health state of kidneys. Depending on the localization of the affection and the character of the pathological processes, chemical compounds can be divided into two groups.

The first one includes those compounds, which mostly affect parenchyma of kidneys and cause the so-called toxic nephropathy. Toxic nephropathy (toxic nephritis) means functional changes or structural changes in kidneys, caused by the impact of exogenous chemical products and their metabolites.

Development of toxic nephropathy is caused bychemical matters, used in national economy: metals and their salts (lead, and mercury), nitrogenated compounds (aniline, nitrobenzene and ammonia) and their halogen derivatives (carbon tetrachloride and hexachloroethane), glycols (antifreeze), ethers (dixan and ethyl acrylate), as well as carbon oxide, acids and other matters.

Occupational diseases of kidneys can be observed among workers, engaged in the production of synthetic caoutchouc, polymer materials, and chlororganic poisonous chemicals. Utilization of the latter in the agriculture caused frequency of the affection of kidneys among the population as well.

It has been stated that the affection of kidneys often appears if the concentration of dust and vapors of nephrotoxic matters in the air of o production premises is higher than the permitted norm. Permeability of poisons into the organism is activated in the process of production activity, especially under conditions of increased temperature of the environment. Frequency and intensiveness of the affection grows in compliance with the increase of the work period in contact with poisoning chemicals.

Poisons permeate into the organism mostly through the digestive apparatus and respiratory apparatus, though other ways are possible as well. Thus, nickel and cobalt permeates through the skin in toxic amounts, and are accumulated in the liver and kidneys in the form of crystals.

Decisive meaning is possessed by the direct impact of poisonous chemicals onto the kidney parenchyma, however the functional capability of kidneys can be also affected by the changes of neuroendocrine regulation of the organs and due to vasomotor disorders.

Disorders of kidney hemodynamics, decrease of the kidney blood flow on the background of the disorder of the general blood flow in response to the chemical trauma is one of the pathogenic mechanisms of the toxic affection of kidneys.

There are cases, when toxic effect is provided by not only chemical matters, which permeated into the organism, and their metabolites (e. g. dock acid when getting poisoned with glycols or products of interaction with other organs and tissues, in particular hemoglobin when affected with hemolytic poisons).

Obstruction of kidney channels takes place with products of hemoglobin degradation (poisoning with arsenious hydrogen, essence of vinegar or copper sulfate), myoglobine, crystals of oxalates (poisoning with glycol or dock acid). Immune mechanism of the affection of kidneys (toxic-allergic) is possible, when acute renal insufficiency develops in case of permeation of small quantity or little toxic chemical compounds. Increased individual sensitivity to the chemical matter is significant.

In case of toxic nephropathy, there are changes of the activity of a number of ferments in the blood and urine, processes of re-amination in themitochondrion of livers and kidneys; the content of amino acids in biologic environments, what proves the disorder of intercellular processes, and increase of the resistance of cellular membranes. There are the data regarding the role of the hyperaminoaciduria, caused by toxic irritation of mucous tunic of the urinary bladder and can lead to the appearing of hemorrhagic cystitis, non-malignant (papilloma) and malignant (cancer) tumors of the urinary bladder. These are mostly aromatic amino compounds (benzidine, dianisidine as well as a- and P-naphthylamine) used in the production of dyes.

Clinic. In case of permeation into of the significant quantity of nephrotoxin into the organism during a short period of time, acute renal insufficiency can be observed, progressing of which has four stages: initial (shock); oligo and anury one; renewal of diuresis or poliuric; and recovery.

Clinical signs of the initial stage are usually symptoms of the main disease, and in particular hemodynamic disorders in parenchymal organs, and in particular in kidneys. The definitive sigh, which should be particularly considered, is a circulatory collapse, which is sometimes unnoticed though their short term of duration.

The decrease of the arterial pressure is accompanied by the decrease of diuresis. Symptoms of the initial stage are often unnoticed due to the severity of the main disease and the shock.

The duration of the initial stage - from several hours to one to three days.

In the second (oligo-anury) period of the acute renal insufficiency, it is possible to notice sudden decrease of complete termination of urination. Often, the disease develops unnoticed. After the normalization of hemodynamic disorders, patients feel a little better, and a period of imaginary well-being takes place, which lasts fro 3 to 5 days. However, at this moment, less and less urine is produced, and its relative thickness decreases (up to 1007 to 1010), at the same time the content of urea, creatine, nitrogen and chlorides in the daily amount of the urine. In cases of hemolisis or moilisis, hemin pigment can be found in the urine. Many erythrocytes and leukocytes, epithelial cells and bacteria can be observed.

On the 5th to 7th day, patients start feeling much worse. They become drowsy, adynamic, lose appetite; patients suffer from vomiting and thirst. Depending on the "background" of the acute renal insufficiency, the body temperature can be normal or increased. Due to the decrease of the resistance of the body and in case of purulo-septic complications, the temperature increases insignificantly, however, it can be subfebrile among some patients without the presence of infectious complications.

"Uraemic" intoxication and changes in water-electrolytic homeostasis often cause the affection of the consciousness; Patients do not orient in space and time. In addition, sometimes, there are "convulsivecrises", which remind epilepsy. When dehydration takes place, asthenia and drowse come along with anxiety, acute psychosis and hallucinations. In very severe cases, coma can be observed.

In case of prolongued anuria, skin becomes dry and then peels off. In addition, often, rash takes place, which reminds scarlatinous or measles rash. At intravascular hemolisis, skin and sclera are icteric. Through the disorder of coagulating properties of the blood, hemodermic hemorrhages, and in particular, in the injection areas and on conjunctive take place. A tongue is dry and is covered with white or brown incrustation. Often, stomatitis can develop. Vomiting with stomach mucus and bile can take place.

During the initial period, oligo- and anuria stages, constipation can be observed, which is replaced with diarrhea in case of the growth of azotemia. When palpating the stomach, the abdomen is painful. Coarse breathing can be heard in lungs, and in severe cases, there are stagnant crepitations can be heard in lower portions. At hyperhydration, which takes place in the result of irrational introduction of liquid, lung swelling takes place - aquatic lung? Effusion into the pleural cavity can be observed. At the significant acidosis, dyspnea increases, and patients who are in severe state have breathing after the type of Kusmaul.

Cardiologic affection can be shown through miocarditis (dull cardiac tones, systolic noise, size increase and pain in the heart, as well as changes on the EKG.

The most serious changes from the side of the heart can be observed in oligo- and anury stage in the result of changes in the content of potassium in blood. At hypercaliemia, bradycardia, arrhythmia, dyspnea, and vascular insufficiency develop; and changes on the ECG can be determined.

Changes in the blood can be characterized by the marked hypochromic anemia, the decrease of the number of erythrocytes and the decrease of the content of hemoglobin. Anemia is well marked in the beginning of the acute renal insufficiency.

During the period of oligo- and anuria, concentration of urea and creatinine has sudden increase in the blood plasma. Hypoproteinemia with the decreased albumno-globuline ratio is characteristic. Hypoalbunemia is joined with the increase of the percentage of a- and y-globulines.

Acid-base balance is affected. Intensified catabolism causes the accumulation of acid products in tissues and the development of metabolic acidosis. Metabolic acidosis can be changed by respiratory alkalosis via intensified ventilation of lungs and excretion of a large number of bicarbonate ions from the organism. This assists the support of the pH level of plasma within normal oscillations, though alkaline reserve is decreased. The disorder of the water exchange is expressed in hyperhydration or dehydration. Outer cellular and inner-cellular dehydration can be distinguished.

Clinical pattern of the intercellular dehydration consists of symptoms of the brain swelling (vomiting, headache, state of coma, and affection of breathing rhythm), symptoms of intercellular (swelling)

and intervascular hyperhydration (hypervolemia, increase of the arterial pressure, left ventricle incompetence with lung swelling).

Extracellular dehydration can be clinically manifested with hypervolemia and skin dryness. Arterial blood pressure is decreased. Pulse is weak. Collapse can often have place.

The duration of oligo- and anuria stages is from two to three weeks.

The third stage (renewal of diuresis) can be characterized by the increase of the amount of the urine excretion. Together with the increase of diuresis, patients feel better. They become less drowsy; their concernedness is clearer, headache and muscle ache lessen, lung-swelling decreases as well. The skin becomes dry. Appetite improves. Together with the increase of diuresis, the degree of azotemia increases; and concentration property of kidneys increases.

