

Lecture 4

Exogenous allergic alveolitis and occupational bronchial asthma.

(2 hours)

Scientific and methodological substantiation of the topic

Occupational asthma is a part of a larger category of diseases known as occupational respiratory diseases and includes occupation-induced rhinitis and laryngitis, tracheitis, bronchitis and bronchiolitis, chronic obstructive pulmonary disease, lung cancer, and interstitial diseases such as fibrosis and granuloma formation. Although both physicians and the lay public are aware of other occupational lung disorders such as silicosis and asbestosis, OA is the most prevalent occupational lung disease in industrialized countries. Findings regarding the significance of occupation as a cause of asthma vary based on the definition used and the methods for patient selection. In addition, people who develop OA often leave the industry in which the illness began (a bias known as the "healthy worker effect"), even when OA has not yet been diagnosed. In general, asthma affects 5% to 10% of people worldwide, and it is estimated that 2% to 15% of asthma may be occupational in origin.

Literature

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Methodical support

1. Media presentation

Lecture time calculation

№	Questions	Time (min)
1.	Occupational exogenous alveolitis (OEA) or hypersensitivity pneumonitis (HP): etiology, pathogenesis, pathology, etiological classification of OEA	10
2.	Clinical presentation, diagnosis, treatment, working capacity examination.	10
3.	Occupational bronchial asthma: etiology, pathogenesis, pathology, classification.	20
4.	Clinical presentation, diagnosis, treatment.	20
5.	GINA.	Students' self-studying 30

1. 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). Signs, 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 bothered with dyspnea, sometimes with moderate fever and drowsiness. 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 *Thermoactinomyces 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 hay to the organism of the human body. In the clinical pattern, 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 1 mg/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 rotting 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.

2. 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, into 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 (pathophysiological 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 hypersensitivity takes place.

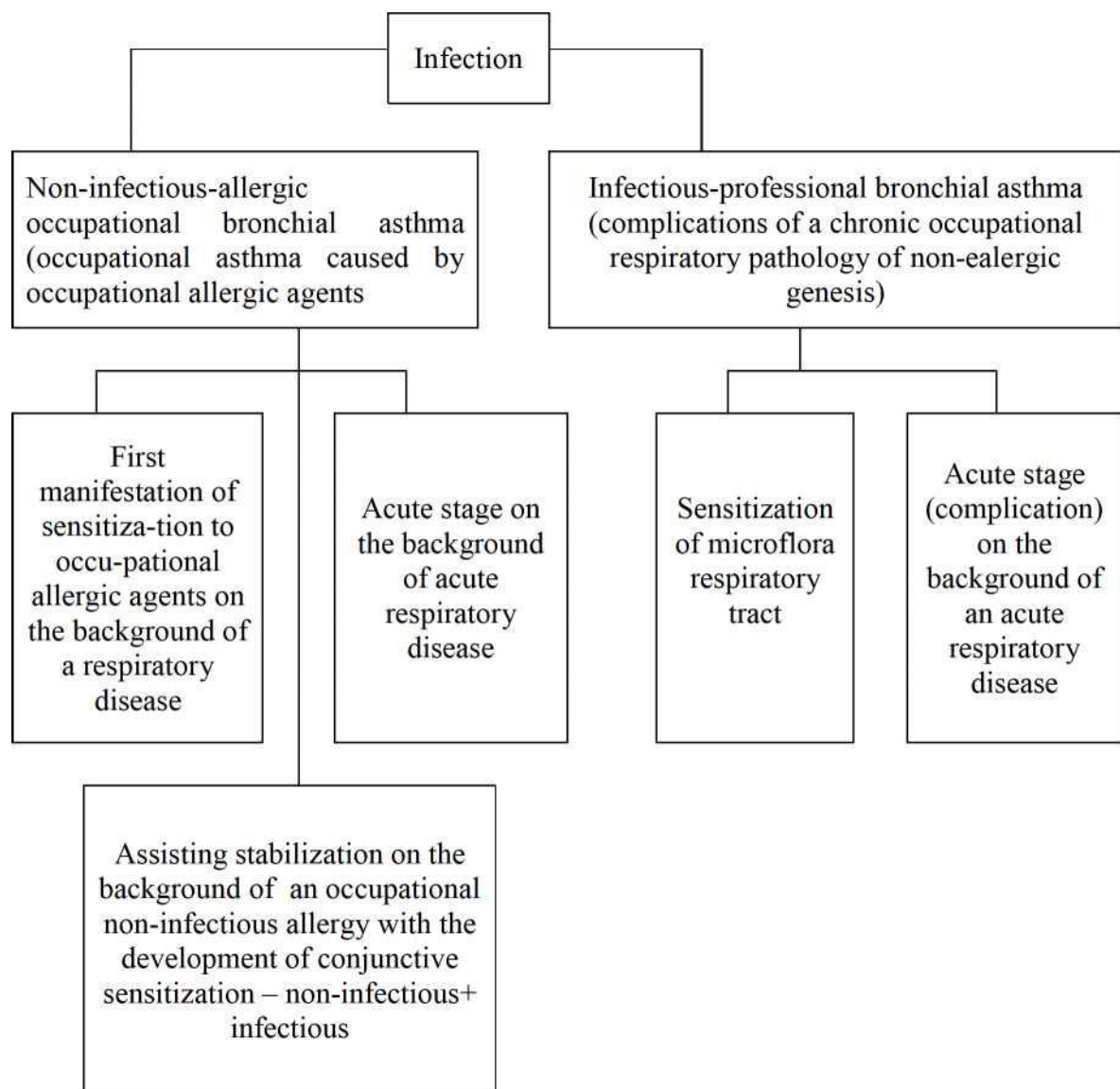
Prolonged impact of asthmogenic agents causes changes in the reactivity of target cells (first of all, mast cells, located along the respiratory tract). Change of reactivity of these cells is first of all accompanied by excessive production of biologically active matters (histamine and leukotriene). 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 the 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 industrial and 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:



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 wave 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. Signs 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 for asthma of joint 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 bronchoectasies, chronic pneumonia and asthmatic status. Severe and prolonged 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 the 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 Charcot and Leyden), protein spectrum of blood serum, increase of the level of histamine, reduction of excretion of 17-hydroxy-corticosteroids 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, though 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 leukocytosis. 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 contraindications 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 an aerosol 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 neutrophils (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 1.4 and 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, a threat 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 p_2 - adrenoreceptors of bronchi. In particular, they include salbutamol, terbutalin, and alupent. It has been proved that in comparison with other adrenoreceptor agonists (adrenalin and ephedrine), which influence not only P_i and α -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. 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: 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 form 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. Glucocorticosteroids 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 to 1 g a day; Tarasov's mixture internally - 1 teaspoon - 1 table spoon with warm milk - 3 - 4 times a day after meals.

Antibacterial means, particularly when there is purulent bronchitis, antritis, 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.