Case 11
The diseases of respiratory organs

1. A cancer of lung. In lungs there is a focus of grey color without clear scopes with infiltrate growth. In etiology of cancer the main role plays carcinogenic matters, smoking of cigarettes. To the precancer conditions rate chronic bronchitis, chronic pneumonias which are accompanied by hyperplasia, metaplasia and dysplasia of epithelium of bronchial tubes. It is designated that a cancer can arise up in the focus of pneumosclerosis after the suffered tuberculosis, infarct of lungs, around alien bodies, so-called "cancer in scars". In scars there is a number of conditions, contributory infringement malignant transformation of cells: depositing of carcinogens, hypoxia, local immunosupression, violation of intercellular co-operations and other.

2. Chronic bronchitis. A chronic bronchitis is chronic inflammation of bronchial tubes, which arises up as a result of the protracted acute bronchitis or protracted operating on the mucus membrane of bronchial tubes of bacteria or viruses, physical and chemical factors (smoking, supercooling of respiratory tracts, muddiness of air by industrial wastes and etc). It is presently proved that chronic bronchitis almost in 100% cases affects smokers. Chronic inflammation can be accompanied by metaplasia of epithelium, the amount of cells which have cilia goes down as a result. Displasia of epithelium can develop at the permanent action of nicotine smoke, up to development of malignant neoplasms. In a clinic, intensifying of disease is combined with the periods of remission. In majorities of sick people with chronic bronchitis develops emphysema of lungs. Complications of chronic bronchitis is rightventricle and pulmonary insufficiency.

Morphological changes. On the early stages of disease the chronic bronchitis of infectious nature can at first have local character, there is inflammation of respiratory bronchiole which have a diameter less than 2 mm. Chronic inflammation can bring to destruction of walls over the bronchiole and nearlaying elastic fibres, that entails development of center lobular emphysema. The decline of pressure of air and pliability of wall of bronchiole, together with corking of space by mucus, bring air over to the considerable hardening of passing. A chronic bronchitis and emphysema is usually observed simultaneously in a different proportion. The clinical symptoms of disease appear at the vast defeat of bronchial tree. A chronic bronchitis developes more frequent in a bronchial tube II, VI, VIII, IX and X segments, where in more frequent focuses of pneumonia and there are unfavorable pre-conditions for resorption of exudate. There is a hypersecretion of mucus in bronchial tubes. The local forms of chronic bronchitis become the source of development of chronic diffuse bronchitis, when the whole bronchial tree is struck. Thus the wall of bronchial tubes becomes thickened, surrounded by the layers of connecting tissue, sometimes there is deformation of bronchial tubes. There
can be sacciform or cylinder bronchoectasia at the protracted motion of bronchitis. Microscopic changes in a bronchial tube at a chronic bronchitis are various. In one cases the phenomena of chronic catarrhal inflammation prevail with atrophy of mucus membrane, cystous transformation of glands, metaplasia of integumentary prismatic epithelium in laminated pavement epithelium, by the increase of number of goblet cells; in other - in a wall to the bronchial tube and especially cellular inflammatory infiltration and excrescence of granulation tissue which knobs in a space of bronchial tube as a polypus in a mucus membrane acutely - polypos chronic bronchitis is determined. At ripening of granulation tissue and excrescence in a wall a muscular layer atrophies the bronchial tube of connecting tissue and a bronchial tube is deformed- deforming chronic bronchitis. At a chronic bronchitis the drainage function of bronchial tubes is violated, which conduces to the delay of their space in lower departments, closing of space of shallow bronchial tubes and bronchiole and development of bronchopulmonal complications, such as atelectasis (an active slump of respirator department of lungs is a result of obturation or compression of bronchial tubes), obstructive emphysema, chronic pneumonia, pneumofibrosis.

