

DISEASES OF THE CARDIOVASCULAR SYSTEM

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DISEASES OF THE CARDIOVASCULAR SYSTEM

□ The most significant diseases of the cardiovascular system are:

Atherosclerosis.

Arterial hypertension.

Ischemic heart disease.

Cerebrovascular diseases.

□ Atherosclerosis and related pathology - ischemic heart disease and cerebrovascular diseases - came in first place among the causes of death in economically developed countries

ATHEROSCLEROSIS

- ❑ Atherosclerosis (from the Greek “athere” - porridge and “sclerosis” - densification) is a chronic disease that occurs as a result of lipid and protein metabolism disorders, characterized by damage to the arteries of the elastic and muscular-elastic type in the form of focal sediment in the inner membrane of lipids and proteins and the reactive growth of the connective tissue.
- ❑ Atherosclerosis is the most common form of arteriosclerosis, reflecting a violation of the metabolism of lipids and proteins (metabolic arteriosclerosis).

ETIOLOGY OF ATHEROSCLEROSIS

- Atherosclerosis is a polyethological disease associated with the influence of various exogenous and endogenous factors (hereditary, environmental and food).

RISK FACTORS

- Age (frequency increases with age)
- Sex (more common in men).
- Family predisposition.
- Hyperlipidemia (hypercholesterolemia) and dyslipoproteinemia:
 - The ratio of atherogenic (LDL and VLDL) and anti-atherogenic (HDL) lipoproteins is 4: 1;
- Arterial hypertension.
- Smoking.
- Diabetes mellitus.
- Stressful situations.
- Hypodinamics, etc.

THEORIES OF THE PATHOGENESIS OF ATHEROSCLEROSIS

□ Lipoprotein theory

- Atherosclerosis should be considered as a reaction of the vascular wall to the appearance of modified lipoproteins of low (LDL) and very low (VLDL) density.
- The initiating moment in this case is the unregulated capture of the intact endothelium.
- A weak link in this theory is the inability to explain those cases of atherosclerosis, in which there is no atherogenic hyperlipidemia.

THEORIES OF THE PATHOGENESIS OF ATHEROSCLEROSIS

□ Theory of the reaction to damage

As an initial factor of atherogenesis, vascular damage that can be caused by a variety of factors: hyperlipidemia, mechanical stress, stress, immune mechanisms, toxins, viruses or other infectious agents, hemodynamic factors (hypertension, recurrent spasms, irregular turbulent blood flows in the area of branching of blood vessels, etc).

STAGES OF PATHOGENESIS

1. The appearance of modified lipoproteins, which are strongly captured by endothelial cells and transferred to the subendothelial space.
2. Damage to the endothelium (modified lipoproteins or any other factors - viruses, immune complexes, bacterial toxins, etc.).
3. Increased vascular permeability and the induction of plasma components, including lipoproteins, into the inner shell of the vessels.
4. Damaged endothelium expresses adhesive molecules, which results to adherence of platelets and monocytes, which turn into macrophages and produce IL-1, TNF α , PDGF.

STAGES OF PATHOGENESIS

5. SMCs under the influence of PDGF migrate into the inner shell of the vessels, proliferate, synthesize collagen, elastic fibers, proteoglycans, i.e. form the basis of an atherosclerotic plaque.
6. Lipoproteins in the inner shell of the vessels undergo peroxidation, form complexes with proteoglycans and are captured by macrophages, which are converted into xanthoma cells (a part of the xanthoma cells are formed from the SMC).
7. Subsequent changes in the plaque are associated with the formation of capillaries in it under the influence of growth factors, the involvement of other cellular elements (T- and B-lymphocytes, fibroblasts), necrosis of the central divisions, sclerosis, hyalinosis, calcification.

MORPHOLOGY OF ATHEROSCLEROSIS

- ❑ The main morphological expression of atherosclerosis is a plaque, narrowing the lumen of the artery, resulting in insufficient blood supply to the organs.
- ❑ Arteries of the elastic (aorta) and muscular-elastic type (large organ arteries) are usually affected.
- ❑ The atherosclerotic process passes through certain stages (phases) that have a macroscopic and microscopic characterization.

MACROSCOPIC STAGES

- Fatty streak.
- Fibrous plaques.
- Complicated lesions.
- Calcification (atherocalcinosis).

FATTY STREAKS

- ❑ Areas of yellow color (spots) that merge and form strips, while not rising above the surface of the intima.
- ❑ The earliest appear in the aorta on the back wall and at the point of divergence of its branches, later in the large arteries.

FATTY STREAKS



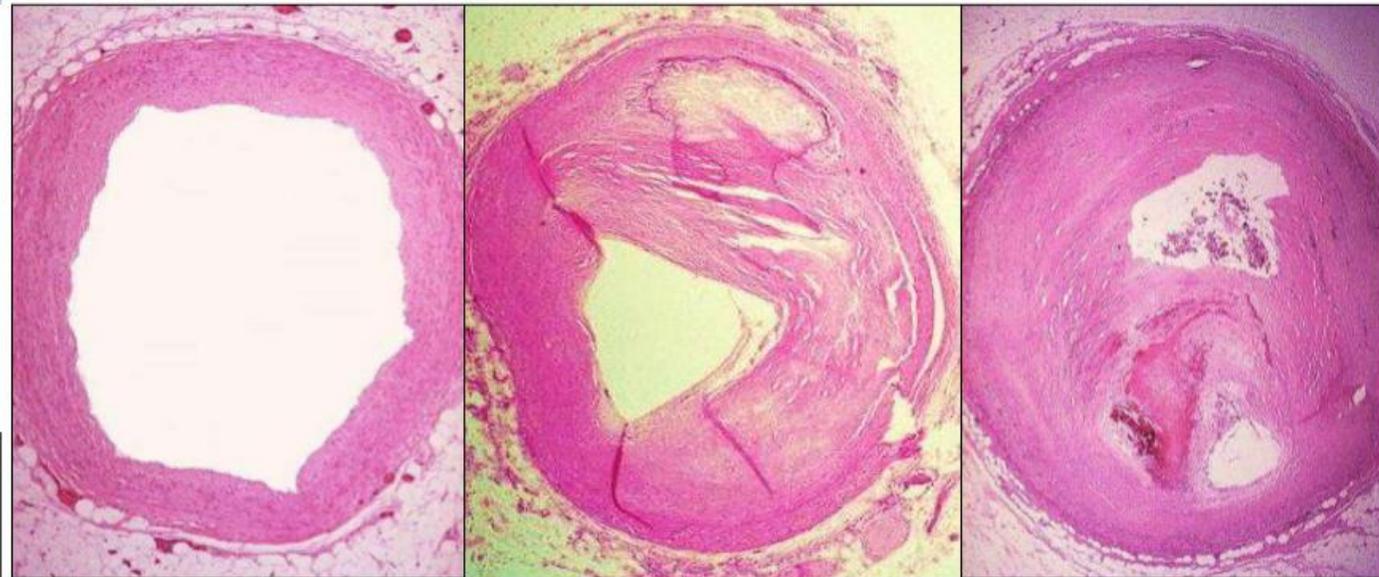
FIBROUS PLAQUES

- ❑ Dense oval yellow-white formations that rise above the surface of the intima.
- ❑ They often merge together, give their inner surface a tuberos appearance and lead to a narrowing of the lumen of the vessel (stenotic atherosclerosis).
- ❑ More often plaques are formed in the abdominal aorta, in the arteries of the heart, brain, kidneys, lower extremities, carotid arteries.
- ❑ Often, they undergo hemodynamic (mechanical) effects - in the branching and bending arteries.

FIBROUS PLAQUES



STENOTIC ATHEROSCLEROSIS



COMPLICATED LESIONS

- ❑ Destruction of plaque and its ulceration (atheromatous ulcer).
- ❑ Hemorrhages in the thickness of the plaque (intramural hematoma).
- ❑ Formation of thrombotic overlays at the site of ulceration of the plaque.
- ❑ The development of an infarction (with acute thrombosis), embolism with both thrombotic and atheromatous masses, the formation of an aneurysm of the vessel at the site of its ulceration, and arterial bleeding when the vessel wall is corroded by atheromatous ulcer are associated with complicated lesions.

COMPLICATED LESIONS



ATHEROCALCINOSIS

- ❑ The terminal phase of atherosclerosis, which is characterized by the deposition of calcium salts in the fibrous plaque, i.e. their calcification.
- ❑ Plaques acquire a stony density (petrification of plaques).
- ❑ The wall of the vessel at the place of petrification is sharply deformed.

MACROSCOPIC STAGES

- Different types of atherosclerotic changes are often combined, which indicates a wavy course of atherosclerosis.