The fourth stage (recovery) can last for 3 - 6 months to one-two years. The state of patients, who underwent acute renal insufficiency, improves gradually. The most stable symptoms are asthenia, anemia and decrease of the concentration of renal properties. Complete renewal of the functional state of kidneys will be in one or two years.

In specific cases, transfer of the acute renal insufficiency into the chronic one can take place. Changes in kidneys, caused by the impact of chemical matters, which create toxic metabolites, that have impact onto kidneys, can be considered as toxic nephropathy. Marked forms of nephropathy can develop in case of severe acute poisons with chemical matters (chlorinated hydrocarbon, organic compounds of mercury, chlor-and phosphoro-organic pesticides, etc) and are accompanied by various degrees of the expression of the acute renal insufficiency.

C h r o n i c p o i s o n i n g with chemical nephrotoxic matters is manifested with changes on the side of the central and peripheral nervous system and organs of blood formation. The first symptoms usually take place after the contact with the poison for three or more years. At first, after three to five years of work, functional activity of kidneys can be intensified: renal blood stream and plasmostream increase, glomerular filtration and clearance of urine increase. For the following 6 to 10years, some normalization of functional properties of kidneys takes place.

If the work period is over 10 year, the activity of compensatory mechanisms with gradual and non-simultaneous inhibition of the listed functions, increase of the filtration fraction, decrease of the ratio ofpurification of the urine, oliguria and nocturia weaken. At first, relative thickness of the urine increases a little, and then it decreases. In urine, some amount of protein, erythrocytes, hyaline cylinders, and cells of renal epithelium can be observed. The activity of cholinesterase decreases. Thus, it is possible to define three phases of chronic toxic nephropathy; the increase of the kidney activity, adaptation and the decrease of the functional property of kidneys.

At chronic intoxication with various chemical matters, the toxic nephropathy is often determining syndrome of intoxication; usually, functional disorders of kidneys is determined on the background of the expanded clinical pattern of intoxication. Only at intoxications with cadmium and P-naphthol, through the affection of kidneys and early forms of these intoxications are diagnosed based in the indicators of the functional state of kidneys.

Nephrotoxic action of heavy metals progresses with relatively mild clinical symptoms. Significant interest is caused by the affection of kidneys in the result of the lead intoxication. In cases of heavy forms of chronic intoxication with lead, changes in renal vessels, hemorrhage, necrosis of epithelium, and fibrosis changes occur. In case of chronic intoxication with lead, passing proteinuriais caused by irritating action of the lead onto the channel epithelium and reverse functional disorder in the area of secernent epithelium.

For saturnism, presence of spastic state of vessels of kidneys, alternative changes of channel epithelium cells with their intranuclear destruction are characteristic to be present. Under the impact of lead, oscillation of the concentration function of kidneys can be observed. Though nowadays, the very lead etiology of the chronic nephritis is not supported by the majority of researchers, but still in those cases,

when lead intoxication is headed by the kidney disease, intoxication with lead can significantly worsen the non-specific inflammatory process in kidneys.

Occupational diseases of the urinary system include tumors of the urine bladder. It has been proven that the cancerogenic action is possessed by P-naphthylamine, benzidine B, adiacetylbenzine and some of their derivatives. These matters permeate into the organism of a human being through the skin, respiratory organs and esophagus.

The beginning of the disease can be characterized by the symptoms of chronic irritation of the mucous tunic of the urinary bladder. Often, patients do not have any complaints for a long time, and except some non-constant more frequent urination mostly during the daytime. Not much deviation can be found in the urine. Further, urination is more often accompanied by sharp pain, some complication and non-constant hematuria. Later, chronic irritation of the mucous tunic of the urinary bladder, what takes place in the result of the discharge of aromatic amines, is manifested by the disorder of urination on the background of which hemorrhagic cysts can develop often with painful impulses to urinate, as well as marked heamturia. At this time, cystoscopy can be expressed mostly in the area of a triangle and a neck of urinary bladder. Sometimes, they are spread onto other areas of the mucous tunic. In complicated conditions, there is a threat of epithelium peeling off.

Sometimes, the appearance of complaints on dysuric phenomena corresponds with the development of papilloma or malignant tumors of the urinary bladder. A tumor can develop in any section of the urinary bladder, but mostly it locates in the area of he triangle.

Affection of kidneys, renal pelvis, urinary tracts and the hind portion of the urinary channel with a tumor process can be both in separate cases, which are about 4 to 5 % among tumor cases of the urinary bladder. Main symptoms of he tumor of the urinary bladder are profuse heamturia, which often follows microheamturia. Non-malignant papillomas are difficult to be separated from malignant new formations, thus a decisive; meaning in the diagnostics is possessed by the morphological research.

Histologically, malignant new formations of the urinary bladder are often papillary fibroepitheliomas and papillary cancer.

In the diagnostics of occupational diseases of the urinary bladder, cystoscopy is more considered. In the analysis of the urine, microhematuria can be noticed. When analyzing urine on the testing of Kakovsky -Addison, small amount of erythrocytes can be in norm as well.

Macrohematuria usually appears on the background of a profound pathological process in the mucous tunic of the urinary bladder. To find cells of a malignant tumor in the urine is usually very complicated, in spite of the developed special methods of separation of cells from mineral and organic parts of the sedimentation.

Taking into consideration the forecast of occupational diseases of the urinary bladder and urinary tracts, it is necessary to note that hemorrhagic cystitis causes the development of a tumor rather rarely. Regarding the papillomas, they are not usually subject to reverse development and can reduce. Recurrent papillomas are often capable to turn into malignant tumors. The possibility to cure the cancer of a urinary bladder, as well as tumors with other location, is determined by the stage of the disease.

Treatment. In cases of severe poisoning with the development of acute kidney insufficiency, for example, due to intoxications with mercurial salts and arsenious hydrogen, patients are subject to obligatory hospitalization in specialized establishments.

At the first stage of the disease, the treatment and preventive measures of the acute renal insufficiency is reduced to the prescription ofspecific antidotes, eradication of circulatory disorders, exchange blood transfusion at hemolisis as well as eradication of destroyed tissues. At the second stage, therapeutic measures should be directed at the decrease of protein catabolism, to support water-electrolyte and acidic-alkaline state, prevention of cardiovascular decompensation and infections. If it is

impossible to achieve compensation, it is necessary to use methods of extrarenal cleaning - hemodialisis with the utilization of the artificial kidney apparatus or peritoneal dialysis.

At the third stage, it is necessary to keep thorough control of the electrolyte composition of the serum. If necessary, its corrections should be done.

In urinological hospitals, treatment of cysts is also carried out, as well as operative interventions on papillomas or cancer of the urinary bladder.

Lately, much success has been achieved in chemical therapy of malignant new formations of the urinary bladder.

Verification of the ability to work. Workers, who were found changes of chronic cyst or papilloma types in the mucous tunic of the urinary bladder after the prophylactic examinations, are subjects to obligatory transfer to the work, which is not connected with possible impact of toxic matters. At the development of new formations, there is a question regarding operative intervention and transfer to the invalidism group. Issues on rational work position should be solved individually.

Preventive measures. It is necessary to introduce non-stop technological processes, to use hermetic machines, improve automation and remote control. It is necessary to check up that workers use means of individual protection.

An important role in prevention of these diseases is played by the conduct of preventive and periodical examinations of workers.

Theme №12. INTOXICATION, WHICH APPEAR UNDER CONDITIONS OF AGRICULTURE (INTOXICATION WITH PESTICIDES)

Pesticides are chemical matters, which are used in order to destroy live organisms - bacteria, viruses, spores, fungi, insects, rodents, as well as plants, which damage agricultural cultures and animals.

The chemical method to protect plants and animals is still one of the most convenient, cheap and effective ones. It conditions a significant growth of production and utilization of pesticides and a big number of people are in contact with it.

Pesticides, which are active biological compounds, can have a negative impact onto useful insects and animals, and have negative impact onto the health of people and cause poisoning.

The committee of WHO experts considers that the danger with pesticide poisoning endangers mostly workers of enterprises, where these compounds are made, also those who are directly involved into pesticide utilizations, as well as those who stay in premises treated with pesticides. However, significant expansion of the production and scales of pesticide utilization led to contamination of soil and water reserves with them, what caused the contact of almost all people all over the world with them.

