1. **Cancer of pancreas with metastases in a spleen.** The cancer of pancreatic gland develops as from the epithelium of channel (adenocarcinoma) so from the epithelium of acinuses parenchima (acinar or alveolar cancer). At preparation we see a tumour in the pancreas of light-grey color without clear verges, a capsule is absent, infiltrative growth is characterized. In a spleen we see the knots of cancer of pancreas - they are metastases. A cancer metastases with lymphogenic way into lymphonoduss located next to the head of gland. It can give and hematogen metastases into liver, lungs and other organs.

2. **Adenoma of pancreas.** In pancreas islets of grey color with clear contours, expansive growth are observed, or on this micropreparation we see benign tumour – adenoma of pancreas. The tumours of islet vehicle of pancreas are the tumours of the APUD-system, or apudomes. Adenomas from the cells of islets name insulomas; they are hormonal active. Three types of insulomas are distinguished: 1) insuloma from β-cells, which products insulin (β-insuloma); 2) insuloma from the α-cells products glucagon (α-insuloma); 3) insuloma from g-cells - synthesizes a gastrin. β-insuloma proceeds with the displays of hyperinsulinism and glucopenia; α-insuloma - paroxismal or permanent hyperglycaemia; C-insuloma - predetermines development of ulcers in a stomach and duodenum (ulcerogenic insuloma), that manifested with the syndrome of Zolinger-Elison. The malignant variants of insulomas name malignant insulomas; which can keep hormonal activity.

3. **Yellow dystrophy of liver.** This is an acute disease which is characterized by making progress massive necrosis of liver and hepatic insufficiency. It can arise up at exogenous and endogenous intoxications. In the first days a liver becomes insignificantly larger, dense or flabby consistencies, light- yellow color as on-the-surface so on a cut. Then it diminishes gradually, becomes limp, its capsule wrinkles, on the section its parenchima acquires a yellow-grey, clay kind. At microscopic research in a liver they find fatty dystrophy of hepatocytes in the center of lobules, necrosis with autolitic destruction.

4. **Liver at the malformed imperforation of bilious ways.** Influence of pathogenic factor during pregnancy in the period of embriogenesis results in the damage of tissues of liver during ripening and differentiation of tissues. A liver is diminished, yellow-green color. Hepatorenal deficiency intoxication is a reason of death.

5. **Compound cirrhosis of liver.** The compound cirrhosis has the signs of both postnecrotical and portal cirrhosis. Extrahepatic signs of cirrhosis of liver: icterus; hemorragic syndrome; hydroperitoneum; splenomegaly. Immune complex and calcinal metastases appears at development of hepatonephric syndrome. In a cerebrum there are dystrophic changes of neurons. Hepatic deficiency, portal hypertension and hepatocellular cancer are the most important complications. As a result of hepatic deficiency there is the decreasing of synthesis of albumens, factors of hemopcoaglation and other proteins of blood. Destruction of endogenous products, such as hormones, itrogen-containing substances is violated. Encephalopathy develops as a result of violation of detoxication. Kidney deficiency often develops (hepatonephric syndrome). As a result
of violation of destruction of steroids repeated aldosteronism develops, which shows up the delay of fluid and ions of natrium, and gynecomastia at men develops as a result of hyperestrogenaemia. Also as a result of hyperestrogenaemia "vascular starlets" develop on a skin.

6, 37. Micronodular cirrhosis of liver. A portal cirrhosis is characterized by homogeneity of microscopic picture – slim-bulked conective- tissue net and small size of unreal lobules. At this cirrhosis microscopically more frequent all the signs of chronic inflammation and fatty dystrophy of hepatocytes appear. A liver is little macroscopically, dense, grainy or finely-hilly. A portal cirrhosis develops slowly (during many years), more frequent - at chronic alcoholism. A primary billiar cirrhosis is the true portal cirrhosis. A liver at a primary billiar cirrhosis is megascopic, dense, on a cut grey-green, surface of its smooth or fine-grained. At the secondary billiar cirrhosis, caused by the obstruction of bilious ways by a stone, tumor, or infection of bilious ways and development of chalangitis (cholangiolytical cirrhosis), a liver is megascopic, dense, green color, on a cut with extended and the channels overcrowded by a bile.

7, 15. Cancer of pancreas. On preparation we see a tumour in the pancreas of light- grey color without clear verges, a capsule is absent, infiltration growth is characterized. A cancer knot germinates channels of pancreas, and then bilious duct. It can arise up in any part of gland, but more frequent – pancreatic gland head. The tumours of body and tail of pancreas achieve largenesses as long time that neither at liver not at pancreas disorders of function are caused. A tumour causes disorders of pancreas and liver (cholangitis, icterus).