3. **Bronchogenic cancer of lung.** According to classification of cancer of lungs they distinguish: radical (central) which grows from rotting, or other parts of segmental bronchial tube. On preparation node of grey color without clear scopes, which narrows a clearance of bronchial tube is observed. Excrescence of tumour can cause the origin of the complete closing of space of bronchial tube and lungs. According to the hystological structure it is distinguished: planocellular (epidermoid) cancer; an anaplastic cancer is undifferentiated (small-celled, large cell); adenocarcinoma.

4. **A central cancer of lung.** On preparation a node of grey color without clear scopes is observed. According to classification of cancer of lungs they distinguish: radical (central), grows from rotting or initial part of segmental bronchial tube. By character of growth they distinguish: exophytic (endobronchial) and endophytic (exo- and peribronchial). According to the macroscopic picture of cancers of lungs there are: massive or nodal, polypoid, little cancer of Penkost, branchy, pneumo-like. On this preparation it is a nodal form of cancer of lungs.

5. **Hemorrhagic tracheobronchitis.** On preparation change on a mucus of trachea and bronchial tubes as plural hemorrhages, mucus hyperemic, faded are observed. At a acute bronchitis the mucus membrane of bronchial tubes becomes plethoric and hydropic, shallow hemorrhages, ulcers, are possible. In a space of bronchial tubes in most cases there is much mucus. In the mucus membrane of bronchial tubes the different forms of catarrhal inflammation develop with the accumulation of mucus, purulent, compound exudate.
Hemorrhagic tracheobronchitis is a acute inflammation of bronchial tubes - can be an independent disease or display of number of illnesses, lobular pneumonias, chronic glomerulonephritis with kidney insufficiency (acute uremic bronchitis). Acute bronchitis, as a rule, has more difficult course for children. Clinically it appears as a cough, dispnoea and tachypnoea.

Complications of acute bronchitis are often related to violation of drainage function of bronchial tubes of that further aspiration of the infected mucus in the distal parts of bronchial tree and development of inflammation of pulmonary tissue (bronchipneumonia). At panbronchitis and panbronchiolitis passing of inflammation is possible not only on peribronchial tissue but also on interstitial tissue of lungs (peribronchial interstitial pneumonia).

6. Croupous pneumonia. Croupous pneumonia is an acute infectiously allergic disease at which one or a few lobes of lungs are damaged (lobar pneumonia), fibrinogenous exudate (fibrinogenous, or croupous pneumonia) appears in alveoles, and on a pleura is fibrinogenous thin deposit (pleuropneumonia). All names of illness are synonyms and represent one of features of disease. Croupous pneumonia is examined as an independent disease. Adults are ill mainly, rarely -children. The causative agent of illness is pneumococcus I, II, III and the IV types. Pneumococcus pneumoniae most often affects people in age from 20 to 50, while partial pneumonia, caused Klebsiella, usually affects senior age, diabetics and alcoholics. On occasion croupous pneumonia is caused by diplobacillus of Fridlender.

Acute beginning of croupous pneumonia among complete healthy and in absence of contacts with patients, as well as the carrier of pneumococcus, by healthy people, allows to bind its development to the self-infection. However in pathogeny of croupous pneumonia a large value is taken by sensibilisation of organism to pneumococcus and overcooling.

Complication. It is distinguished lung and outlung complication of croupous pneumonia. Pulmonary complications are developed in connection with violation of fibrinolytic function of neutrophils. At insufficiency of this function mass of fibrin organizations are added in alveoles, that germinate granulation tissue, which, ripening, grows into mature fibred connecting tissue. This process of organization is named carnification (from carno- meat). A lung grows into dense fleshy airless tissue. At surplus activity of neutrophils is possible development of abscess and gangrene of lungs. Tacking of pus to the fibrinous pleuritis conduces to empyema of pleura. At lymphogenic generalization arise up purulent mediastinitis and pericarditis, at hematogenic-peritonitis, metastatic abscesses in a cerebrum, purulent meningitis, acute ulcerous endocarditis or polypoulcerosa, purulent arthritis etc. The modern methods of treatment of croupous pneumonia changed its clinical and morphological picture acutely, that allows to talk about induced pahtomorphism of this illness.
9. **Tuberculoma.** Tuberculoma is a form of the secondary tuberculosis which arises up as an original phase of evolution of infiltrative tuberculosis, when perifocal inflammation resolves and remains focus of caseose necrosis, surrounded by capsule. Tuberculoma achieves at 2-5 sm in a diameter, located in I or the II segment, more frequent in the right side. Quite often at a X-ray research as a result of the well enough outlined scopes it is thought as a peripheral cancer of lungs by mistake.