As pesticides, a significant number of chemical matters are used, which vary as to their chemical structure, action character, etc. With the purpose of convenience at the production utilization of pesticides, development and implementation of means, directed at the prevention of possible intoxications, various classifications of pesticide preparations are used: production, chemical and hygienic.

In the basis of production classification, there are two indicators: purpose of pesticides and the goal of their utilization. In compliance with this classification, pesticides against various insects called insecticides; against bacteria - bactericides; against fungi - fungicides; against weeds -herbicides, etc. This classification includes such preparations, which are used to destroy leaves of plants - defoliants, and herbal drying -desiccants.

Based on the chemical structure, there are chlororganic compounds (COC), phosphororganic compounds (POC), and mercurious organic compounds (MOC), derivatives of aminoformic acid, etc.

Hygienic classification envisages the division of pesticide preparations according to the degree of their toxicity with the consideration of mean-lethal dosage - LD50 (drastic, high-toxic, meant-toxic and little toxic matters), the degree of volatility, cumulation, durability, etc.

Main ways of pesticides permeating into organism are respiratory organs, gastric system, and skin. According to the clinical progressing, there are acute, sub-acute and chronic intoxications. Acute intoxications develop in the result of permeation of a great number of pesticides into the organism. The development of acute intoxications there are the following periods: latent (from the moment of permeation of poisoning into the organism to the first indications of intoxications); the period of forerunner, for which it is characteristic to have non-specific, one-type reactions of the organism under the influence of many chemical compounds (nausea, vomiting, general weakness, and headache); period of marked intoxication, when together with changes, general for many chemical matters, specific poisons onto organism can take place. For sub-acute intoxications, not such a vivid reaction of the organism onto the action of poison and longer duration of the progressing of pathological process are characteristic, than with acute poisoning. Chronic intoxication develops in case of prolonged permeation of relatively small amount of pesticides into the organism (sometimes for several years).

Clinical pattern of intoxication with pesticides is mostly made of manifestations of their polytrophic action onto the organism, for which it is characteristic to have the development of pathological changes in various organs and systems. Due to the fact, at the development of both acute and chronic intoxications, it is possible to distinguish a number of clinical syndromes.

Thus, at the development of acute intoxication, it is possible to distinguish neurotoxic syndrome, conditioned by the impact of poison onto the nervous system. It is manifested by the headache, dizziness, and various disorders of the consciousness (agitation, dormancy, and coma). Coma can be accompanied by movement reactions, up to the development of clinical and toxic spasms.

At severe poisoning with COC and POC, as a rule, neurotoxic syndrome appears initially, and the clinics of severe acute intoxication can be manifested by the coma in the result of direct toxic action of poisons onto the central nervous system. Coma can develop secondarily as well, in the result of dysfunctional metabolic disorders. Second on the frequency acute syndrome of intoxication with pesticides - gastroenteric syndrome, this develops initially in case of pre-oral permeation of MOC, COC and POC. The development of this syndrome is accompanied by vomiting, nausea, diarrhea and pain in stomach.

Syndrome of respiratory disorders is conditioned by several indicators. COC, POC, MOC and other pesticides inhibit respiratory centers, located in the medulla. When poisoning with POC, derivatives of aminoformic acid is damaged by the intervention of diaphragm and other muscles, which participate in the act of respiration. The syndrome of affection of cardiovascular system appears at the action of many pesticides and is the result of both affection of central sectors of nervous system (COC and POC), and of direct action of poisoning onto the cardiomuscle (POC) and vascular system - walls of vessels (MOC, arsenious pesticides). At this, there are various disorders of rhythm (tachycardia, at the action of MOC and COC; bradycardia at the affection of POC), what is often accompanied by worsening of contractive ability of myocardium with the development of deficiency of blood circulation and swelling of lungs. The arterial pressure can be increased up to 200/140 mm of mercury column at poisoning with POC and the decrease up to the development of collapse.

Hepatorenal syndrome, and in severe cases - acute renal and liver insufficiency, can develop initially at direct action of pesticides onto parenchymatous cells of liver and kidneys. Repeated development of this syndrome is possible in the result of toxic shock, prolonged disorder of hemodynamics, which is accompanied by the fall of the arterial pressure and the volume of circulating blood.

In the clinical pattern of c h r o n i c intoxication with pesticides are mostly observed by changes from the side **of the nervous system**. At the initial stages of intoxication, syndromes can be outlined, which are conditioned by functional disorder of the central nervous system - asthenic and asthenovegetative. In cases of severe intoxication, there is a threat of the development of organic disorders of the cerebrum - toxic encephalopathy.

The impact of Trichlorfon, COC and arsenious pesticides can cause the affection of toxic sensory, and vegetative-sensory polyneuritis. In severe cases of chronic intoxication, mercury and chlororganic pesticides, the development of diffusive affection of the nervous system on the type of encephalomyelopolyradiculoneuritis.

The disorder of the stomach system (chronic gastritis, dyskinesia of bile tracts, cholecystitis, pancreatitis, and colitis) can be often observed at affection with COC, POC, etc.

Many pesticides cause changes in the system of haemopoiesis. Thus, at long impact of COC and POC, anemia and leucopoenia can develop; toxic grain in neutrophils can appear. Metaphos, many carbamates cause anemia, reticulocytosis, and assist to the formation of methemoglobin. Pesticides, which contain copper, can cause hemolytic syndrome, Warfarin-containing ones - hemorrhagic syndrome.

Some pesticides (COC, POC, MOC, and arsenious compounds) have allergic affection of the skin, asthmatic bronchitis, and bronchial asthma, toxic and allergic myocarditis. Intoxication with Organophosphorus Compounds

Organophosphorus compounds (POC) are widely used in the industry and medicine. Many POC are active multifunctional additive to lubricants. They combine properties of washing, anti-corrosive and antiwear additives and are anti-oxidant and depressants. Besides, POC is used in the industry at flotation of ores, polymerization, in production of solvents, etc.

As medicinal means, POC is used to treat glaucomas, myasthenias, atony of intestines, chemiotherapy, tuberculosis and cancer.

POC can be distinguished by high biological activity, and many of them are the strongest of all the known poisons,

As to the chemical structure, POC is ether of the following:

1) phosphoric acid (dibromide and gardon);

2) thiophosphoric acid (Thiophos, metaphos, and methyl mercaptophos);

3) dithionic phosphoric acid (carbophos, phosphamide and amiphos);

4) phosphonic acid (Trichlorfon);

5) amides of pyrophosphoric acid (oct-methyl).

POC can be used as highly effective insecticides, acaricides, defoliants, as well as pesticides to protect crops of cotton-wool, orchid trees, grain and a number of agricultural crops from pests.

The majority of POC have sharp unpleasant smell. They are unstable in the environment, can be easily ruined in case of thermal treatment. There is data on unfavorable impact of POC onto the process of embryo development.

Mostly, poisons with POC are the result of the violation of work rules with this matter during agricultural work or in the process of the production of these compounds. Poisoning can take place via skin, or when inhaling its vapors. POC can be extracted via urine.

Pathogenesis. The mechanism of the action of POC is based on the inhibitive impact of these compounds onto the ferment of cholinesterase. This ferment plays an important role in the process of synaptic transfer of the nervous impulse in cholinergic formations. The action of POC onto cholinesterase causes the formation of stable of phosphorized ferment. Phosphorized cholinesterase (cholinesterase + the residual of POC, which contains phosphorus) hydrolyzes very slowly, without enabling the ferment to catalyze various reactions.

From the pharmacological point of view, all symptoms of poisoning with POC, which can be considered as effects, caused by acetylcholines, and can be divided into three groups: muscarinic-like, nicotine-like and central.

Muscarinic effect can be clinically characterized by ample, feeling of pressure in chest, bronchospasms, increase of bronchial secretion,hypersalvation, loss of taste, nausea, vomiting, pain in stomach, diarrhea, narrowing of irises, and bradycardia.

Nicotine effect is conditioned by irritation of choline receptors. It is manifested through twitching of muscles of eyelids, face, neck, tongue, and the increase of the arterial blood tension.

The central effect can be characterized by a headache, feeling of anxiety, sleep disorder, psyche disorder and spasms.

Pathologic and anatomic pattern. At gistologic research, it is morphologically possible to find phenomena of blurred swelling in the liver and heart muscle, atheroma of liver, and changes in the kidney parenchyma. Early changes are phenomena of vacuolization in saliva and mucous tunic and the decrease of mitochondrion in them.