8. Stone of gall-bladder. Stone or concrements (from latin concrementum is grafft), are dense formations which appear from composition of secret or excret and freely lie in a gall-bladder or conclusions channels. Reasons of formation of stone varies and concerne by both general and local factors. The metabolic disorders belong to the general factors. It is well-known connection of cholelithiasis with general obesity and atherosclerosis, urolithiasis - with a gout, oxaluria. Local factors: disorder of processes of secretion and resorbtion in an organ (thickening of secret), stagnation of secret is accompanied by the increase of concentration of dense masses; inflammative processes (desquamated cell, leucocytes, mucus, bacteria can become organic basis of stone; at inflammation possible change of рН in an alkaline side).

10. Calculary cholecystitis. There are stones of different size in space of gall-bladder. The disorder secreion, as well as stagnation of secret, cause increasing of concentration of substanaces, from which stone are built that further them to precipitate from solution and densitiing of secret .In case of inflammation the albuminous substances appear in a secret, that creates an organic (colloid) matrix, in which salts (crystalloid component) are deposited. Afterwards a stone and inflammation quite often become complement to each other, that determines progress of formation of stone.

11. Empyema of gall-bladder on a background cholelithiasis. On preparation enlarged gall-bladder, in space there is a bile with a purulent exsudate. As a result of pressure of stone on the wall of gall-bladder there can be its necrosis - bedsore, that can cause the perforation, joints, fistules, empyema. Stones often are reason of inflammation, as injure tissue, create the gate of infection, cause stagnation and and it is base of cholelithiasis.
12. Nodule cancer of liver. In a liver there is the knot of grey color without clear verges, a capsule is absent, character of growth is infiltrative. More frequent it is hepatocellular cancer on the histological structure.

13. Metastases of cancer of stomach into liver. In a liver there are knots grey-rose color of different sizes. They macroscopically differ from the structure of parenchima of liver, and microscopically the cells of tissue of different level of differentiation appear and are presented by glandular structures with the signs of cellular atypism.

14. Fatty hepatosis. On preparation liver is megascopical, littleblood, has a yellow or ochre-yellow color, with fat shine on a cut. At a cut on a knife-blade and surface of cut raid of fat. Is observed Reasons fatty dystrophy vary: anoxaemia (tissue hypox). In the conditions of hypox the parts of liver, which are in functional tension, suffer from all things; that is why fatty dystrophy so often meets at the diseases of the cardiovascular system, chronic diseases of lungs, anaemias, chronic alcoholism etc.; infections (diphtheria, tuberculosis, sepsis); intoxications (phosphorus, arsenic, chloroform, alcohol), illnesses which cause violations of metabolism; avitaminoses and one-sided (with insufficient maintenance of albumens) nutrition. Microscopic signs of fatty dystrophy: any fat that is in tissues dissolves in solvents which are used for colouring of tissues for microscopic research. That is why at colouring of tissue haematoxiline and eosine of cell on the earliest stages of fatty dystrophy have a pale and foamy cytoplasm. As far as the increase of the fatty including small vacuoles appear in a cytoplasm.

16, 32. Billiar cirrhosis of liver. On preparation morphological signs of cirrhosis: violation of lobular structure of liver, wrinkling and deformation of organ are observed. Microscopically: dystrophy of hepatocytes (hydroptical, bladderous, fatty) is determined; necrosis of hepatocytes; presence of knots of regenerators - unreal lobules (strengthening of regeneration, presence of mytoses of hepatocytes); diffuse fibrosis (excrescence of connecting tissue).

17. Chronic calculary cholecystitis. The presence of stone in a gall-bladder results the formation of inflammatory process in the wall of gall-bladder. The direct mechanism of formation of stone consists of two processes: formation of organic matrix and cristization, thus each of these processes in certain situations can be primary. Gall-stones can be cholesterine, pigmental, limestones or cholesterine-pigment-calcificous (compound, or combined stone). The size of stone is different. There are enormous stone and microlithiums. They can be single and plural.

18. Varicose enlargement of the veins of the mucous membrane of the gullet. It is seen: the enlarged veins in submucous layer and the atrophied mucous membrane. Causes: disturbance of blood circulation in the liver under portal cirrhosises. Consequently porto-cavalous anastomosis are opened. Hemorrhages often occur from such veins.

19. Protein dystrophy of liver. An example of parenchymal disproteinosis with the deposition of protein granules in hepatocytes. The liver is enlarged, infirm, grey-yellow. Causes: hypoxia, intoxication, infectious diseases (virus hepatitis), metabolic-endocrinuous disturbances. Outcomes: the function is decreased, in the case of progression of the destructive alterations and afterwards cirrhosis is developed.
25. **Cancer of liver on a background a calculary cholecystitis**. On preparation megascopic gall-bladder filled by stone is observed. Near in the parenchima of liver takes place knot of grey color without clear verges with infiltrative character of growth. On microscopic structure more frequent it is hepatic-cellular (hepatocellular) cancer. It develops as one large knot, which takes the almost whole lobule of liver (knotted form) or shallow knots which are disseminated in tissue of liver (diffuse form). A tumour is built from atypical hepatocytes, which forms tubules, acinuses or trabecules (tubular, acinar, trabecular, solid cancer).