MICROSCOPIC STAGES

- Prelipid;
- Lipoidosis;
- Liposclerosis;
- Atheromatosis;
- Ulceration;
- Atherocalcinosis

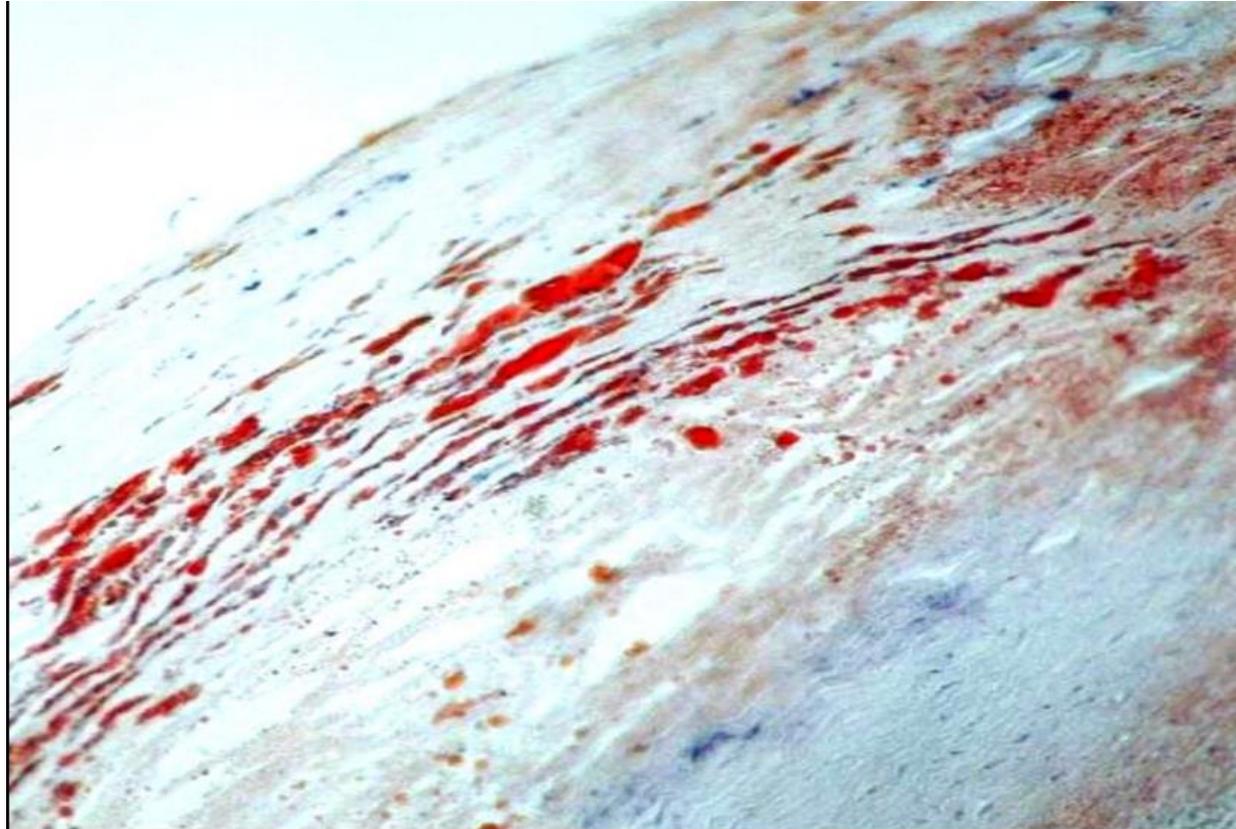
PRELIPID STAGE

- ❑ Increased permeability and damage to the inner shell of the vessel.
- ❑ Lipid droplets appear in endothelial cells.
- ❑ The appearance in the subendothelial layer of droplets of fat, plasma proteins, fibrin.
- ❑ Proliferation of SMC and macrophages.

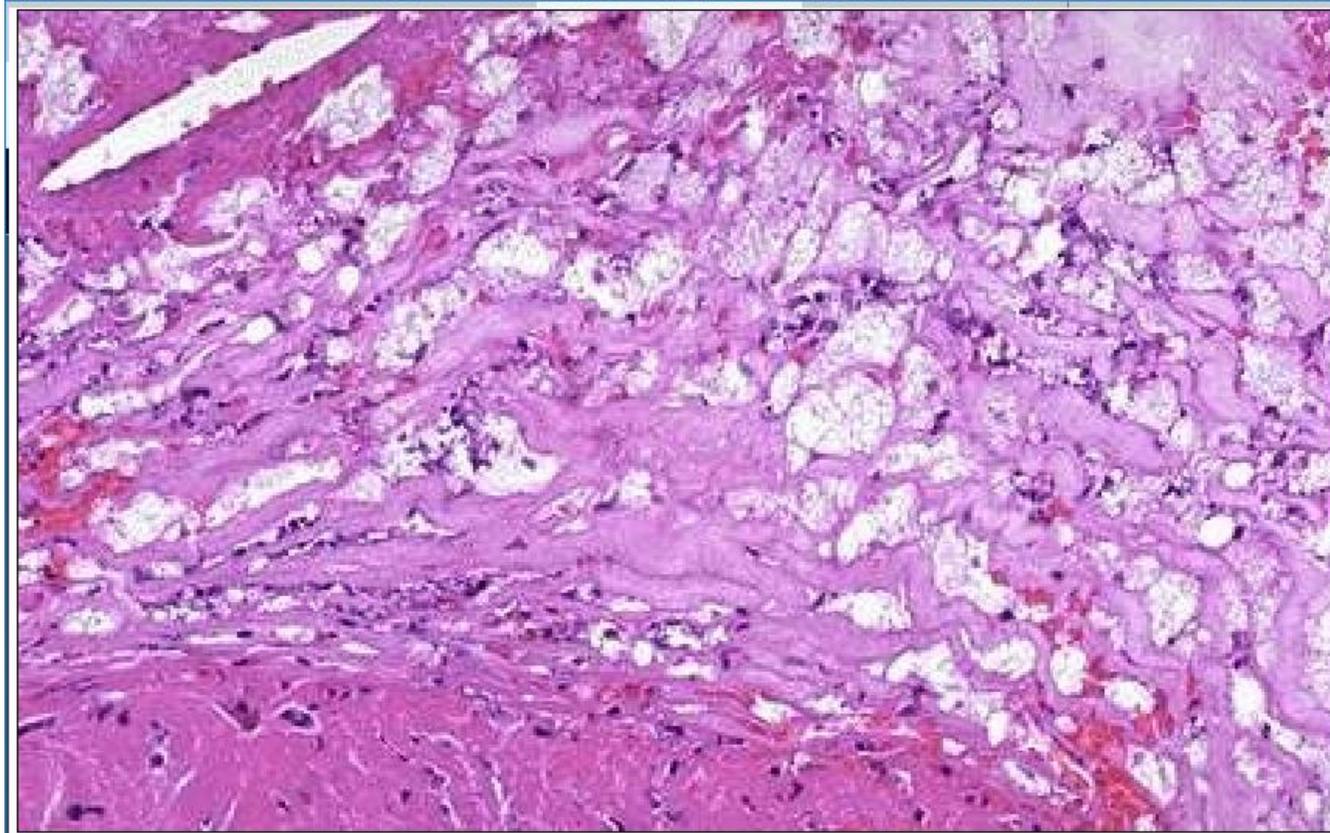
LIPOIDOSIS

- ❑ Focal infiltration of intima with lipids (cholesterol), lipoproteins, proteins.
- ❑ Leads to the formation of fat spots and streaks.
- ❑ Lipids diffusely impregnate the inner shell and accumulate in the SMC and macrophages, which turn into xanthomous (foamy) cells.
- ❑ Microscopically detected with Sudan III.

LIPOIDOSIS



XANTHOM (FOAMY) CELLS



LIPOSCLEROSIS

- ❑ Growth of connective tissue elements of the inner shell of vessels in the areas of lipid and protein deposition, which leads to the formation of fibrous plaque.
- ❑ At the edges of the plaque, new thin-walled vessels are formed, which also become an additional source of lipoproteins and plasma proteins.