Clinics. A c u t e p o i s o n i n g. Symptoms of acute poisoning take place suddenly, its progressing can be mild, mean and severe.

At mild form of poisoning with POC there is general weakness, moderate headache, light dizziness, nausea, and excessive salivation. At clinical research, it is possible to note moderate paleness of skin coverlets and the change of the frequency of the heart contraction, dullness of tones, and single type coarseness in lungs. All these phenomena can stay for several hours, maximum - for a day.

In case of mean poisoning, there are marked disorders of the central nervous system. Patients have depression, apathy, headache, speaking disorders, increase of the threshold of analyzers of vision, taste and smell. There can be dystrophy of myocardium, accompanied by the decrease of the arterial blood pressure, some increase and pain in the liver, small proteinuria and microheamturia. The duration of this stage is from several hours to several days.

Severe form of poisoning with POC can be characterized by polysymptom pattern. At first, there is movement disorder, sometimes, psychosis, which is accompanied by vision and auditory hallucinations. There are fibril twitching of eyelids, tongue, face and neck muscles, and with time accompanied by spasms of epileptoforming or clone-tonic character. Then, a patient loses consciousness and deep coma takes place. Specific smell of poisonous chemicals can be smelt from the mouth of a patient. The secretion of salivary and bronchial glands fills in the mouth cavity, and lumen of respiratory tracts. There are signs of respiratory insufficiency: dyspnea, and cyanosis. In addition, the development of pneumonia and lung swelling can be observed.

Well marked dystrophic changes in myocardium can be observed, manifested by the dullness of tones and tachycardia. Stable hypotonia takes place. Together with it, patients have signs of toxic affection of liver and kidneys: increase of sizes ad painfulness, proteinuria and microhematuria. The body temperature at poisoning of POC is normal as a rule, a littlesubfebrile, and only at lasting coma, hypertermia can take place. Severe progressing of poisoning can be often accompanied by hyperleukemia and glycosuria.

Thus, symptoms of acute poising with PC are various, conditioned by the agitation of the autonomous nervous system. As a rule, the first are muscarine-like symptoms, and then nicotine-like and then central ones.

C h r o n i c p o i s o n i n g with POC is possible at long contact with small dosages of preparations.

Pathogenesis of chronicpoisonin is maybe more complicated and less studies than the acute one. It is not always possible to determine the meaning of the cholisterase inhibition.

Clinics. In the clinical patter of chronic poisoning, there are mostly vegetative disorders with clear exaggeration of cholinergic effect (hypotonia and bradycardia) or functional disorder of internal organs - liver and heart.

When examining people, who have worked for a long time in the POC production, there are phenomena of vegetative dystonia (stable red dermographism, acrocyanosis, and acutely positive clinostatic reflex).

Sometimes, there is the disorder of the central and peripheric nervous system, language disorders, finger trembling, and spastic paralyses. In rare cases, there are psychological disorders: hallucination and depression. There are characteristic disorders of carbohydrate and protein forming functions of liver (change

of sugar curve, decrease of the concentration of albumin and the increase of globulin mostly due to a1-, a2-, and P-fractions), as well as inhibition of the secretor function of the stomach.

Treatment. Tactics of provision of the first aid to patients is determined depending on the way of permeation to some pesticide. In case of permeation of pesticide preparation by inhalation, it is necessary to take the patient out of the contaminated zone, change his/her clothes, and remove obstacles for free breathing. If the preparation touched the skin, it is necessary to wash it with warm water and soap, treat with the ammonia solution (5 - 10 %), or chloramines (2 - 5 %). Eyes should be washed with warm water or the solution of sodium hydrocarbonate. When the pesticide gets into the stomach, it is necessary to cause vomiting, flush stomach with warm water, 2 % solution of sodium hydrocarbonate, 0. 25 % - 0. 5 % solution of potassium permanganate. To remove it from intestines, it is necessary to utilize high siphon enema or saline laxatives: 20 to 30 g of magnesium sulfate or salt cake in a glass of water.

To administer adsorbed pesticide preparation is mostly used as a method of forced diuresis. During 2 to3 years, patients are given water burden - isotonic solution of sodium chloride is administered intravenously and 5 % glucose solution (1. 5 to 2. 0 l). A full time catheter is introduced to the urinary bladder for diuresis measuring by the hour, and thenintravenously - 30 % solution of the urine, prepared based on 10 % solution of glucose or 10 % solution of manit. The solution of diuretic matters is introduced in a flow for 10 to 20 min based on the calculation 1 g per 1 kg of the patient's mass. After water burden, it is possible to administer furosemide intravenously in the amount of 40 to 200 mg. Furosemide is also recommended after the introduction of the urea, if diuretic effect was failed to be called. After administering of diuretic preparations, it is necessary to continue water burden, which includes 4. 5 g of calcium chloride, 6 g of sodium chloride and 10 g of glucose per 1 l of water. Similar cycle can be repeated in 4 to 5 hours until poison disappears from the blood flow.

Detoxication of organism is recommended by the method of hemosorption.

An important peculiarity of urgent therapy at acute poisoning with POC is to use specific antidote means as soon as possible. They include: anticholinergic drug and cholinesterase reactivator. Effective antidote is first of all atropine sulfate, 0. 1 % solution is introduced hypodermically (1 ml) or intravenously (2 to 4 ml) in 5 % solution of glucose, at the necessity - and then (20 to 80 ml and more per day until threatening of symptoms of intoxication to life disappear). Good result is provided by utilization of anticholinergic drug of the central and peripheric action: arpenal (1 to 2 ml of 2 to 5 % solution hypodermically or intramuscularly), aprophen (1 to 2 ml of 1 % solution hypodermically or intramuscularly), benactyzine (0. 001 to 0. 002 g inside 3 to 6 times a day). Throughout the first three days, it is necessary to join anticholinergic drugs, which remove "muscarinic" and "nicotine" effects, with cholinesterase reactivators. Among preparations of this group, the most widely used are trimedoxime bromide (1 ml single dosage of 15 % solution, the treatment course is from 3-4 to 7-10 ml intramuscularly or intravenously), isonitrosine (3 ml of 40 % solution, for the treatment course - up to 4 g).

To release nicotine-like reaction, it is necessary to administer ganglionic blockers: benzo-hexamethonium (0. 5 - 1. 5 ml of 2 % solution), pentaime (0. 1-0. 3 ml of 5 % solution), and hygronium(1 ml of 0. 1 % solution).

In case of appearing of POC poisoning, which is accompanied by movement agitation and spasms, it is necessary to use sodium oxybutyrate (40 ml of 10 % solution intramuscularly or intravenously), magnesium sulfate (20 ml of 25 % solution intramuscularly or intravenously).

Urgent therapy at acute POC poisoning should include a complex of intensive therapy, directed at the support of functions of the central nervous system, cardiovascular and respiratory systems, as well as liver, kidney and blood. Treatment of chronic POC intoxication is mostly symptomatic, with the consideration of main clinical syndromes, through which intoxication is shown in every separate case.

Verification of the ability to work. After having acute POC intoxication in the mild form, as well as in case of weak manifestation of chronic impact (moderate astehnisation, vegetative and vascular dystonia),

the ability of patients is kept. In such cases, where there is acute intoxication or marked chronic intoxication, further work in the contact with toxic matters, with much physical loading is prohibited.

Preventive measures. Prevention of POC poisonings includes a complex of hygienic, sanitary and technical, treatment-preventive and other measures.

Among hygienic measures, first of all, it is necessary to note the following: hygienic selection of pesticide preparations, replacing of hazardous pesticides with less hazardous ones, and hygienic reglamentation of POC utilization.

Sanitary and technical measures can include the following: improvement of methods and ways to use pesticides, as well as rational work organization.

Treatment and preventive measures include preventive and periodical medical examinations. It is necessary to remember that teenagers below 18, men over 55, women over 50, pregnant and breast-feeding women, as well as those who underwent infectious diseases or surgeries for the last 12 months.

Medical examinations should include utilization of a complex of laboratory research. When contacting with pesticides of all groups the following should be done: blood research (hemoglobin, leukocytes, and ESR); X-ray; defining of the content of bilirubin, fructose monophosphate-aldolase in the blood serum, and the general urine analysis.