10. **Microfocal hematogenic tuberculosis.** Generalized hematogenic tuberculosis. Is met rarely, presents by itself the most difficult form of disease with the even rush of tubercular humps in many organs. In one cases in all of organs necrotic focuses are formed without proliferative or with ill-defined exudative reaction (so-called necrotic type of generalized tuberculosis). It is 1) acutest tubercular sepsis. In other cases shallow miliary productive humps appear in all of organs. This form is marked as 2) acute disseminated miliary tuberculosis. It often ends with meningitis. Finally, on occasion observed 3) acute disseminated largefocus tuberculosis from which usually weakened patients are suffering and it is characterized by spreading on the different organs of large (by a diameter of 1 sm) tubercular focus. In every case of generalized hematogenic tuberculosis it is necessary to find a focus which is the source of spreading; usually it is the focus of period of primary infection, that was not wholly healed in a lymphatic node, genitals, bone system and etc

At acute miliary tuberculosis lungs are exaggerated, fluffy, in it like grains of sand shallow humps amount of which especially big in the overhead segments are found. Quite often this form of tuberculosis ends with meningitis. At chronic miliary tuberculosis possibly scarring of humps and development of proof emphysema of lungs, in this connection loading on a heart is increased and the hypertrophy of right ventricle develops (pulmonary heart).

11. **Haemorrhagic pneumonia.** Macroscopically plural focuses of hemorrhages appear in lungs. Interstitial inflammation is hystologically determined, in exudate lymphocytes, macrophagocytes and plasmatic cells are found. In space of alveolas and bronchioles is plenty of hyaline membranes which appear from fibrinous exudate. The space of alveolas is often remained free. A virus of flu can be reason of acute quick as lightning haemorrhagic pneumonia which can cause rapid death of organism.

12. **Pneumosclerosis.** In lungs areas of pneumofibrosis and carnification are observed. Along lymphatic vessels in interlobular partitions, in perivascular and peribronchial tissue chronic inflammation and fibrosis develop. Inflammatory and sclerotic changes appear in the walls of shallow and larger vessels, up to obliteration of space.

13, 8, 33. **Macrofocal bronchopneumonia.**
14. Focus bronchopneumonia.
15. Core bronchopneumonia.
17. Microfocal bronchopneumonia. Bronchopneumonia is inflammation of lungs which develops in connection with a bronchitis or bronchiolitis (capillary bronchitis). It has focus character, can be the morphological manifestation of both primary (for example, at respiratory viral infections) and secondary (as complication of some diseases) acute pneumonias. The basal parts of lungs are usually struck on either side, which on a section have a grey or grey-red color. Inflammatory changes in tissue of lungs can be shown at easy pressure on the affected area: a normal lung at pressure does not render considerable resistance (as a sponge), while at pneumonia small resistance is determined. At histological research typical acute inflammation with exudation is determined.

Bronchopneumonia is characterized by the presence of plural focuses of defeat of pulmonary tissue, located around inflamed bronchial tubes or bronchioles with spreading of process on surrounding alveolas. This type of pneumonia most often affect children, old and sick with weakened resistance (for example, patients with malignant neoplasms, cardiac insufficiency, chronic kidney insufficiency and other). Bronchopneumonia also can develop as complication of acute bronchitis, mucoviscidosis and other diseases which are characterized by the obstruction of respiratory tracts. Violation of bronchial secretion, which is often observed in a postoperation period, also causes development of bronchopneumonia.