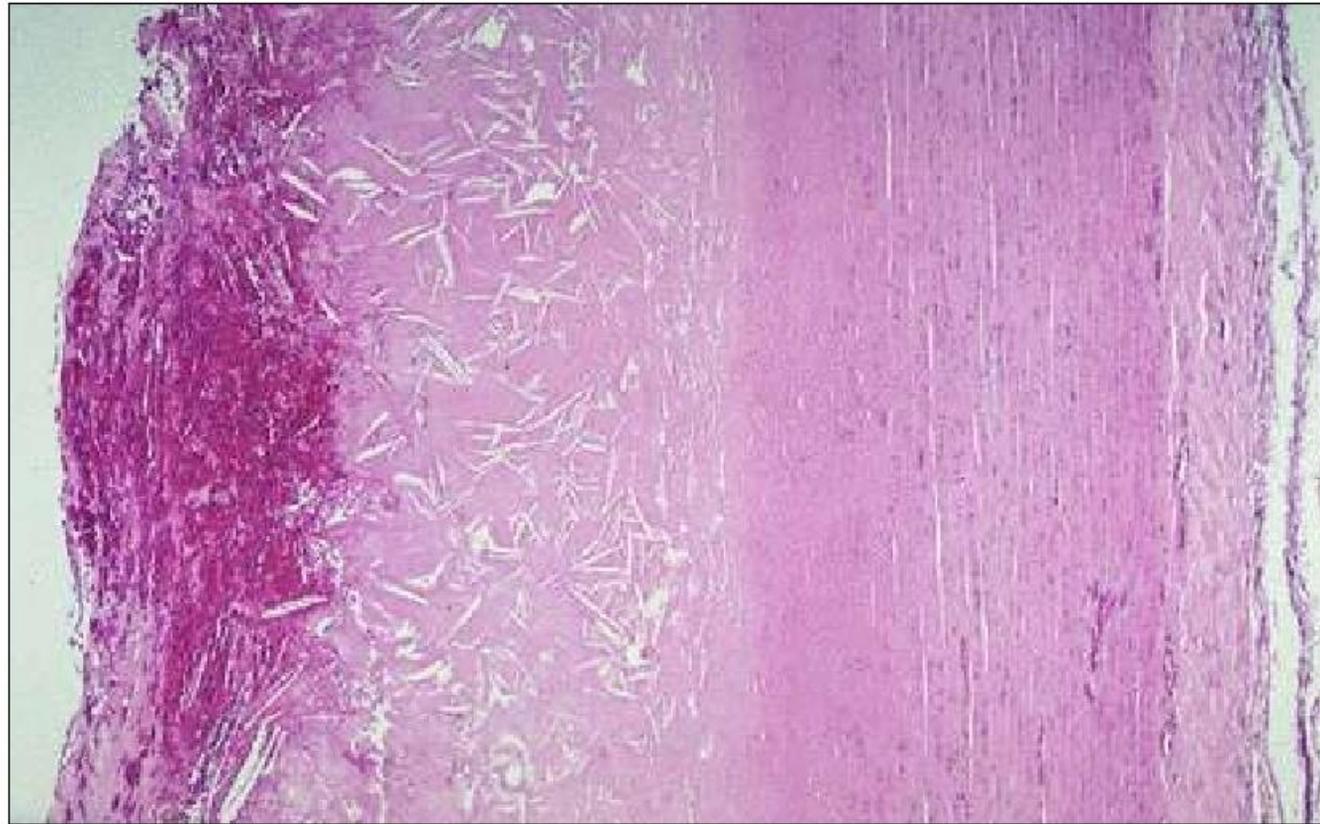
ATHEROMATOSIS

- ❑ Decay of lipid masses that make up the central part of the plaque, as well as collagen and elastic fibers.
- ❑ An amorphous mass is formed in which cholesterol crystals are detected (atheromatous detritus).
- ❑ Numerous vessels growing from vasa vasorum, as well as xantom cells, lymphocytes, and plasma cells are identified at the edges of the plaque.

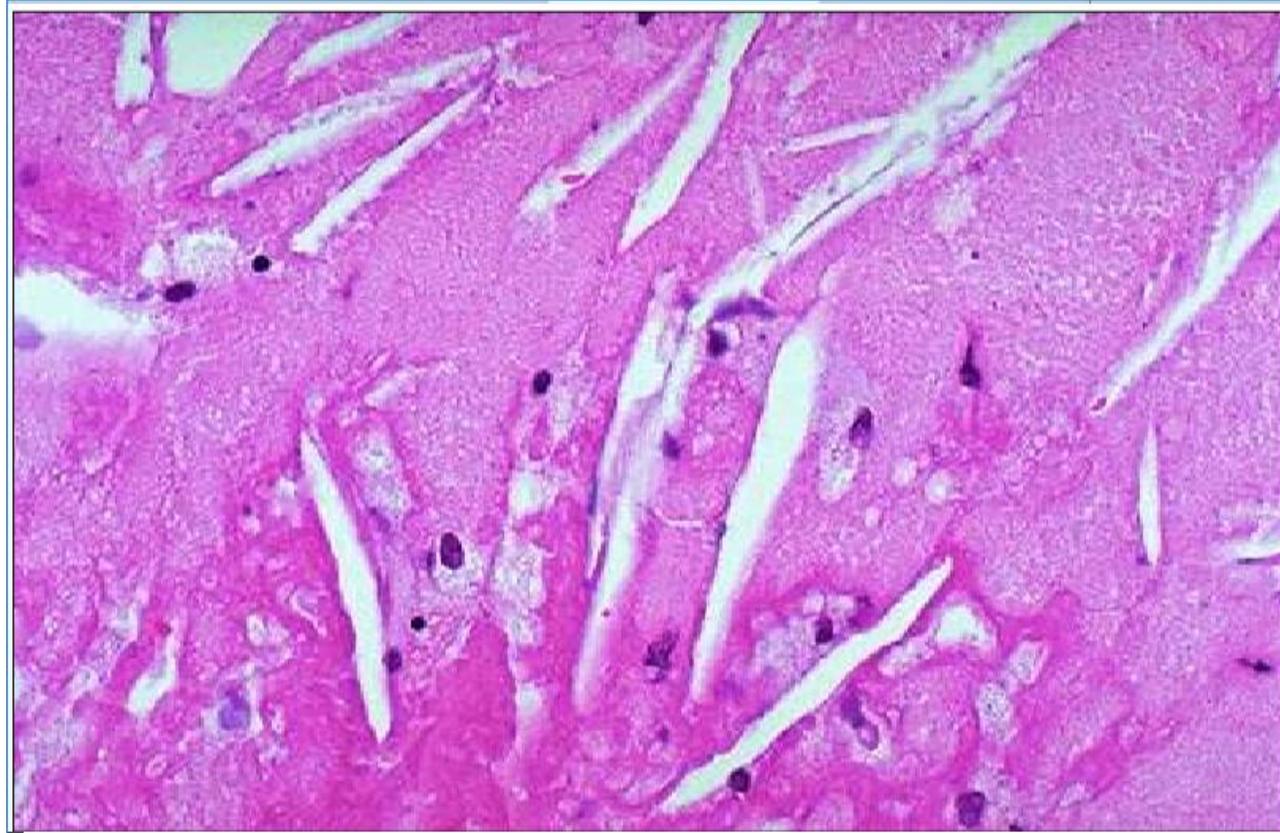
ATHEROMATOSIS

- ❑ Atheromatous masses are delimited from the lumen of the vessel by a layer of connective tissue, sometimes hyalinized ("plaque cover").
- ❑ The muscular layer is often atrophied, sometimes subjected to atheromatous decay, resulting in a plaque in some cases reaching adventitia.
- ❑ With progression of atheromatosis due to the destruction of newly formed vessels, a hemorrhage into the thickness of the plaque occurs (intramural hematoma).

ATHEROMATOSIS



CHOLESTEROL CRYSTALS



ULCERATION

- ❑ In case of destruction of the plaque cover, an atheromatous ulcer is formed.
- ❑ Defect of the inner shell of the vessel is often covered by thrombotic masses.

ATHEROCALCINOSIS

- ❑ The final stage of morphogenesis of atherosclerosis.
- ❑ Characterized by dystrophic calcification of atheromatous masses.

CLINICAL AND MORPHOLOGICAL FORMS

- ❑ Atherosclerosis of the aorta.
- ❑ Atherosclerosis of the coronary arteries of the heart (cardiac form, ischemic heart disease).
- ❑ Atherosclerosis of cerebral arteries (cerebral form, cerebrovascular diseases). Atherosclerosis of renal arteries (renal form).
- ❑ Atherosclerosis of the intestinal arteries (intestinal form).
- ❑ Atherosclerosis of the arteries of the lower extremities.

CLINICAL AND MORPHOLOGICAL FORMS

- ❑ Slow narrowing of the feeding artery by an atherosclerotic plaque leads to chronic insufficiency of blood supply and ischemic changes - dystrophy and atrophy of the parenchyma, diffuse or fine-sclerosis of the stroma.
- ❑ Acute occlusion of the feeding artery, usually due to complicated lesions, leads to acute blood supply insufficiency and development of necrosis - infarction, gangrene.
- ❑ In addition, in some cases, deep atheromatous ulcers can lead to the development of an aneurysm of the artery - the swelling of the artery wall at the site of the lesion, followed by its rupture and hemorrhage.

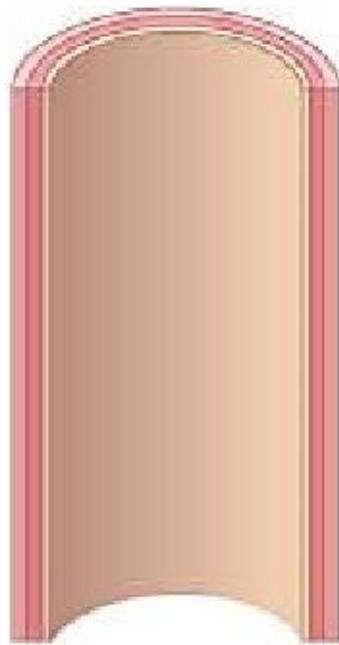
ATHEROSCLEROSIS OF THE AORTA

- ❑ The most common form.
- ❑ More sharply expressed in the abdominal part and characterized by usually complicated lesions and calcification.
- ❑ Most often accompanied by thrombosis, thromboembolism and embolism atheromatous masses with the development of infarcts and gangrene (intestines, lower limbs).

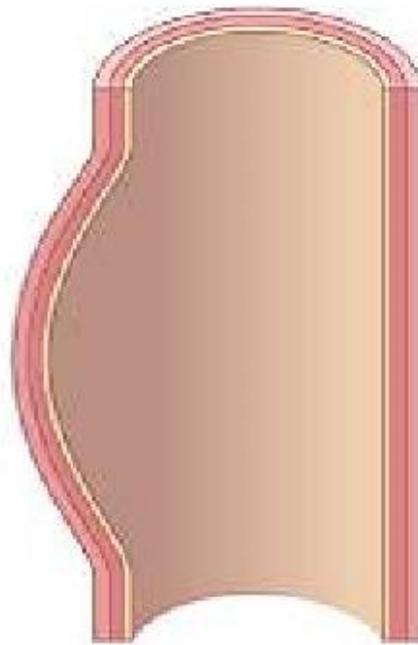
AORTIC ANEURYSM

- ❑ Aortic aneurysm often develops, which may be cylindrical, saccate or herniated.
- ❑ The wall of the aneurysm in some cases forms aorta (true aneurysm), in others - adjacent organs and hematoma (false aneurysm).
- ❑ If the blood exfoliates the middle layer from the inner or outer, leading to the formation of a canal covered by the endothelium, then we speak of a delaminating aneurysm.
- ❑ The formation of an aneurysm is fraught with its rupture and bleeding with the formation of a retroperitoneal hematoma.