An important diagnostic criterion for those who work in contact with POC is the activity of cholinesterase in the blood serum and erythrocytes. The decrease of the activity of cholinesterase by 25 % of its initial meaning is the reason to terminate the contact with pesticides of this group.

During the period of intensive work in contact with POC, it is recommended to give 0. 5 g of pancreatin two or three times a day, which connects POC and has positive impact onto the functions of gastrointestinal tract.

Intoxication with Chlororganic Compounds. Chlororganic compounds (COC) are referred to the most widely used pesticides. Until now, the following ones are used most often: hexachloran, dichlorodiphenyltrichloroethane, polychloropinene, chlorindan, heptachlor, dildrin and polychlorpinen. The peculiarity of COC is its high resistance inthe environment. This can be explained by the fact that the temperature, humidity, acids, and alkaline do not have any impact onto them and do not ruin their microorganisms. These pesticide organisms get into the organism with water, food, respiratory system, skin and placenta. They can be discharged by kidneys, intestines, and milk glands. Besides, COC can create deposits in the organism, in particular in adipose tissue. From this deposit, they permeate to blood and stay there for a long period. COC are not as toxic as POC, however they are more dangerous as they can cause chronic poisoning. As to their character, they are neurotropic and parenchymatous poisons.

COC is a strong allergen matter. After contact with it, the following diseases can appear: bronchial asthma, hives, allergic rhinitis, dermatitis and eczema. Besides that, COC can cause gonad-toxic and embryo-toxic action; in the experiment under the impact of small concentrations, the duration changes, and the number of estrous cycles decreases, number of fruits decreases as well. Their teratogenic action has been noted as well; vital capacity of born animals is relatively less, their mass gain is slower, and they are behind in their physical development.

Pathogenesis. Mechanism of the action of COC onto the body of a person has been determined completely yet. It is supposed that the initial factor of the action of COC is the inhibition of ferment systems of the organism, which condition conditioned reflex activity, morphological disorders and clinical manifestations.

All COC are strong protoplasmic poisons, which affect the nervous system and parenchymatous organs. Thus, clinical pattern of poisoning with COC can be characterized by significant polymorphism.

Pathologic and anatomic pattern. At acute poisoning with COC, well marked plethora of internal organs and brain, fine nuclear and diffusive hemorrhages into lungs can be observed. As to the histological

state, there is swelling of vascular walls, and dystrophic changes of the nervous cells can be observed in the cerebral cortex; single fine nuclear infiltrates from cells of lymphoid type and histiocytes can be noted in the heart muscle; as well as swelling of cells of liver and kidneys.

At chronic action of COC, perivascular swelling with dystrophic changes of nervous cells of the cerebral cortex can be observed. There are areas of hemorrhages and degenerative -inflammatory changes in lungs, liver, kidneys and myocardium.

Clinics. In the clinical pattern of poisons with COC there are acute and chronic poisons.

A c u t e p o i s o n i n g. Their clinics depend on the way of permeation of the pesticide into the organism. In case of permeation through respiratory organs, the pattern appears in the first 1 or 2 years. State of coma develops; salivation and bronchorrhea increase; respirationis affected; and collapse can be noticed. Death comes due to inhibition of cardiovascular activity and paralysis of the respiratory center. If a patient survives, he/she develops toxic affection of liver and kidneys (acute renohepatic insufficiency).

Mild inhalation poisoning is manifested through the headache, general weakness, irritation of mucous tunic of the upper respiratory ways, and coughing. There can be nausea, vomiting, moderate increase of liver and pain in the right hypochondrium.

In case of permeation of pesticide to the gastrointestinal tract, at first there are gastric disorders, then the disorder of the function of the central nervous system. In severe cases, dyspnea, cyanosis, increased agitation, trembling, ataxia, fits of clonic and tonic spasms, psyche disorder, vision affection, as well as symptoms of the affection of liver, kidneys, heart and lungs, accompanied by signs of acidosis can take place.

When some products permeate through the skin the following symptoms can take place: skin reddening, rash and dermatitis.

C h r o n i c p o i s o n i n g. They can be characterized by a headache, increased fatigability and irritation, sleep disorder and weight loss. There are signs of the affection of the nervous system (vegetative dystonia) and the pathology of internal organs. At the earliest stage of intoxication, neurological disorder can be manifested through non-specific toxic asthenia. Sudden headache with nausea, general weakness and profuse sweating or fit-like dizziness, accompanied by skin paling and bradycardia can take place.

In later stages of chronic intoxication with COC, the pathological progress involves peripheral nervous system (vegetative-sensor polyneuritis).

The disorder of cardio-vascular system can be characterized by mostly vegetative and vascular dystonia with the inclination to the arterial hypotony, as well as extracardial disorders with cardiac rhythm (sinus bradycardia) and functions of myocardium conductivity. Often toxic dystrophy of myocardium or myocarditis of toxic and allergic character can often develop, in particular among those, who have gone through acute COC intoxication.

Already in early stages of chronic intoxication with COC, secretor function of the stomach is in disorder, for more marked stages of the characteristic development of chronic gastritis with the inhibition of the secretor function of the stomach, up to histamine-resistant achylia.

The disorder of the functional state of the liver at chronic intoxication, at first, can be manifested through the increase of the activity of organo-specific ferments in the blood serum, and later it is accompanied by hydrocarbon and anti-toxic functions. At severe cases of intoxication, toxic hepatitis can develop, which develops without jaundice. As to kidneys, it is characteristic to have some phases in the development of function disorders: at the initial stage, functional activity increases due to the increase of blood circulation in kidneys and glomerular filtration, and at later stages due to the development of toxic nephropathy of kidney functions can be significantly affected, and sings of azotemia takes place.

The analysis of blood shows that the anemia, moderate leucopenia, relative lymphocytosis, and eosiopenia. The number of trombocytes decreases and ESR slows down.

Thus, COC causes the affection, first, of the nervous system and parenchymatous organs, and liver suffers most of it. Due to that, COC is referred to hepatotropic poisons.

Treatment. General principles of the provision of the first aid are analogous by the fact that when POC poisoning. Besides, it is necessary to take into consideration, that at acute poisoning with COC, especially when the pathological process involve kidneys, hemodialysis is recommended. A good result is also given by the utilization of peritoneal dialysis in connection of accumulation of COC in fat deposits, as well as hemosorption.

To treat acute poisoning with COC, it is necessary to use anti-oxidants, which prevent their oxidation, formation of toxic products of their transformation. They include some vitamins and amino acids (a-tocopherol and galascorbin).

Treatment of chronic pesticide intoxications is mostly symptomatic, with the consideration of main clinical syndromes, with which intoxication is manifested in every specific case.

In case of functional disorders of the central nervous system treatment should be complex, with the utilization of medicinal drugs, physiotherapeutic procedures, therapeutic exercises, keeping to the labor schedule, rest and feeding.

In case of vegetative and vascular dystonia it is necessary to have preparations, which have adrenolytic action (ergotamine and dihydroergotamine), as well as anticholinergic drugs (atropine). At the presence of angiospasms and arterial hypertension, electrophoresis with magnesium sulfate is recommended. Good results have radon baths, elecrophoresis with Novocain onto the collar zone.

If the pathological process involves hypothalamic area, depending on the character of crises it is possible to use spasmpolitic, adrenolitic and cholinolitic preparations, as well as ganglioblockators. It is recommended to use piroxane, and antihistamine preparations (Dimedrol and suprastin).

With the purpose of desintoxication, it is advised to use glucose with ascorbic acid, vitamin preparations, glutamine acid, and oxygen hypodermically. To liquidate pain, it is recommended to utilize analgin; at vegetative character of this feeling, it is recommended to utilize pachycarpin, gangleron and aminazine.

At changes in cardio-vascular system, treatment measures are directed at the normalization of vascular tone - utilization of toning or sedative preparations. Oxygenotherapy, also oxygen, salt and pine needle bath, and nitrogen baths.

In case of dystrophic changes of myocardium to improve exchange processes in cardiac muscle, it is necessary to include vitamins of group B (thiamine, pyridoxine and cyanocobalamin), as well as ascorbic acid and retinal.

Good results onto energetic processes in myocardium are given by riboxine, and potassium salt.

At toxic hepatitis together with action therapy it is recommended to use preparations, which improve the exchange in liver cells, vitamins of group B, antioxidants (acetate tocopherol) unithiol and sirepar. In severe cases, hormonotherapy is recommended.