Etiology. Usually causative agents are microorganisms, especially for persons with immunodeficiency, which for healthy people do not cause development of similar disease. They are usually staphylococcus, streptococcus, Haemophilus influenzae, Escherichia coli and fungi. Patients often suffer from septicemia and toxemia, that appears with fever and violation of consciousness. Bronchopneumonia develops also at the influence of chemical and physical factors, that allows to distinguish uremic, lipid, dust, radiation pneumonia.

Pathogenesis. Development of bronchopneumonia is caused by the acute bronchitis or bronchiolitis, thus inflammation more frequent spreads on pulmonary tissue intrabronchialy (by a descending way, usually at a catarrhal bronchitis or bronchiolitis), rarer peribronchialy (usually at a destructive bronchitis or bronchiolitis). Bronchopneumonia arises up by hematogenic way which is met, when an infection generalize (septic pneumonias). In development of focus pneumonia a self-infection has a large value at aspiration-aspiration pneumonia, stagnant phenomena in lungs- hypostatic pneumonia, aspiration and neuroreflectory disorders- postoperation pneumonia. In spite of certain differences depending on reason of bronchopneumonia, morphological changes have a number of general lines. At any etiology acute bronchitis or bronchiolitis lies in basis of bronchopneumonia, which shows up in different forms of catarrh (serous, mucus, purulent, mixed). Thus a mucus membrane
becomes sanguineous and plethored, the producing of mucus by glands and globet-like cell is acutely increased; the integumentary prismatic epithelium of mucus membrane desquamates, that causes damage of mucocellular mechanism of cleaning of bronchial tree. The walls of bronchial tubes and bronchioles become thick due to edema and cellular infiltration. More frequent there is panbronchitis and panbronchiolitis in the distal parts of bronchial tubes, and in proximal - endomesobronchitis. Edema and cellular infiltration of wall break the drainage function of bronchial tubes, which favours to aspiration of the infected mucus into the distal parts of bronchial tree, fleeting expansions of space of bronchial tubes - transitor bronchoektasia can appear at cough inciment. The focus of inflammation at bronchopneumonia usually arise up in back and back-low segments of lungs - II, VI, VIII, IX, X. They are of different sizes, dense, on a cut, grey-red. Depending on the size of focus they distinguish miliary (alveolitis), acinar, lobular, lobular confluent pneumonia, segmental and polisegmental bronchopneumonia. In alveolas the accumulation of exudate with the admixture of mucus, a lot of neutrophils, macrophages, red corpuscles, cast-off alveolar epithelium are observed; the small amount of fibrin is sometimes determined. Exudate is spread unevenly: in one alveolas is a big amount of it, in other - little. Interalveolar partitions are saturated with cellular infiltrate.

16. **Tuberculosis of kidneys.** Tuberculosis of kidneys is usually one-sided, more frequent it affects young people in the period of the sexual ripening, and also in senile age. Early focus arises up in a cortical layer. At progress of process they appear in papilla of pyramids; here a destructive process begins with formation of cavity. Outside of cavity interstice of kidney tissue is infiltrated with lymphocytes, histiocye with the admixture of epithelioid cells- a chronic interstitial nephritis. Closing of the space of ureter by caseose masses lead to development of pyonephrosis. Gradually a specific inflammatory process passes to the urinary tracts, urinary bladder, prostate, in addition to testicle. For women- mucus membranes of uterus, uterine tube, rarely ovaries are damaged.

18. **Focus pneumonia on a background of anthracosis.** The focus of inflammation and focus of deposit of coal dust appear in lungs (focus of anthracosis).

19. **Carcinomatosis of pleura.** On pleura shallow nodes of grey color are observed. It is a type of metastasis of lung cancer by implantation.