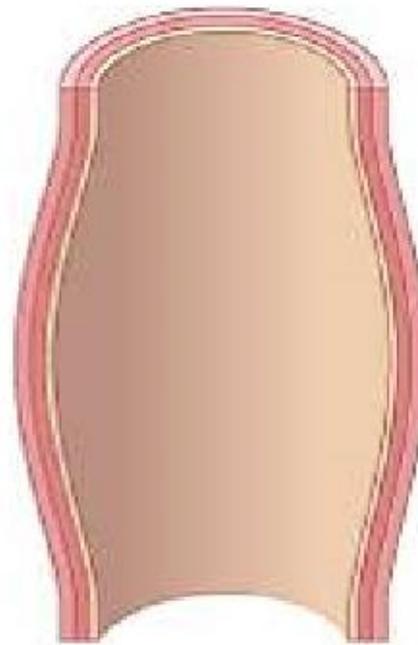
AORTIC ANEURYSM



A. Normal vessel

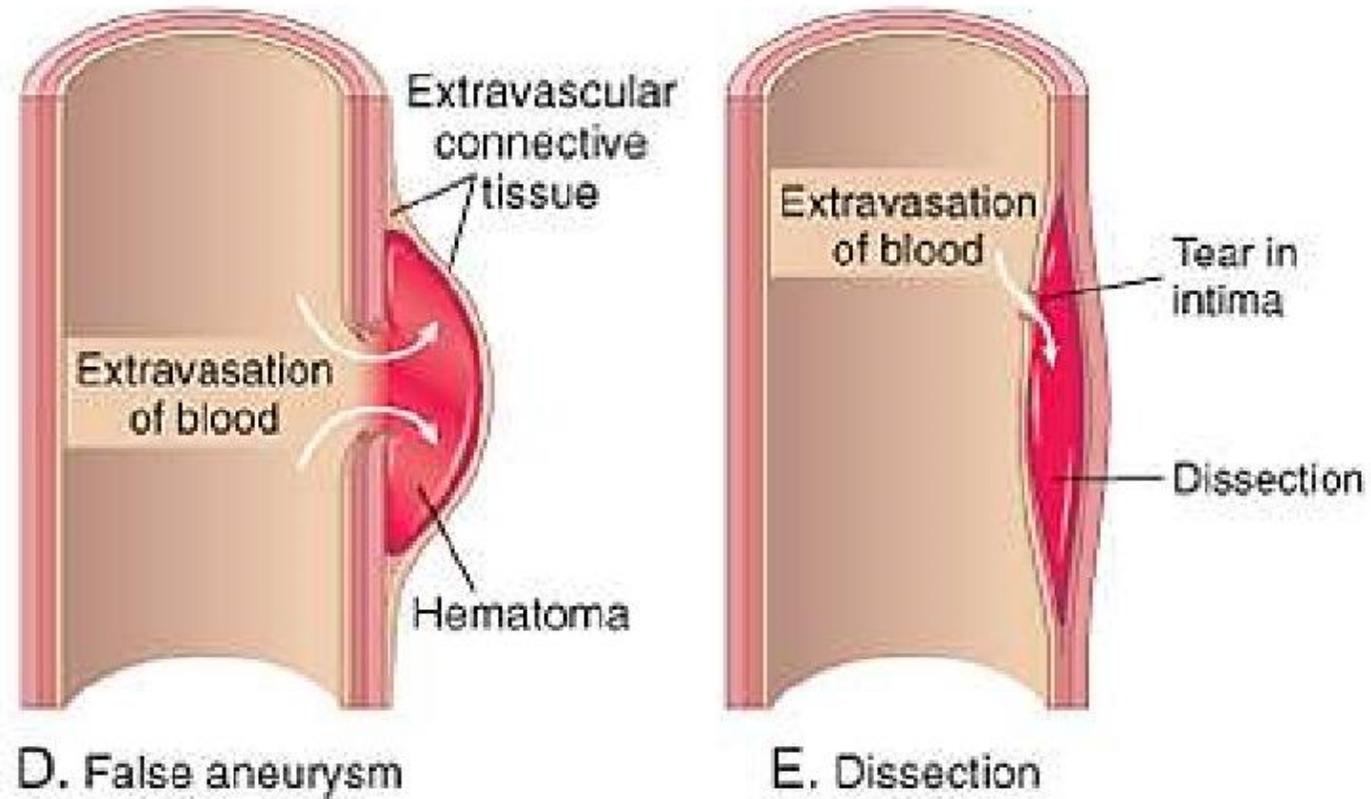


B. True aneurysm
(saccular)

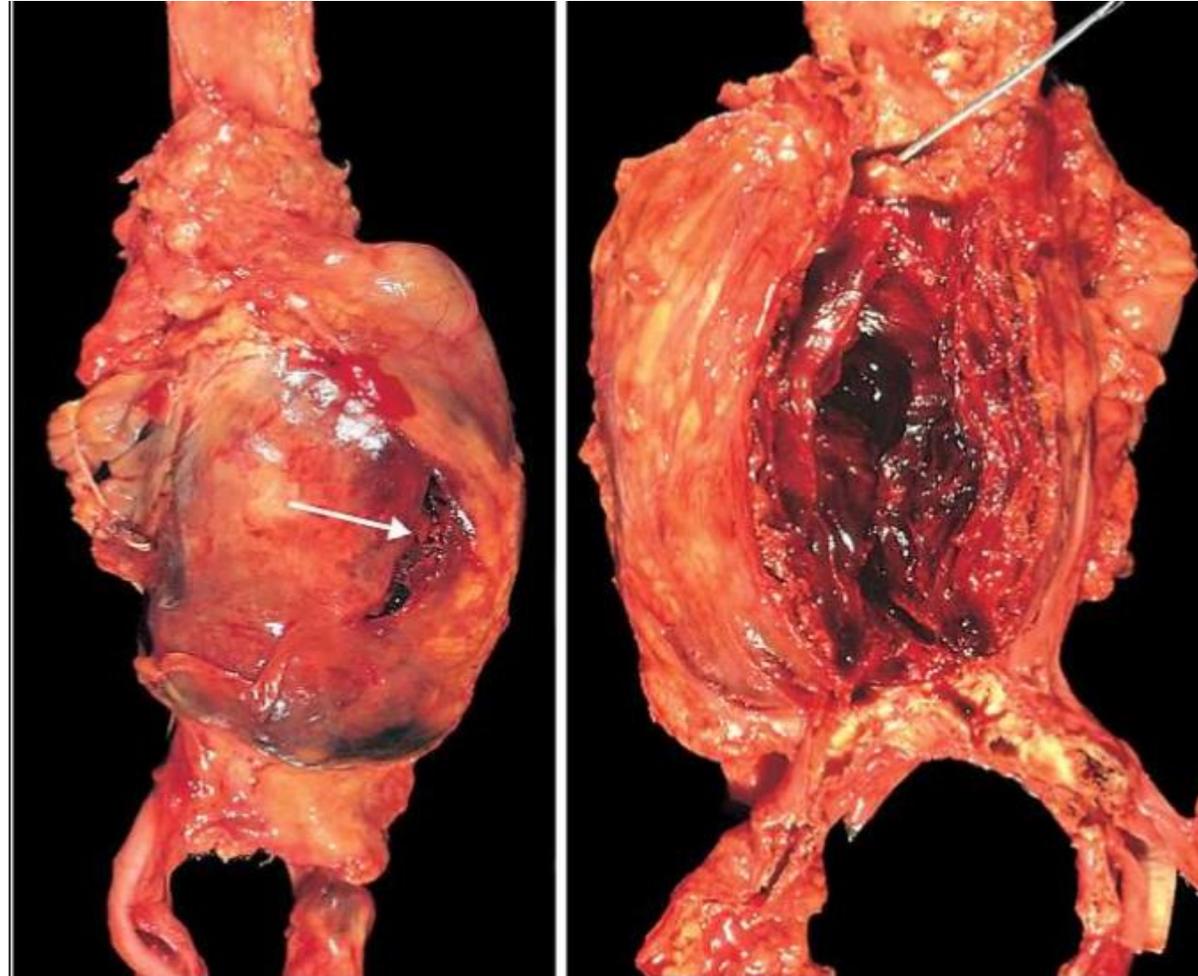


C. True aneurysm
(fusiform)

AORTIC ANEURYSM



RUPTURE OF THE AORTIC ANEURYSM



ATHEROSCLEROSIS OF CORONARY ARTERIES

- ❑ It is the basis of coronary heart disease.
- ❑ Morphological expression is focal ischemic dystrophy, myocardial infarction, large-focal (post-infarction) and diffuse small-focal cardiosclerosis.

ATHEROSCLEROSIS OF THE CEREBRAL ARTERIES

- ❑ It is the basis of cerebrovascular diseases.
- ❑ The most characteristic manifestations are ischemic and hemorrhagic cerebral infarction (stroke).
- ❑ Prolonged ischemia of the cerebral cortex due to stenosing atherosclerosis leads to atrophy of the cerebral cortex, development of atherosclerotic dementia.