Considering the fact that toxic hepatitis is complicated with cholicestite, it is widely recommended to use antispastic preparations (no-shpa), antibiotics, "blind" dosage with utilization of cholagogue preparations.

Verification of the ability to work. In case of acute COC poisoning, as a rule, it is recommended to temporarily terminate from work, which are connected with the action of toxic matters.

At the presence of chronic intoxication, patients should be transferred to the work beyond the contact with pesticides; also work with hard physical burden and under conditions of intensive sun irradiation. It is also necessary to terminate any further contact with COC at relapse, organic affection of the nervous system and toxic hepatitis.

Prevention measures. To prevent intoxication with COC the following is important: qualitative render of preliminary and periodical medical examinations; thorough sanitary observation with prevention

and utilization of pesticides; utilization of individual protection means when working with them (duration of the work day should be limited by 6 hours when working in contact with hexachloran, heptachloran, etc).

Intoxication with Mercuric Organic Compounds. Mercuric organic compounds (MOC) refer to the most effective pesticides. They are used in industry (e. g. in paper industry to decontaminate woods), they are a portion of paints, which are used to cover bottoms of ships, hydroplane, internal surface of water pipes, and submarine acoustical devices. Main mercuric organic pesticides: ethyl mercuric phosphate, granosan, and mercuran. All the preparations of this group are highly toxic, with marked cumulative properties.

Boundary permitted concentration of the content of mercury for all the matters is 0. 005mg/m3.

It permeates into the organism through respiratory organs, gastrointestinal tract and skin. They circulate in blood, can be found in all biosubstrates; and they permeate into blood through placenta. It accumulates in the cerebrum, liver, kidneys, and adrenal glands.

These compounds discharge very slowly, mostly through kidneys and esophagus, as well as with mild, then with bile and saliva. MOC are more toxic than non-organic mercuric compounds. They are referred to enzymatic poisons. Besides, MOC possess well-marked gonado-toxic, embryotoxic and mutagenic effect. It has also been stated that these compounds cause allergenic action as well.

Pathogenesis. MOC acts onto the organism with metal mercury. They are protoplasmic ferment poisons and have impact onto carbothiolic groups of numerous cellular enzymes, which catalyses various types of exchange. It has been known that for normal functioning of ferments of their sulfahydril groups should be free. Bonding with compounds of mercury, they are blocked and in the result, the ferment inactivates, what leads to deep disorders in the tissue exchange. Besides, mercuric compounds stay in the cell membrane, affecting metabolism of cells and decreasing their content in RNA. The development of pathologic changes in various organs is much assisted by capillary-toxic action of MOC.

Pathologic and anatomic pattern. Mercuric organic pesticides cause sharp fatigability, symmetric atrophy of the cortex of frontal lobe and cerebellar hemispheres. Plethora of folds of small intestine, small hemorrhages under pericardium and endocardium of the left heart ventricle, and under the mucous coat of stomach can be observed. Histologically, nervous cells of the grain layer of cerebellar cortex are wrinkled; neuroglia expanding in the layer of Purkinier cells can be noted. Dissolving of mieline can be observed in lateral columns of the spinal cord and myeline coat of cauda equina roots. There are dystrophic changes in the liver, kidneys, and heart muscle.

Clinics. A c u t e p o i s o n i n g. At permeation of pesticides through the respiration organs, clinical manifestation is manifested rather fast, and through gastrointestinal it takes place a bit later. In general some consecution in the dependence on the way of pesticide permeation into the organism can be observed: at inhalation permeation - first of all, changes start in the nervous system and in case of permeation of poisoning through a mouth - dyspeptic phenomena can be observed. Initial manifestations of poisoning are manifested through illness, general weakness, headache, and dyspeptic disorders. In the mouth, there can be unpleasant "metallic" taste, and in particular cases, gums swell and bleed. Asthenovegetative syndrome develops. This period of the disease lasts for several days, and then the state of the patient worsens.

Some patients have ulcerous gingivitis and stomatitis. Signs of the affection of the central and peripheral nervous system develop. Lumbosacral radiculitis, polyradiculoneuritis, toxic and encephalomyeloneuritis develop; pain and parastesia appear in extremities, mostly in distal sectors; there are also superficial disorders in the form of "gloves", "socks' and "stockings". Distal sectors of extremities become cyanotic and cold.

In severe cases, walking disorders, memory decreases, trembling of stretched upper limbs takes place and speaking is unclear.

Some patients have a clinical picture with features, characteristic for the diencephalons syndrome. The temperature and arterial pressure increase, palpitation, body trembling and constant thirst take place.

Besides, nervous system, gastrointestinal channel and kidneys suffer as well. Appetite decreases, nausea and vomiting, pain in right hypochondrium, liver increases, and gingivitis develops. In kidneys - toxic nephrosis.

Patients have pain in heart and palpitation. The development of toxic myocarditis is possible. Arterial pressure usually decreases.

For the affection of organs of sense are characteristic disorders of scent, decrease of hearing and sight.

For severe poisoning, it is characteristic to have moderate anemia, some toxicosis with the shift to the left, toxic grain of leucocytes, and the decrease of monocytes.

C h r o n i c p o i s o n i n g. Chronic intoxication with MOC develops in the form of more or less outlined clinical stages. At the initial stage, there is an asthenovgetative syndrome with elements of erythrism. If the action of pesticides is continued, then trembling of extremities increases, and character that is more marked gets vegetative disorder, including erythrism.

The second stage of the MOC chronic intoxication can be characterized by major affection of hypothalamus. In the clinical pattern, there are disorders of sleep, melancholy, unexplained fear, disorder of thermal regulation and cachexy.

At the third state, organic nucleuses or diffusive disorders of the central nervous system are brought to the forefront: mostly toxic encephalitis and encephalomyelitis develop.

Together with the development of the pathology of the nervous system on the clinical pattern of the chronic intoxication with MOC therecan be signs of toxic dystrophy of myocardium can develop, as well as toxic hepatitis, for which it is not characteristic to have fast progressing and the development of jaundice.

Often, at the chronic intoxication with MOC, hypochromic anemia develops, anisocytosis and poikilocytosis appear, and the content of reticulocytes increases. The number of leukocytes first increases and then leucopoenia develops. The content of thrombocytes decreases and the ESR increases.

One of the most important signs of the MOC impact onto the organism, there is presence of mercury in biological environments. At intoxications of mild and mean severity, mercury can be determined in blood and urine; its last excretion is usually more than 0. 01 mg/l and increases along the increase of intoxication. However, complete dependence between severe intoxications and the content of mercury in the urine is absent,

Treatment. In case of appearing of acute poisoning with MOC, utilization of antidote therapy should be envisaged (substances of the carbothiolic group). Positive result can be achieved by administering of a domestic preparation of unithiol (5 - 10 ml of 5 % solution intramuscularly or intravenously, every 3 to 6 hours). At the course of treatment, it is necessary to have 50, and in severe cases up to 200 ml of 5 % solution of unithiol. In case of peroral poisoning, peroral can be taken in (100 - 150 ml of 5 % solution) with further gastric lavage.

Besides unithiol it is possible to use complexing agents at poisoning with MOC (tatacin-calcium, pentacin, etc), as well as preparations, which are synthesized based on the physiological metabolites (succimer, 0. 3 g of alkaline solution intramuscularly on the first and second days every 6 hours; on the third and fifth days - every 8 hours, and on the sixth and seventh day - every 12 hours).

Besides antidotic therapy, symptomatic treatment is conducted, which is targeted at the normalization of the functional state of main organism systems.

Treatment of chronic poisoning with MOC can be conducted at the presence of some poisoning syndromes. To fix the results of the treatment, it is recommended to prescribe hydrogen sulfide baths in sanatoriums.

Verification of the ability to work. In case of the suspicion of the presence of intoxication (mercury presence n urine), it is necessary to relieve the sick temporarily from further contact with mercury. In case of "mild" intoxications, and moreover after acute poisoning with mean and severe phases, it is recommended to terminate any contact with mercury in full. It is also possible to provide a person with occupational invalidism.

Preventive measures. Sealing-in of machines, utilization of individual protecting means (respirators, overalls, protective glasses, andrubber gloves), and removal of manual operations. In case of necessity, it is recommended to conduct demercurization of the premises, aerosol inhalations with 5 % solution of unithiol in the dosage of 5 ml three ties a week during a month, as well as preliminary and periodical medical examinations of workers who contact with mercury.