20. **Carcinomatosis of lung.** In parenchyma of lungs shallow focus of grey color, that characteristically for metastatical character of spreading of cancer of lungs appeares. At metastasing the first lymphogenic metastases arise up in peribronchial and bifurcational lymphatic nodes, then - neck, retroperitoneal.
Among hematogenic metastases for a cancer of lungs are characteristic metastases in liver, cerebrum, bones (especially in a vertebra) and adrenal gland.

21. **Pulmonary heart.** On macropreparation an increase of sizes of heart and hypertrophy of right ventricle, bulge of its wall is 1,0 см (in a norm thickness of wall of right ventricle 3) is observed. The chronic diseases of lungs (chronic pneumonia, chronic obstructive bronchitis, bronchial asthma and other) are favourable in the origin of pulmonary heart, there is hypertension of small circle of circulation of blood, which causes the hypertrophy of right heart (pulmonary heart). To the cardiac insufficiency is joined pulmonary insufficiency which on the certain stage becomes the main illness.

22. **Bullous proximal emphysema of lungs.** Emphysema of lungs name a disease which is characterized by surplus maintenance of air in lungs and increase of their sizes (from greek. emphysao - blow up). Bullous emphysema is not a separate type of emphysema, but term which shows a presence of bulls of size more than 10 mm in a diameter. We can meet bulls at all four basic types of emphysema. Bulls are often develop, that causes development of spontaneous pneumothorax. Usually bulls are disposed on the apex of lungs subpleurally. Reduction of capillary microcirculation vessels takes a place on the limited area of lungs, that is why there is not high blood pressure of small circle of circulation of blood at perifocal emphysema. Lungs are enlarged, cover the edges of front mediastinum, exaggerated, pale, soft, not deflated, cut with a crunch. From space of bronchial tubes the walls of which are thickened, mucus-purulent exudate is squeezed out. The mucus membrane of bronchial tubes is sanguineous, with inflammatory infiltrate, plenty of goblet cells; the uneven hypertrophy of muscular layer is marked, especially in shallow bronchial tubes. The stretch of wall of acinus conduces to the stretch and thinning of elastic fibres, expansion of alveolar motions, change of alveolar partitions. The walls of alveolas are thinned and flattened, interalveolar pores are broaden, capillaries become empty. Conducting respiratory bronchioles are broadened, alveolar sacs are shortened. Hereupon there is the acute diminishing of area of interchange of gases, a ventilation function of lungs is violated. A capillary network is reduced in respirator part of acinus, that causes formation of alveolar-capillary block. Collagenic fibres overgrow in interalveolar capillaries, an intracapillar sclerosis develops. Thus there is formation of the new not quite typically built capillaries, which has a adaptive value.

23. **Chronic tubercular cavern.** Chronic form. Arises up, foremost, in those cases, when at a primary affect which had begun, is healed, an inflammatory specific process in the lymph glandular component of primary complex accepts the progress course with the alternation of ictuses and calming down. Sensibilization of organism comes – its sensitiveness rises to the different sort
of the unspecific influencing. Reactivity of organism clinically is manifested by tuberculin skin test and appearance in tissues and organs of paraspecific changes, which are cellular reactions - different mesenchymal cellular reaction as diffuse or node proliferation of lymphocytes and macrophagocytes, hyperplastic processes in hematopoietic tissue, fibrinoid changes of connecting tissue and walls of arteriols in organs, disproteinosis, sometimes development of amyloidosis. About chronic primary tuberculosis it is talked also, when a primary pulmonary cavity appears.

