

dia, tachypnoea have a high predictive value. It is very hazardous to judge the state of the patient just according to the clinical symptomatology alone. It is inevitable to measure the tension of respiratory gases in blood.

Clinical symptomatology is always represented by a triad of symptoms, which are constantly observed: dyspnoea, cough and wheezing. They are always present during the attack in advanced phases of the disease. Attacks often occur at night. The cause is not quite clear. It may reflect the circadian variations in catecholamine and histamine levels or the increased tone of parasympathetic nervous system at night. During the attack the expiration is prolonged, tachypnoea, tachycardia and increased systolic pressure are present. Sometimes also an unproductive cough occurs. The lungs are *overfilled* with air, the back to front diameter of the thorax increases. The retained air in lungs increases the pressure in the thoracic cavity. That is why the breathing becomes more difficult, the patient is restless, cyanosis, tachycardia and increased filling of jugular veins may occur. The episode of attack usually ends with coughing out some viscous sputum, containing cylinders of mucus formed in distal parts of bronchioles (Curschmann's spirals – i.e. cylinders from the bronchioles, consisting of mucus and cells), eosinophils and Charcot-Leyden crystals (crystallized proteins of eosinophils).

Sometimes the cough during the attack may be ineffective. The mucus can obturate some bronchioles and as result atelectasis in some areas of the lungs occurs. Pneumothorax is a very rare complication.

An attack of bronchial asthma has to be distinguished from other diseases associated with dyspnoea. Wheezing occurs also in other endobronchial diseases (neoplasms, aspiration of foreign bodies), or in bronchial stenosis. An attack of bronchial asthma can resemble the acute failure of the left ventricle. During the left ventricle failure gallop rhythm occurs and the sputum is sanguinolent.

The therapy of asthma is aimed, above all, at the elimination of bronchoconstriction. The relaxation of bronchial smooth muscles can be induced by stimulation of beta 2-adrenergic receptors. Several substances having these effects are used in therapy of asthma (fenoterol, salbutamol, terbutaline).

Theophylline is also used as an moderately effec-

tive bronchodilator. It prevents the adenosine formation and inhibits its receptors.

Antihistamines act as antagonists of H₁ receptors. They block the effect of histamine on smooth muscles of airways.

Anticholinergic drugs inhibit the release of acetylcholine from nervus vagus endings in the airways. The effect of atropine is known for more than a hundred years.

Substances acting as antagonists of LTD₄ and PAF receptors are tested nowadays. The glucocorticoids are widely used in the therapy of asthma. These drugs reduce the allergic and inflammatory reactions, decrease the number of cells participating in the development of inflammation. They can be applied also by inhalation. The bronchoconstriction is influenced indirectly, mediated by mitigation of the mucous membrane inflammation.

1.7 Bronchiectasis

Bronchiectasis is an irreversible dilatation of one, or several bronchi. Dilatation is caused by destruction of the bronchial elastic-muscular wall. It occurs usually as result of a chronic inflammatory process. The surroundings of the bronchus are affected by inflammatory alterations leading either to destruction of the bronchial wall, or they develop secondarily due to the stagnation of the secretion. Before the era of antibiotics bronchiectasis was an incurable disease, which participated not only in morbidity, but also in mortality of patients suffering from infections of the respiratory system.

In addition to the infections associated with the destruction of bronchial wall there are other alterations and disturbances (long lasting obstinate cough) which participate in the development of bronchiectasis. Several disorders of mucociliary function form **favourable conditions for bronchiectasis development**. Dyskinesia of cilia or the complete absence of their motility may be transmitted as an autosomal recessive trait (primary ciliary dyskinesia, immotile cilia syndrome). In men with this disorder sterility occurs because of absence of the sperm cell motility. Also in women decreased fertility ap-

pears because the cilia of the oviducts are affected, and the tubal pregnancy occurs more often in such cases. These people are predisposed to the respiratory system infections. The absence of *the clearing mechanism* of mucociliary system participates in the development of infections. Also providing the phagocytosis is intact, *problems occur* due to the fact that *the waste of phagocytosis* is not removed. In patients with the Kartagener's syndrome a triad of symptoms due to immotility of cilia is observed i.e. sinusitis, bronchiectasis and complete situs viscerum inversus. (Situs viscerum inversus occurs most often in presence of ciliar dyskinesia). In the embryonal tissues the motility of cilia is a condition allowing normal rotation of organs. If the ciliar motility is absent, the rotation of organs is accidental, and the dextrorotation and the sinistrorotation are equally frequent.

Bronchiectasis occurs also **in patients with cystic fibrosis** when the lungs are involved. In cystic fibrosis the secretion of exocrine glands is disturbed. Production of viscous secretion aids the development of infection.

Bronchiectasis occurs in **immunodeficient conditions** due usually to defective humoral immunity. Bronchiectasis occurs especially in patients with agammaglobulinaemia. The cause resides in greater disposition to repeated infections and their progression.

Bronchiectasis occurs also after **necrotising pneumonia** and in allergic pulmonary aspergillosis. Bronchiectasis occurs in an infrequent *yellow nail syndrome*. It is a combination of lymphoedema of lower extremities, recurrent pneumonia and yellow nails. There is also a so called poststenotic bronchiectasis. In such case stagnation of secretion occurs and bronchiectasis develops due to the stenosis of the bronchial lumen (caused by e.g. tumor, foreign body or pressure of enlarged lymph-node in hilus). The so called cirrhotic form arises by mechanical traction developed in the surroundings of bronchus due the fibrotic alterations of lungs (fibrotic tuberculosis, diffuse pulmonary fibrosis and sarcoidosis).

At the onset of the development of bronchiectasis an infectious or chemical pathogen is always present. Damage of the epithelium develops. The situation

can be complicated by a decrease in function of mucociliary system, or by immunodeficiency. Dilatation of bronchi occurs in central parts of the respiratory system. The bronchodilatation can reach fourfold dimensions in comparison with those under physiological conditions. Peripheral parts of lungs are poorly ventilated. The stagnation of the secretion, the disturbance of the mucociliary system function and the proteolytic activity of polymorphonuclear leucocytes participate in the progressive destruction of the tissue. Purulent secretion contributes to the destruction of tissue by its high content of proteases (elastase, collagenase, katepsine G). The mucous membrane in the dilated part of bronchi is oedematous, inflamed, with possible necrotic defects. Granulomatous tissue can be produced.

According to radiographic findings three types of bronchiectasis can be distinguished :

- cylindrical bronchiectasis
- varicose (fusiform) bronchiectasis
- saccular or cystic bronchiectasis

In saccular bronchiectasis the preformed cavities are filled with pus. It can reach the peripheral regions of lungs.

Bronchiectasis is associated often with cough of paroxysmal type. In the morning after awakening expectoration increases. It occurs in 90 per cent of patients. About 50 per cent of them have haemoptysis. The disturbance of respiratory function depends on the anatomical type and the extent of bronchiectasis. In advanced cases also dyspnoea can be present. In extensive bronchiectasis, hypoxia can develop usually without hypercapnia. The diagnosis is confirmed by X-ray examination of the bronchial tree, by bronchography and computed tomography. The cultivation of sputum enables an aimed causal antibiotic treatment. Such a therapy minimizes the respiratory dysfunction and the progression of the disease. Bronchiectasis can be complicated by gradually developing pulmonary hypertension and cor pulmonale chronicum.