Intoxication with Carbamates. Carbamates, which are used as pesticides, are derivatives of aminoformic, thiocarbamic and dithiocarbamic acids. As pesticides, ethers of aniformic acid, oxide salts of alkali element and heavy metals of dithiocarbamic acid.

Preparations of this group are used as herbicides, insecticides, acaricide, fungicides, and bactericides. The majority of carbamates are highly toxic for insects and little or medium toxic for people. It is very important that carbamates have impact onto insects, which are not sensitive to POC and COC.

Together with these carbamates, there are a number of significant drawbacks. Thus, they destroy a number of useful insects, and are very toxic for earthworms.

Carbamades are poisons of parenchymatous and neurotropic actions. Besides, some carbamates are characteristic to have embryotoxic, gonadotropic, teratogenic and mutagenic actions, many of them are active allergens, and some of them are cancerogenic.

Pathogenesis. Carbamates are "direct" inhibitors of cholesterase. In the process of joining of the ferment with carbamates, there is cholesterase carbamilizing with formation of a complex of "carbamate-cholesterase". It is characteristic that this complex is very unstable. This cholinesterase is capable to spontaneous reactivation.

The mechanism of actions of ditiocarbamates is reduced to inhibition of ferments of oxide-renovating cycle; most likely, in the result of the interaction with their sulfhydryl groups. Besides, ditiocarbamates disturb carbohydrate exchange, by increasing glycolitic processes and inhibit the accumulation of oxygen by tissues. In the process of decomposition of ditiocarbamates, the following is discharged: carbon bisulfide, which is bound with amino groups of amino acids, peptides, and proteins, it blocks them, disturbs protein exchange and causes changes in many organs and systems. Thus, carbon bisulfide, which is formed in the organism endogenically in the process of metabolism of ditiocarbonates, is a significant component of their toxic impact. Besides, it has been stated that ditiocarbonates disturb the exchange of microelements.

Clinics. When getting poisoned with carbamates, there are symptoms of the irritation of parasympatic nervous system: narrowing of pupils, spasm of accommodation, bradycardia, hypersalivvation, fibril twitching ofmuscles, there is nausea, vomiting, pain in abdomen, and diarrhea. Further, there are phenomena of bronchorrhea, bronchospasms, and pulmonary swelling is possible. As to the nervous system, at first, agitation takes place, then entanglement of consciousness, and spasms. In the blood, there is a decrease of cholinesterase. Dermatosis, nettle-rash, conjunctivitis, and irritation of the upper respiratory tracts can develop (rhinitis, bronchitis and tracheitis).

The peculiarity of the impact of carbamates onto the organism is their ability to cause changes in the structure and functions of glands, which have no ducts, and first of all, of thyroid gland.

Many carbamates cause the affection of the nervous system and organs of haemopoiesis, have allergenic properties and form methemoglobin. Sevin, cineb and ciram are the most toxic for people.

Poisoning with sevin. Sevin is naphthylmethylcarbamate, and is referred to derivatives of aminoformic acid. This compound with the most marked insecticide properties comparing with all carbamates has anticholesterase activity.

Acute poisoning with sevin is characterized by agitation of M and N - cholinoreceptors. The following takes place: headache, dizziness, nausea, vomiting, salvation, coughing, and complicated breathing. In lungs, there are dry crepitations. The preparation inhibits the immune reactivity of the organism and causes various allergic reactions of respiratory organs and skin.

The earliest symptom of poisoning with Sevin is the decrease of the activity and cholinestase. Based on other indications, some diagnostic meaning is possessed by the decrease of erythrocytes and hemoglobin.

Chronic intoxications with Sevin are not described.

Poisoning with cineb. In case of poisoning with cineb, the body temperature can significantly increase, pulse and breathing become more rapid, skin and whites of eyes become bluish. There are cases of hemolytic anemia, sulfhemoglobinemia, which are connected with the impact of carbon bisulfide, formed at the decomposition of the preparation.

Poisoning with ciram. Ciram has strong irritating properties, in particular when getting to the skin, mucous tunic of eyes and upper respiratory ways.

All these preparations inhibit leucociytopoesis. Leucopoenia develops due to the decrease of the content of neutrophil and eosinophilic granulocyte. Together with this, often the content of erythrocytes decreases in blood, and hypochromic anemia develops.

Treatment. At acute poisoning with carbamates, a patient is prescribed to inhale 25 % solution of ammonia. The antidote is atropine (12 ml of 0. 1 % solution intramuscularly, in case of necessity injections are repeated every 8 to 10 min until state improves). In comparison withpoisoning with POC, overdosage of atropine is dangerous, and utilization of acetyl cholinesterase reactivators is non-expedient (due to fast spontaneous reactivation).

Treatment of patients with chronic poisoning is syndromic (alkali inhalations, disensibilizing preparations, hemostimulators, vitamins E, thiamine and pirodixin) together with amino acids, which contain sulfur and copper preparations.

Verification of the ability to work. Expert issues can be solved with the consideration of the degree of severity of the undergone poisoning. Mild forms of acute intoxications are reverse, and thus the ability to work of such people is not disturbed. In case of more severe poisoning and at chronic intoxications, further work under condition of the action of carbamates is contraindicated.

Preventive measures. To prevent poisoning with carbamates, it is necessary to keep to generally accepting preventive measures. During the period of intensive work in contact with these pesticides, it is necessary to research the activity of cholinesterase in blood. In case of the decrease of the activity of this ferment by 25 %, further contact with pesticides should be terminated until complete renovation of this indicator.

Intoxication with Compounds which Contain Arsenic. Arsenious compounds, which are used in agriculture, include calcium arsenate and Paris green. These pesticides are highly toxic compounds and they are very stable in the environment. These substances permeate into the organism via lungs, gastrointestinal channel and damaged skin. They are discharged with urine, bile, faeces, as well as with excretions sweat-glands, mammary glands, and with exhaled air. These preparations are capable to accumulate in bones, liver, kidneys, and mucous tunic of the stomach, skin, hair and nails. After exceeding the blood-brain barrier, they deposit in hypophysis.

Pathogenesis. Compounds of arsenic lock numerous ferment processes, disturbing carbohydrate and fatty exchange, as well as tissue respiration. Carbothiolic ferments, which form stable toxic compounds with sulfhydryl groups, are the most sensitive to the action of arsenious compounds. Arsenious compounds

increase the permeability of walls of capillars and cause hemolysis of erythrocytes. Small dosages of arsenious preparations stimulate haemopoiesis, and big ones - inhibit it, up to the development of hypoplastic anemia.

Pathologic and anatomic pattern. At the action of pesticide preparations with arsenic onto the blood of a human being, plethora of internal organs, small hemorrhages into the pleura and pericardium, fatty degeneration of the liver with the transition to atrophy, as well as the increase of spleen with the decomposition of erythrocytes. In varioussections of the central nervous system, there are hemorrhages, nucleus affection of cells of lateral horn and anterior horn of the spinal cord, and peripheral nerves. And in marrow, hyaline degeneration is up to the atrophy of myeloid elements.

Clinics. A c u t u t e p o i s o n i n g. Clinical manifestation of poisoning with arsenious compounds depends on the way of the poison permeation, its dosage, concentration and individual sensitivity. According to the type of the permeation of the poison to the organism there are three main clinical forms of acute poisoning: gastrointestinal, paralytic and the one which is manifested through the affection of respiratory tracts.

At gastro-intestinal form, patients get metallic taste in their mouth, burning in pharynx, pain and difficulties when swallowing, sharp pain in stomach, irrepressible vomiting, which can last from several hours to several days. Vomiting can be with a mix of bile, and sometimes of green color due to the presence of arsenious compounds. Then it is accompanied by liquid faeces with tenesmus. Emptying looks like rice-water, sometimes with blood. Due to significant loss of water, organism dehydrates fast. Clinical pattern reminds cholera, and this semblance is increased due to joining by the increased general weakness with dizziness and loss of consciousness. Temperature decreases, cramps in gastrocnemius muscles can take place; the number of urine decreases up to the development of anuria. This form of acute poisoning is often lethal.

Among patients with acute poisoning, gastro-intestinal phenomena do not have time to develop, as paralytic form of poisoning takes place rather rapidly, when general weakness, feeling of fear, drowse, dizziness and coma appear and develop fast. During the state of coma, there can be cramps, and sometimes epileptiform fits. Death can develop within a day.