ATHEROSCLEROSIS OF THE RENAL ARTERIES

- ❑ In the kidneys, wedge-shaped areas of parenchymal atrophy with collapse and sclerosis of the stroma are formed, or infarcts develop, followed by the formation of retracted scars.
- ❑ There is a large-hollowed atherosclerotic wrinkled kidney (atherosclerotic nephrosclerosis) - a primary-wrinkled kidney.
- ❑ As a result of ischemia of the renal tissue, symptomatic (renovascular) hypertension occurs.

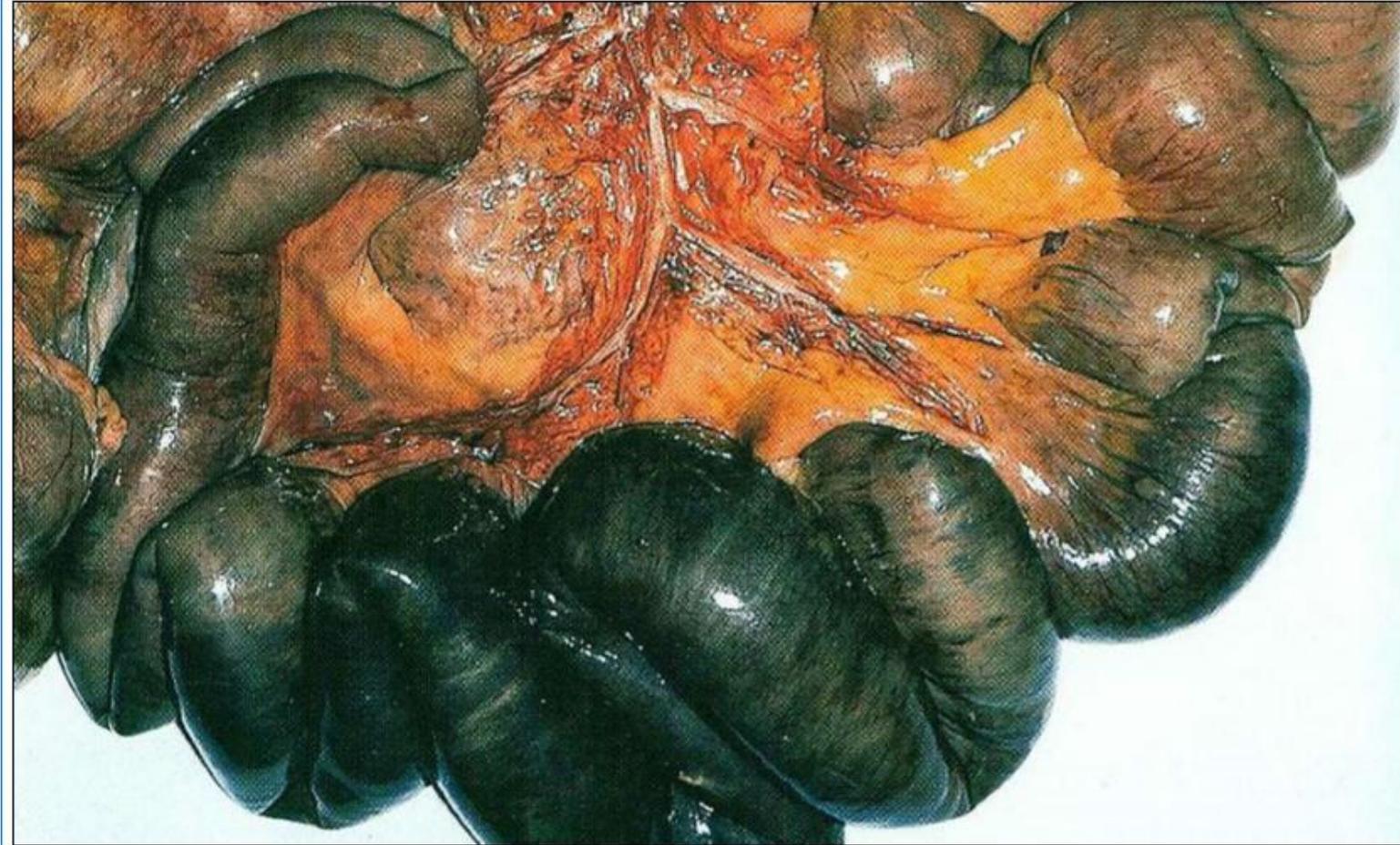
ATHEROSCLEROTIC WRINKLED KIDNEY



ATHEROSCLEROSIS OF THE INTESTINAL ARTERIES

- ❑ In case of complication with thrombosis, it leads to gangrene of the gut.
- ❑ Stenosing atherosclerosis of the mesenteric arteries can lead to the development of ischemic colitis, in which the splenic angle and rectosigmoid parts of the colon are more often affected.

GANGRENE OF INTESTINE



ATHEROSCLEROSIS OF THE ARTERIES OF THE EXTREMITIES

- ❑ Femoral arteries are more often affected.
- ❑ Stenosing atherosclerosis with insufficient collateral circulation leads to muscle atrophy and a characteristic symptom of intermittent claudication (pain that occurs in the legs when walking).
- ❑ If atherosclerosis is complicated by thrombosis, the gangrene of the limb develops.

GANGRENE OF LOWER LIMB



HYPERTONIC DISEASE

- ❑ Under arterial hypertension is a persistent increase in blood pressure: systolic - above 140 and diastolic - above 90 mm Hg.
- ❑ In most cases (90 to 95%), the cause of hypertension can not be established.
- ❑ This hypertension was called primary and isolated as an independent nosological form - hypertensive disease (essential hypertension).
- ❑ Arterial hypertension, which is a symptom of another disease, is called secondary, or symptomatic.

TYPES OF SYMPTOMATIC HYPERTENSION

- ❑ **Renal** (associated with kidney disease - renal or renal vessels - renovascular).
- ❑ **Endocrine** (in case of illness or syndrome of Cushing, primary and secondary aldosteronism, pheochromocytoma, etc.).
- ❑ **Neurogenic** (with increased intracranial pressure due to trauma, tumor, abscess, hemorrhage, with damage to the hypothalamus and brainstem associated with psychogenic factors).
- ❑ **Other** (due to coarctation of the aorta and other abnormalities of blood vessels, increased volume of circulating blood with excessive transfusion, polycythemia, etc.).

RISK FACTORS OF HD

- ❑ Hereditary predisposition.
- ❑ Chronic psychoemotional overstrain (frequent stresses, conflict situations, etc.).
- ❑ Excessive consumption of salt.
- ❑ In addition, a certain role is played by obesity, smoking, sedentary lifestyle (hypodynamia).

PATHOGENESIS OF HD

- ❑ The development of arterial hypertension can be caused by defects of any links (pressor and depressor) mechanism that determines the normal pressure.
- ❑ The main role in fixing, chronicating arterial hypertension is played by the kidneys.
- ❑ Several theories of the pathogenesis of essential hypertension have been proposed, which differently interpret the nature of the starting (initial) pathogenetic link.

THEORIES OF THE PATHOGENESIS OF HD

- ❑ **Theory of GF Lang and AL Myasnikov:** the initial factor is the psychoemotional overexertion with a decrease in the inhibitory effect of the cerebral cortex on the subcortical autonomic centers, especially the pressor centers, which causes their persistent overexcitation.
- ❑ **Theory A. Guyton et al .:** an initial factor is a genetically determined defect in the renal-volume mechanism of blood pressure regulation; Trigger (trigger) - increased salt intake.
- ❑ **Membrane theory of V. Postnova and S. N. Orlova:** the initial factor is the generalized hereditary defect of the membrane ion pumps of the cell, which leads to an excess of Ca^{2+} and Na^{+} in the cytoplasm of the SMC and causes their spasm, as well as an increase in sensitivity to pressor factors.

CHARACTER OF THE CURRENT HD

- It can be malignant (malignant hypertension) and benign (benign hypertension).

MALIGNANT HYPERTENSION

- ❑ Currently, malignant hypertension is rare.
- ❑ The diastolic pressure level exceeds 110 - 120 mmHg.
- ❑ Primary or complicating benign hypertension may occur.
- ❑ Rapidly progressing, leading to a fatal outcome (in the absence of adequate therapy) after 1 to 2 years.
- ❑ Occurs predominantly in men aged 35 to 50 years, sometimes up to 30 years.

MALIGNANT HYPERTENSION

- ❑ Fibrinoid necrosis of vessels with attached thrombosis and associated organ changes (infarcts, hemorrhages).
- ❑ Two-sided edema of the optic disc, accompanied by protein effusions and hemorrhages in the retina.
- ❑ In the kidneys develops malignant nephrosclerosis (nephrosclerosis Fara), which is characterized by fibrinoid necrosis of arterioles and capillary gland loops, edema and hemorrhages.
- ❑ Rapid progression of the process leads to the development of renal failure and death.
- ❑ The brain develops fibrinoid necrosis of arterioles, edema, hemorrhages.

BENIGN HYPERTENSION

- Given the prolonged development of the disease, three stages are identified that have certain morphological differences:
 - preclinical,
 - widespread changes in the arteries,
 - changes in organs due to changes in the arteries and violation of intraorganic circulation.

HYPERTENSIVE CRISIS

- Hypertensive crisis - a sharp increase in blood pressure due to spasm of arterioles - can occur at any stage.
- Morphological changes during a crisis:
 - Spasm of arterioles: corrugation and destruction of the basement membrane of the endothelium with its peculiar arrangement in the form of a stockade.
 - Plasma impregnation.
 - Fibrinoid necrosis of the arteriolar wall.
 - Thrombosis.
 - Diapedetic hemorrhages.