24. Bronchoectasis. Bronchoectasis is characterized by stable expansion of bronchial tube or bronchiole. Bronchoectasis can be inherited and acquired. Inherited bronchoectasis is met comparatively rarely and develops in connection with damages of forming of bronchial tree. The hystological manifestation of inherited bronchoectasis is a disorderly location in its wall of structural elements of the bronchial tube. Inherited bronchoectasis appears usually at suppuration of their maintenance. Bronchoectasis practically always met at illnesses which are characterized by considerable inflammation and obstruction of airway. Even at inherited pathologies (for example, at the syndrome of Kartagener, or syndrome of immobile cilia) arising of bronchoectasis almost always takes a place at inflammation which causes destruction of pulmonary tissue and subsequent fibrosis. Most often bronchoectasis develops in lower lobes. A bronchial secret, which creates conditions for development of microorganisms, accumulates in bronchoectasis. For such patients basic symptoms is a permanent cough and abstraction of sputa with an unpleasant smell, sometimes with the admixtures of blood. An infectious process from bronchial tubes can spread locally or systematically. Bronchoectasis can be cylinder, sacciform or fusiform; its form does not have prognostic or etiologic value. Intrabronchial pressure which rises during cough shoves influencing on the changed bronchial wall at chronic inflammation, conduces to its thrusting out and development of bronchial tube broadens. Bronchioles extended on the basis of inflammation are named bronchoectasis. They are usually plural, surface of cut lungs here is hexagonal, such lung is named cellular, as it reminds bee’s cells. The cavity of bronchoectasis is covered by prismatic epithelium, but quite often laminated pavement epithelium which arose up as a result of metaplasia. In a wall there is chronic inflammation bronchoectasis, elastic and muscular fibres on considerable part are blasted and replaced connecting tissue. In a cavity there is purulent contents of bronchoectasis. Cavity of bronchoectasis pulmonary tissue is changed acutely, in it there are focuses of inflammation (abscesses, areas of organization of exudate), field of fibrosis. There is obstructive emphysema which conduces to hypertenion in the small circle of circulation of blood and hypertrophy of right ventricle of heart (pulmonary heart). According to this patients’ hypoxia appears with subsequent violation of trophic of tissues. Very characteristic is a bulge of tissues of nail phalanxes of fingers of hands and feet: fingers acquire
type of drumsticks. All the complex of pulmonary and extrapulmonary changes in case of bronchoectasis is named bronchoectatic illness.

Complications of bronchoectasis are: pneumonia; empiema of pleura; septicemia; meningitis; metastatic abscesses, for example, in a brain; secondary system amyloidosis.

25. Ghon's focus. Cicatrization of focuss of primary complex. At localization of primary tubercular affect in lungs perifocal inflammation is resolved at first. An exudative tissue reaction will be replaced by productive: around focus of caseose pneumonia a billow appears which is made by epithelioid and lymphoid cells, that as though separates a focus from surrounding pulmonary tissue. Around primary affect a capsule is formed, the external layers of which consist of areolar tissue with the presence of shallow vessels, surrounded by lymphocytes. There is the formation of capsule (encapsulation). Caseose mass is gradually dehydrated, become dense and calcificated (petrifaction). In course of time in the way of metaplasia bone beams with cells in interbeam spaces form. This way petrificated primary focus transforms into ossified. A primary focus which healed with such way, got the name of Ghon's focus, on the name the Czech pathologist which described it firstly.

26. Acute abscess of lungs. Abscess of lungs has pneumoniogenic origin. Pneumoniogenic abscess of lungs arises up as complication of pneumonia of any etiology, more frequent at staphylococcus and streptococcus. Necrosis of the inflamed pulmonary tissue preceeds the purulent melting of focus, before which suppuration of focus of pneumonia takes place. Molten necrotic suppurative mass with a sputum is excreted through bronchial tubes, the cavity of abscess appears. In a pus and in the inflamed pulmonary tissue plenty of pyogenic bacterias appears. Acute abscess is localized more frequent in II, VI, VIII, IX and X segments, where the focuses of acute bronchopneumonias are usually located. In most cases an abscess unites with the space of bronchial tubes (drainage bronchial tubes) through which pus is excreted with sputum. Gangrene of lungs is the most severe type of complications of acute destructive processes of lungs. It arises up after pneumonia or abscess of lungs of any genesis at joining of putrefactive microorganisms. Pulmonary tissue is attached with moist necrosis, becomes grey dirty, gives out a bad smell. Gangrene of lungs usually leads to death.