When the crisis is over, a patient will have signs of diffusive affection of the nervous system in12 to 14 days: headache, dizziness, and possibly comatose state.

However, peripheral nervous system suffers most often. There are polyneuritis and myelopolyneuronitis. They can be characterized by active progressing, significant expression of pain syndrome, expansion of paralyses, and symmetry of the affection. Mostly, they affect radial nerves and lesser occipital nerve. At first, paresthesia takes place, and then weakness appears in upper and lower limbs, which start with distal sectors and then spreads and progresses up to the degree, that walking becomes unstable and weak due to the affection of muscle sensitivity - atactic form of polyneuritis. In other case, parastesia of bones is accompanied by intensive burning pain. It can appear either spontaneously or from touching. However, only subjective feeling of pain stays very soon, hyperesthesia is replaced with dulling of pain, tactile and temperaturesensitivity, and in distal parts of limbs - anesthesia, which is a specific ability of muscle polyneuritis.

Pareses of soles and fingers appear quickly. In the most severe cases, tetraparesis develops. Often, there is pigmentation and peeling of skin, hair falls out and nails are fragile. Approximately in two months after the disease started, white-gray cross Mees's lines appear on nails -impregnation with arcenious acid, pathognomonic symptom of poisoning with arsenic.

In case of permeation of arcenious compound through respiratory system, initial stages of the disease can be characterized by sharp pain in eyes, lacrimation, nosebleed, coughing, hemoptysis and pain in chest. The temperature increases. In more severe cases, it is also accompanied by diarrhea, loss of appetite, pain in stomach, nausea and sometimes vomiting.

C h r o n i c i n t o x i c a t i o n. In case of systematic permeation of poison into the organism, the amount of which increases gradually, accustoming to it can take place. In the majority of cases, at prolong impact of small dosages of poison into the organism, chronic intoxication develops.

Patients have progressive loss of weight, loss of appetite; often have metal taste in the mouth, general weakness, fast fatigability, and decrease of temperature, sharp pain in eyes, coughing, and nosebleed. Dryness of the mucous tunic of a nose and especially of the mouth cavity is a specific feature of intoxication with arsenious compounds, what makes it look like poisoning with chromium and fluorine.

At objective examination of patients, it is possible to find inflammation of the nasal part of gullet and it covering with ulcers, and sometimes breaking of the nose partitioning, laryngitis, tracheitis and bronchitis.

Periodically, there are dyspeptic phenomena: nausea, vomiting, diarrhea, and pain in stomach. Memory and ability to work worsen. They suffer from headaches, fine trembling of upper limbs and language disorder.

Polyneuritis, which appear at chronic intoxication with arsenic, are very painful, and develop with the affection of sensitivity and have inclination to fast development of repeated contractions, pareses with further muscle atrophy.

It is characteristic for chronic intoxication with arsenious compounds to have significant skin pigmentation - arsenious melanoderma, which starts with inguinal folds and palms and then spreads to peripapillary circles breasts, Sergent's white line and then spreads around the face, locating diffusively or in spots. Face and mucous tunic are not affected. Simultaneously, X-disease develops in these areas. In some cases, chronic intoxication with arsenious compounds develops with carious affections of haemopoiesis: anemia, leucopoenia and, sometimes, agranulocytosis.

At prolong impact of arsenious compounds, skin cancer can develop.

Treatment. Treatment measures envisage termination of the contact with poison, gastric lavage with the solution of slack magnesia (1 - 1 V spoons per 5 glasses of boiled water) with further administering of 25 to 30 g of magnesium sulfate with 400 g of water, and antidotes (Antidotum arsenici or Antidotum metallorum) via a probe.

An antidote is also a mixture of non-dissolved ferrous hydroxide with the solution of magnesia sulphite, which is done right before administering by mixing the solution of slat sulphite (100 units per 300 units of water) and slacked magnesia, which is triturated with water (20 units per 300 units of water). The received solution is mixed and is drunk in the amount of 1 spoon in five minutes.

Positive effect is provided by prescription of carbothiolic preparations: unithiol (5 % solution - 10 ml), sodium tiosulphate (30 % solution - 5 - 50 ml), lipoic acid (0. 5 % - 2 ml) and BAL.

In case of development of gastro-intestinal form, it is necessary to administer intravenously a solution of glucose (20 ml - 25-40 %) with ascorbic acid (500 mg); at severe forms, corticosteroids and hemodialisis are recommended.

At arsenious polyneuritis, positive result is shown by administering vitamins (B1, B2, B6, B12, ascorbic and glutamine acids), as well as physiotherapeutic procedures (UV irradiation and ozokerite), massage, and curative gymnastics.

Verification of the ability to work. At mild forms of chronic intoxications, it is recommended to transfer a patient to another job for some period of time with the provision of the sick leave for the period of two months. At marked forms of poisoning, it is recommended to get fulltime job, and here can be an issue regarding the transfer of the patient to an occupational invalidism group.

Preventive measures. Individual protection when working with dust includes respirators, protective glasses, and gloves, and keeping to measures of personal hygiene. Preliminary and periodical medical examinations with the content of arsenic in urine in the amount should not be more than 0. 5 - 1 mg/l, as

well as in hair and nails. Treatment and preventive meals, daily administering of 150 mg of ascorbic acid, and mild (intensifies discharge of arsenic from the body).

Intoxication with Pirethroids. Pirethroids are chemical compounds, which are a very perspective group to use against pests in agriculture. They are synthetic analogues ofnatural pirethrins, which are contained in chamomile flowers. Mostly widely spread are representatives of this group of pesticides as decis, rovucuritis, phenopropatrin, permetrin, etc.

The majority of perithorids has low volatility, is badly dissolved in water, and destroys fast under the impact of light. Significant advantage of pirethroids is their high insecticide activity, in the result of what the necessary amount to use is very small, what deceases the danger of the development of intoxication.

Pathogenesis. It has been found out that these preparations are capable to inhibit cholinesterase. By changing the activity of cholinesterase in erythrocytes, piretroids can affect the structure of cell membranes, causing inactivation of Na - K - adenosine triphosphate system, which is located in them.

Besides, pirethroids can change the content of free radicals, cytochrome P-450 and metal complexes in tissues. Many of them are moderate inducers of monooxygenase system, at long impact of piretroid, it is induced with cytochorme P-450 and the activity of NADF-H-cytochrome - C-reductase increases.

Clinics. On the pattern of acute poisoning with pirethroids, symptoms of the affection of the nervous system dominate: trembling, affection of movement coordination, clonic-tonic cramps, and extremities paresis.

Together with this, the affection of piretroids due to hepathotoxic action cause changes of the activity of cholinesterase of the liver and blood serum, ferments, alkaline phosphatase, as well as the decrease of protein and urea in blood serum.

Some preparations of this group cause local irritation.

Prevention of poisoning with pesticides. Prevention of poisoning with pesticides includes a complex of hygienic, sanitary-technical, treatment-preventive and other measures.

Among hygienic means, first it is necessary to note the following: hygienic selecting of pesticide preparations; replacement of hazardous pesticides with less dangerous ones; as well as hygienic regulation of pesticide utilization.

Sanitary and technical measures include improvement of ways and methods to utilize pesticides, and rational organizational work.

Treatment and preventive measures include preliminary and periodical medical examinations. It is necessary to remember, that the following categories of people are not permitted to work with pesticides: teenagers up to 18, men over 55 and women over 50, as well as pregnant women, breast feeding women, and also those who had suffered infection diseases or surgeries for the last 12 years. Medical examinations should certainly include utilization of a complex of laboratory research. When contacting with pesticides, the following should be done for all the groups: blood testing (hemoglobin, leukocytes, and ESR); fluorography, determination of the content of bilirubin, fructose diphosphate aldolase in blood serum; and general urine analysis.

At medical examination of those, who contact with COC, it is necessary to research blood with definition of a number of thrombocytes, transferases, as well as COC in blood and urine.

The most important diagnostic criterion of those, who work in contact with POC, is an activity of cholinesterase by 25 % of its outcome and is an indication to termination of the contact with pesticides of this group.

At medical examination of those, who are in contact with POC, it is necessary to research the state of the mouth cavity through a possible development of gingivitis and stomatitis; diagnostic value has the definition of the content of mercury in urine.