PRECLINICAL STAGE

- ❑ Characterized by episodes of high blood pressure - transient hypertension.
- ❑ Arterioles and small arteries show hypertrophy of the muscular layer and elastic structures, with a crisis - signs of spasm of arterioles, plasma impregnation and fibrinoid necrosis.
- ❑ In the heart there is a moderate compensatory hypertrophy of the left ventricle, which is not accompanied by an expansion of the cavities - concentric hypertrophy.

THE STAGE OF COMMON CHANGES IN THE ARTERIES

- ❑ It is characterized by a persistent increase in blood pressure.
- ❑ Arterioles and small arteries of the muscular type exhibit hyalinosis (the outcome of plasma impregnation) or arteriolosclerosis.
- ❑ Arteriologialinosis is noted in the kidneys, brain, pancreas, intestines, retina, capsule of the adrenal glands.

HYALINE ARTERIOLES WITH HD



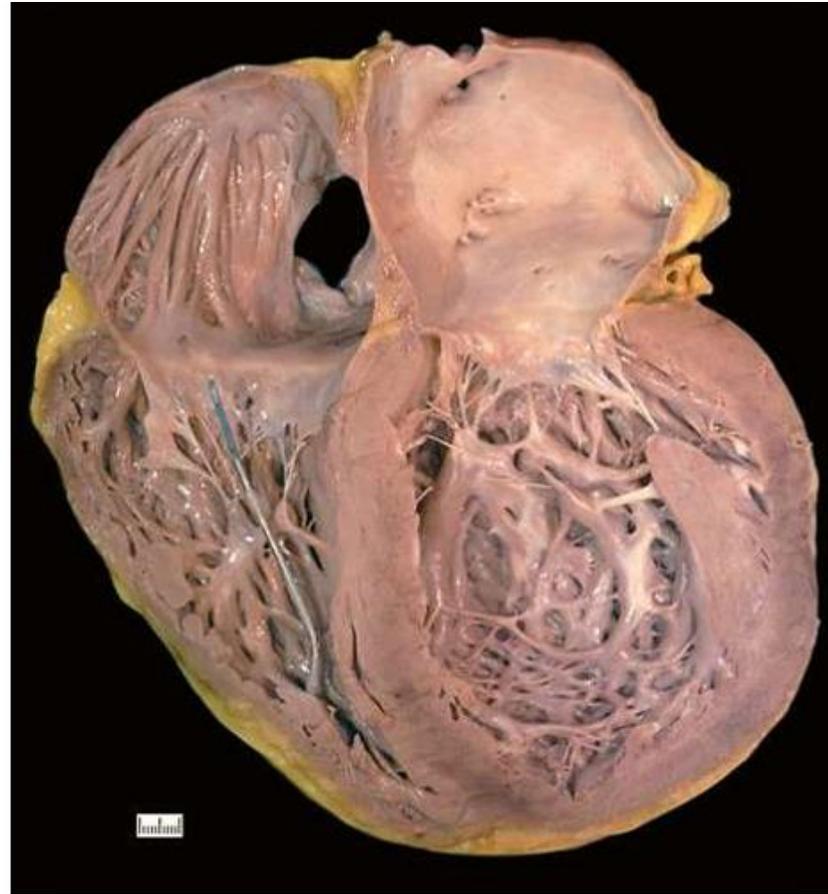
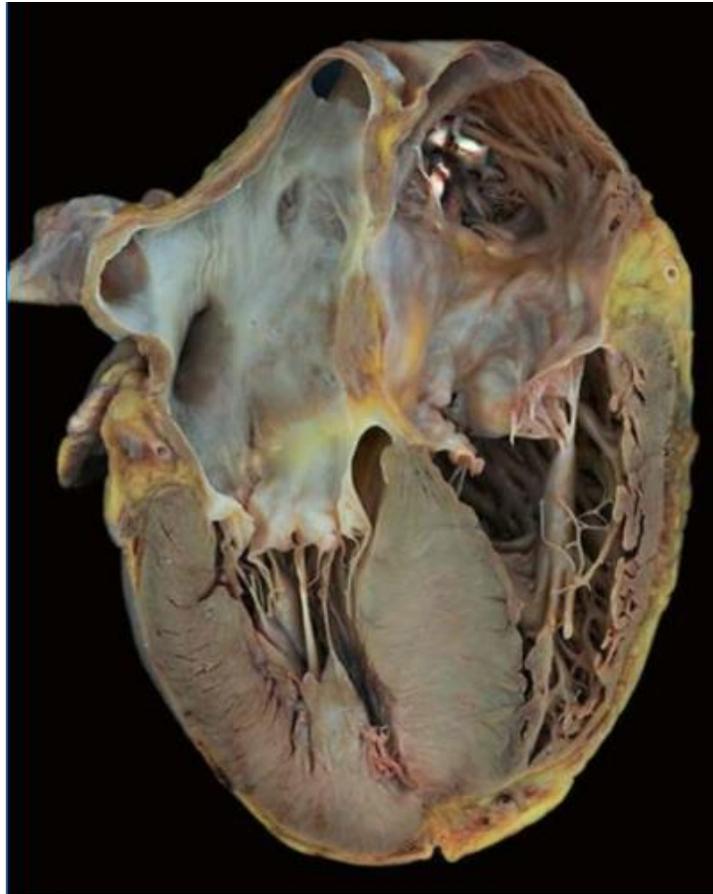
THE STAGE OF COMMON CHANGES IN THE ARTERIES

- ❑ In the arteries of the elastic, muscular-elastic and muscular types develop:
- ❑ Elastofibrosis - hyperplasia and splitting of the internal elastic membrane, sclerosis.
- ❑ Atherosclerosis, which has a number of peculiarities:
 - is more common, involvement arteries of the muscular type, which does not happen in the absence of arterial hypertension;
 - fibrotic plaques are circular, not segmental, which leads to a more significant narrowing of the lumen of the vessel.

THE STAGE OF COMMON CHANGES IN THE ARTERIES

- The degree of myocardial hypertrophy is increasing.
- In connection with the relative insufficiency of the blood supply (increase in heart mass, changes in arterioles and arteries) develop: fatty degeneration of the myocardium,
 - myogenic expansion of the heart cavities - eccentric hypertrophy of the myocardium,
 - diffuse small-focal cardiosclerosis,
 - signs of cardiac decompensation.

CONCENTRIC AND ECCENTRIC HYPERTROPHY OF THE LEFT VENTRICLE OF THE HEART WITH HD



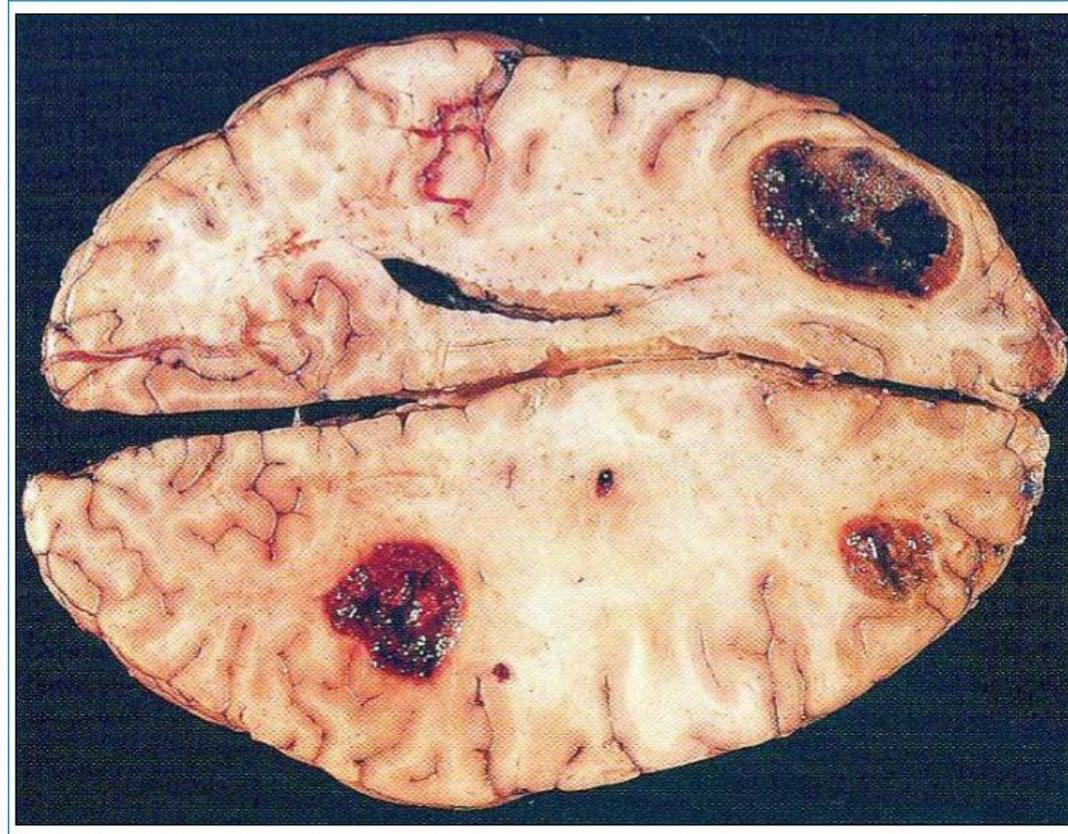
THE STAGE OF ORGAN CHANGES

- ❑ Secondary organ changes can develop slowly on the ground of arteriolo- and atherosclerotic occlusion of vessels, leading to atrophy of the parenchyma and sclerosis of the stroma.
- ❑ With the addition of thrombosis, spasm, fibrinoid necrosis (during a crisis), acute changes occur: hemorrhages, heart attacks.