27. Fatty dystrophy of liver. Fatty dystrophy of liver appears with the acute increase of contants and change of composition of fats in hepatocytes. In the cells of liver the granules of lipids (pulverulent obesity) appear at first, then shallow drops (smalldropped obesity) which in future met in large drops (largedroped obesity) or in one fatty vacuole, which fills all of cytoplasm and pushes back a nucleus on periphery. Macroscopically a liver at fatty dystrophy is enlarged, anaemic, flabby, has a yellow or ochreous -yellow color, with fat
brilliance on a cut. At a cut on the blade of knife and surface of cut the fat is visible.

28. Acute abscess of lungs. To the acute destructive processes lungs belong an abscess and gangrene of lungs. Abscess of lungs can have both pneumoniogenic and bronchogenic origin.

Bronchogenic abscess of lungs appears at destruction of wall with bronchoectasis and transition of inflammation on nearby pulmonary tissue with subsequent development of necrosis, suppuration and formings of cavity – abscess in it. The wall of abscess is presented both by bronchoectasis and compact pulmonary tissue. Bronchogenic abscesses of lungs usually are plural. Acute abscess of lungs sometimes heals spontaneously, but more frequent accepts chronic motion.

29. A chronic abscess of lungs. A chronic abscess of lungs usually develops after acute and localized more frequent in II, VI, IX and X segments of right, rarer at left lungs, that in those departments of lungs, where usually there are focuses of acute bronchopneumonia and acute abscesses. A structure of wall of chronic abscess of lungs does not differ from the chronic abscess of other localization. In a process lymphatic drainages of lungs are brought into process. After direction of outflow of lymph from the wall of chronic abscess to the root of lungs whitish layers of connecting tissue which conduces lungs to fibrosis and deformation of tissue appear. A chronic abscess is the source of bronchogenic spreading of purulent inflammation in lungs. Development of the secondary amyloidosis is possible.

30. Metastases of cancer in lung. In lungs the great number of nodes of grey color and different sizes appears. They are the metastases of cancer.

31. Croupous pneumonia in the stage of grey hepatisation.

Lung on the cut has grey brown color, dense consistency with impositions of fibrin on a pleura.

Stage of grey hepatization. This stage can also last a few days and characterized by the accumulation of fibrin and destruction of white and red cells of blood in exudate. Lungs on a cut become grey brown and dense.

The classic chart of development of croupous pneumonia is sometimes violated - grey hepatization precedes red. On occasion the focus of pneumonia occupies central part of lobe lungs (central pneumonia), in addition, it can appear both in one or in other lobe (migrant pneumonia). The dystrophic changes of parenchymatous organs, their plethora belong to the general manifestations of croupous pneumonia, hyperplasia of spleen and marrow, plethora and swollen cerebrum. Lymphatic nodes of root lungs are enlarged, white-pink; at their hystological research the picture of acute inflammation is found.
25. (Case 4). **Fibrocavernous tuberculosis of lung.** Fibrocavernous tuberculosis arises up after acute tuberculosis in those cases, when a process accepts chronic motion. The wall of cavity is dense and has three layers: internal - pyogenic, rich of leucocytes that disintegrated; middle is a layer of tubercular granulation tissue; external - presented by dense fibred connecting tissue. An internal surface is unequal, it is an obliterated bronchial tube or thrombosed vessel with the focus of necrosis. A cavity can occupy one or both segments. Bronchoectasis appears alongside. A process gradually spreads in apiko-caudal direction, spreads from apikal segments to low through a bronchial tube, occupying new areas of lungs. The oldest changes at fibrocavernous tuberculosis are observed in apikal parts of lungs, and the most fresh – in low. In course of time a process passes through bronchial tubes into an opposite lung. Reason of deaths of sick with pulmonary tuberculosis: pulmonary-cardiac insufficiency; pulmonary bleeding; secondary amyloidosis (uremia).