26. **Toxic dystrophy of liver (Stage of red dystrophy)**. This is an acute disease which is characterized by making progress massive necrosis of liver and hepatic insufficiency. During the third week of illness a liver progressively diminishes and acquires the red colouring, it is stage of red dystrophy. Lipoprotein detritus is subject of fagocytosis, resolves partly, resorbed, as a result reticular stroma uncoveres with the sharply extended bloods vessels and sinusoids. At massive necrosis of liver there is an icterus, hyperplasia of periportal lymphatic knots and spleen, plural dermatorrhagias, mucus and serosal membranes, lungs, necrosis of epithelium of kidneys, dystrophic and necrobiotic changes in a pancreas, myocardium and CNS. Patients die from acute or chronic renal insufficiency (hepatonephric syndrome). The postnecrotic cirrhosis of liver can be investigation of toxic dystrophy of liver.

27. **Cyst of spleen.** In a spleen the cavity which is filled by a transparent liquid is observed. Its capsule is presented by connecting tissue. This benign neoplasm can be found at the section, during life it can give no clinical symptoms.

28. **Hypertrophy of the spleen. (Splenomegaly)**. The spleen is enlarged in 3-4 times, dark-cherrish colour, dense. These changes appears under the diseases of liver. The enlargement of spleen is a result of compensatory hypertrophyc changes.

   **Outcomes**: the function is increased, it becomes an organ of sanguification, sometimes it may be a rupture at the insignificant injury.

29. **Heart and kidney at a DVC- syndrome.** There is the damage of heart and kidneys At a DVC-syndrome. Kidneys are megascopic, its surface is pale with microfocal hemorrhages. There are the dystrophic and necrotizing changes in the parenchima of kidney. Microscopically in the parenchima of kidney appears stasis, hemorrhages, formations of blood clots in a microcirculation, dystrophy and necrotizing changes of epithelium of tubules of kidney. A kidney on a cut has a pale-grey color cork layer and stagnant dark - red color cerebral layer. Lethal outcome can develop in hard cases. Small haemorrhagies are found under the epicardium.

30. **Macronodular cirrhosis of liver.** Macroscopically a liver is diminished in sizes, dense, with large knots parted wide and deep furrows. It is characterized by early hepatargy (hepatic deficiency) and late portal hypertension. Such cirrhosis develops as a result of massive necroses of hepatic parenchima. Areas of necrosis are replaced by dense cicatrical tissue. For this cirrhosis approchement of portal triads and central veins is a pathognomical morphological sign. Unreal lobules consist of newformed hepatic tissue and contain the great number of polynuclear hepatic cells. Often there is the expressed cholestasis proliferation of cholangiols.
31. **Hydrocholecystis on a background cholelithiasis.** On preparation we see a megascopic gall-bladder in space of which there is the fluid of light-yellow color. The form of stone repeats a cavity which he fills: round or oval stone Quite often they have smooth and shiny, grinding in to each other surfaces. The surface of stone can be not only smooth and shiny but also "rough". Sawing stone have the radial structure (crystalloid) in one cases, in other - stratified (colloid), in the third – stratified-radial (colloid-crystalloid). Stone can have not clinical manifestations and can appear by chance at the pathoanatomical section. However, they can entail and very serious consequences. In contempt of leadin gout of secret, they conduce to heavy complications of general character (for example, icterus at corking of general bilious channel).

35. **Chronic pancreatitis.** On preparation characteristic changes are observed: the parenchima of gland is diminished in size, excrescence of connecting and fatty tissue are observed, on a cut lobular structure is observed. Chronic development of disease results in the expressed sclerosis. Exocrine part of gland is replaced by fibrotic tissue, that is why there can be knots which at palpation can be percepted as tumours. Endocrine part usually is not damaged. At a chronic pancreatitis as a result of decline of exocrine secretion a malabsortbion syndrome often develops, especially of fats, that clinically appears by steatorrhea. Absorption of vitamins of Å, D, E, K is also violated.

36. **Alcoholic cirrhosis of liver.** On preparation violation of architecture of liver as fibrosis key regeneration is observed. Cirrhosis of liver is heterospecific disease; it is the final stage of diseases which result in the chronic damage of cells of liver. The amount of connecting tissue sharply grows and cells of liver not form acinuses and lobuls, but regenerate as knots which do not have the correct structure of lobule. The cirrhosis of liver is classified on morphological and etiologic principles. After morphological signs cirrhosis is classified on: littlenodule - knots to 3 mm in a diameter; largenodule are knots more than 3 mm in a diameter. Most often reason of littlenodule cirrhosis is alcoholism. At a largenodule cirrhosis, regardless of reason, there is the increase of risk of development of cancer of liver. On etiologic principle cirrhosises are divided on: viral (viral hepatitis B and C); alcoholic; at a hemochromatosis; autoimmune ("lupoid" hepatitis and primary billiar cirrhosis); as a result of obstruction of cholangial; at illness of Vilson.