THE STAGE OF ORGAN CHANGES

- ❑ The most characteristic hemorrhages in the brain.
- ❑ They can be small, arising through diapedesis, or large with the destruction of brain tissue - hematoma.
- ❑ Hematomas usually develop with rupture of microaneurysms, which occur, as a rule, due to hyalinosis and fibrinoid necrosis; They are especially often found in small perforating cerebral arteries (less than 1 mm in diameter) of predominantly subcortical nuclei and the subcortical layer.
- ❑ In the outcome of hemorrhages in the brain tissue, rusty cysts are formed.

RUSTED CYST IN THE BRAIN



THE STAGE OF ORGAN CHANGES

- ❑ In the kidneys develops arteriolosclerotic nephrosclerosis, or primary-wrinkled kidneys, which are based on arteriologialinosis with the subsequent development of atrophic and sclerotic changes.
- ❑ Arteriolosclerotic nephrosclerosis can lead to the development of chronic renal failure.

PRIMARYLY WRINKLED KIDNEYS

□ **Macroscopic picture:**

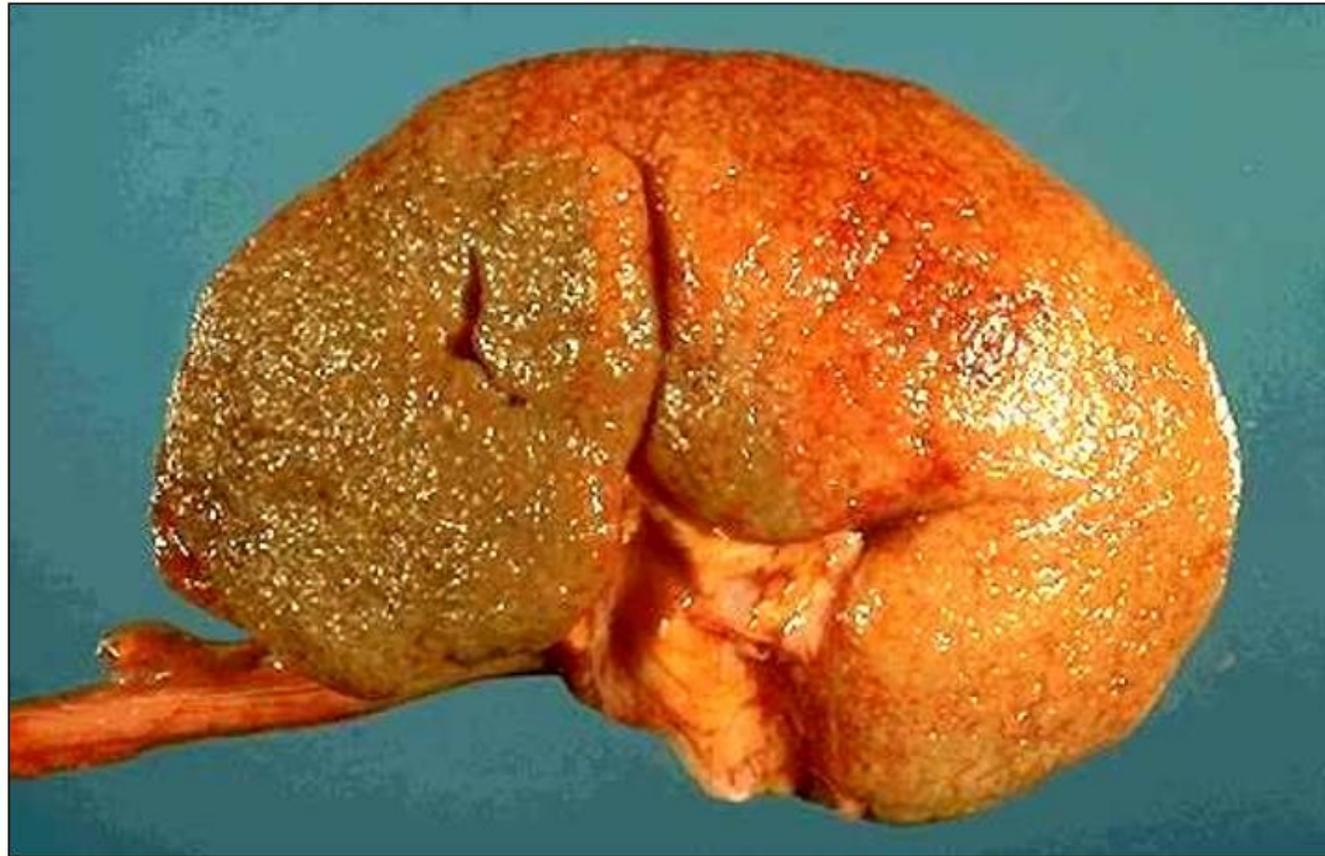
- the kidneys are considerably reduced in size,
- their surface is fine-grained (the waning areas correspond to atrophied nephrons, bulging - the remaining hypertrophied glomeruli).
- The thinning of cortical and cerebral layers, the growth of fatty tissue around the pelvis.

PRIMARYLY WRINKLED KIDNEYS

□ **Microscopic picture:**

- Walls of arterioles are considerably thickened due to the accumulation in the intima of homogeneous unstructured masses of hyaline, the lumen is narrowed, sometimes obliterated.
- The glomeruli are collapsed (collapsed), many are replaced by connective tissue or hyaline masses.
- The tubules are atrophied.
- The amount of interstitial connective tissue is increased.
- The remaining nephrons are compensatory hypertrophied.

PRIMARYLY WRINKLED KIDNEY WITH HD



CLINICAL AND MORPHOLOGICAL FORMS OF HD

❑ Cardiac form of hypertensive disease.

- Like the heart form of atherosclerosis, it is the essence of ischemic heart disease.

❑ Brain form of hypertensive disease.

- Like the atherosclerosis of the brain vessels, it has now become the basis of cerebrovascular diseases.

❑ Renal form of hypertension.

- Arteriolonecrosis (with malignant HD), renal infarction, arteriosclerotic nephrosclerosis (with benign HD).

CAUSES OF DEATH IN HD

- Most patients with benign hypertension die from:
 - heart failure,
 - myocardial infarction,
 - cerebral stroke (ischemic or hemorrhagic).
- A very small number of patients older than 60 years die from renal failure due to atheroarteriolosclerotic nephrosclerosis

ISCHEMIC HEART DISEASE

- ❑ A group of diseases caused by absolute or relative insufficiency of the coronary circulation.
- ❑ IHD develops with atherosclerosis of the coronary arteries, i.e. Is a cardiac form of atherosclerosis and hypertension.
- ❑ Highlighted as an independent nosological group in 1965 due to its great social importance.
- ❑ Atherosclerosis and hypertension in IHD are considered as background diseases

ISCHEMIC HEART DISEASE

- All other variants of ischemic myocardial damage associated with congenital coronary artery anomalies, arteritis, coronary artery thromboembolism, anemia, CO poisonings, etc., are regarded as complications of these diseases and are not included in IHD.

RISK FACTORS FOR IHD

- ❑ Hypercholesterolemia (dyslipoproteinemia).
- ❑ Smoking.
- ❑ Hypertension.
- ❑ In addition, hypodynamia, obesity, stress, a decrease in glucose tolerance, belonging to the male sex, age, etc. are important.

PATHOGENESIS OF IHD

- ❑ The main link in the pathogenesis of IHD is the discrepancy between the level of oxygen supply to the myocardium and the need for it, caused by atherosclerotic changes in the coronary arteries.
- ❑ In one third of patients with coronary artery disease one coronary artery is injured, 1/3 - two arteries, the rest - all three.
- ❑ The first 2 cm of the left anterior descending and enveloping arteries are often affected.
- ❑ More than 90% of CHD patients have stenosing coronary artery atherosclerosis with a stenosis degree of more than 75% of at least one arterial artery.

CAUSES OF ISCHEMIC MYOCARDIUM DAMAGE

- ❑ Thrombosis of coronary arteries.
- ❑ Thromboembolism of the coronary arteries.
- ❑ Prolonged spasm of the coronary arteries.
- ❑ Functional overstrain of the myocardium in conditions of stenosing atherosclerosis of coronary arteries and insufficient collateral circulation.

ATHEROSCLEROSIS OF CORONARY ARTERIES



ISCHEMIC INJURY OF THE MYOCARDIUM

- ❑ Ischemic damage to the myocardium can be reversible and irreversible.
- ❑ Reversible ischemic lesions develop in the first 20 to 30 minutes after the onset of ischemia and after the cessation of the effect of the factor that caused them completely disappear.
- ❑ Irreversible ischemic damage to cardiomyocytes begins with ischemia lasting more than 20 to 30 minutes.

ISCHEMIC INJURY OF THE MYOCARDIUM

- ❑ The first 18 hours after the development of ischemia, morphological changes are recorded only by electron microscopy (the appearance of calcium in the mitochondria), histochemical and luminescent methods.
- ❑ After 18-24 hours, micro- and macroscopic signs of necrosis appear, i.e. A myocardial infarct is formed.

CLASSIFICATION OF IHD

❑ ACUTE IHD

1. Sudden cardiac (coronary) death
2. Acute focal ischemic myocardial dystrophy
3. Myocardial infarction

❑ CHRONIC IHD

1. Large-heart (postinfarction) cardiosclerosis
2. Small-focal diffuse cardiosclerosis

SUDDEN CARDIAC DEATH

□ Criteria for sudden coronary death in accordance with WHO recommendations:

Death occurring within the first 6 hours after the onset of acute ischemia.

This death is most likely due to ventricular fibrillation.

There are no signs of linking this death with another disease.

SUDDEN CARDIAC DEATH

- ❑ ECG is not informative.
- ❑ At autopsy, as a rule, a severe (with a stenosis of more than 75%), common (with damage to all arteries), atherosclerosis; Thrombi in the coronary arteries are detected in less than half of the deceased.
- ❑ The main cause of sudden cardiac death is ventricular fibrillation, which can be detected microscopically by applying additional techniques (with Rego staining) in the form of re-reduction of myofibrils until coarse contractures and ruptures appear.
- ❑ The development of fibrillation is associated with electrolyte (increasing the level of extracellular potassium) and metabolic disorders, leading to the accumulation of arrhythmogenic substances - lysophosphoglycerides, cAMP, and others.

ACUTE FOCAL ISCHEMIC MYOCARDIAL DYSTROPHY

- ❑ Form AIMD, developing in the first 6 - 18 hours after the onset of acute myocardial ischemia.
- ❑ Has a characteristic change on the ECG.
- ❑ Blood (more than 12 hours after the onset of ischemia, there may be a slight increase in the concentration of enzymes from the damaged myocardium - creatinine phosphokinase (CPK) and aspartate aminotransferase (AST).
- ❑ Causes of death:
 - ventricular fibrillation,
 - asystole,
 - acute heart failure.

ACUTE FOCAL ISCHEMIC DYSTROPHY OF THE MYOCARDIUM

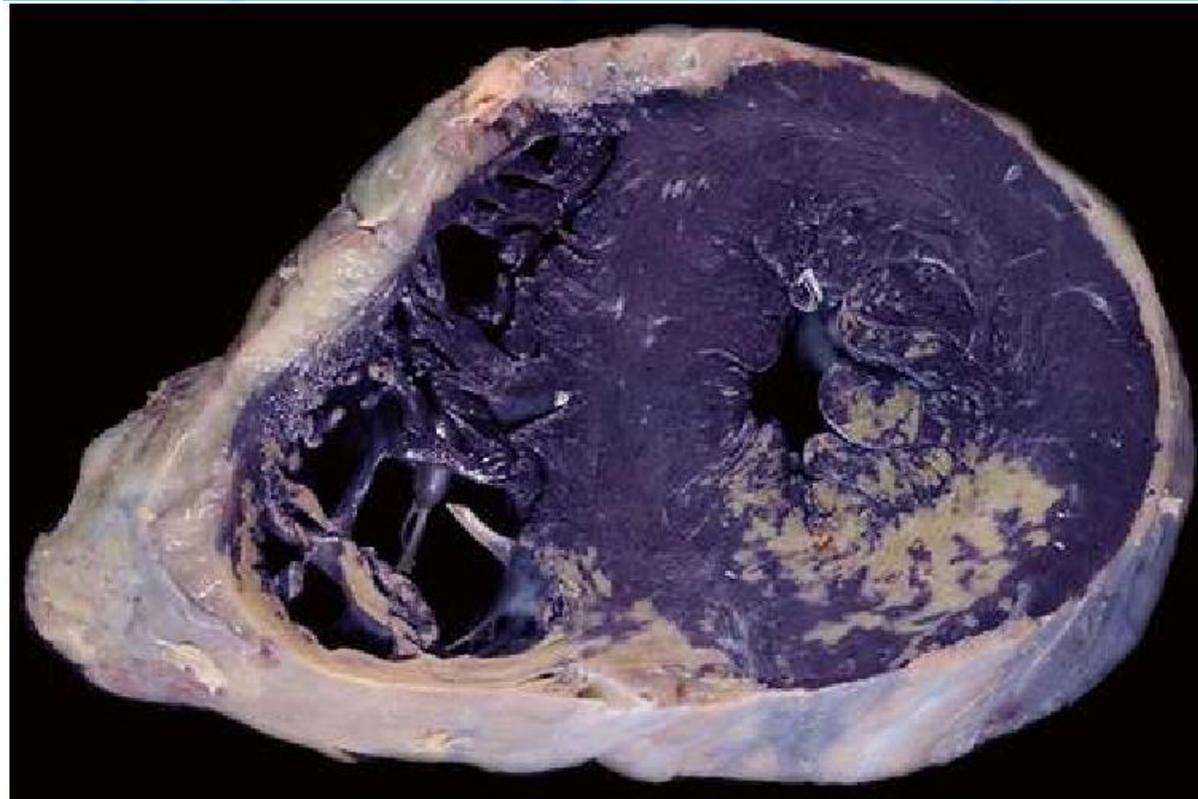
❑ **Macroscopic picture:**

- At autopsy, ischemic lesions are diagnosed with potassium tellurite and tetrazolium salts that do not stain the ischemia zone due to a decrease in dehydrogenase activity.

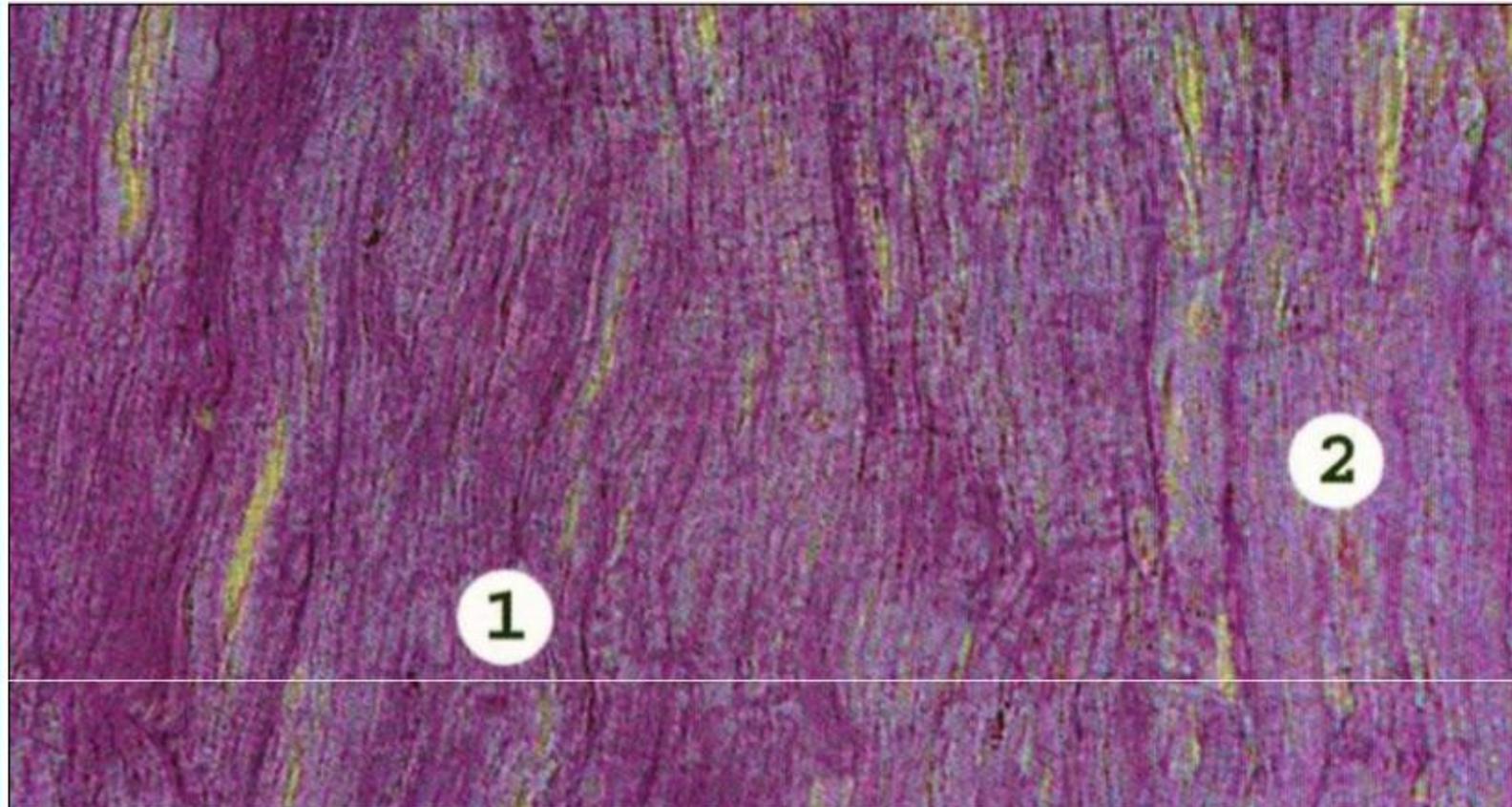
❑ **Microscopic picture:**

- when the PAS-reaction is detected, the disappearance of glycogen from the ischemia zone, in the surviving cardiomyocytes glycogen is colored in crimson color.

ACUTE FOCAL ISCHEMIC MYOCARDIAL DYSTROPHY (TETRAZOLIUM SALTS)



ACUTE FOCAL ISCHEMIC MYOCARDIAL DYSTROPHY PAS-REACTION



MYOCARDIAL INFARCTION

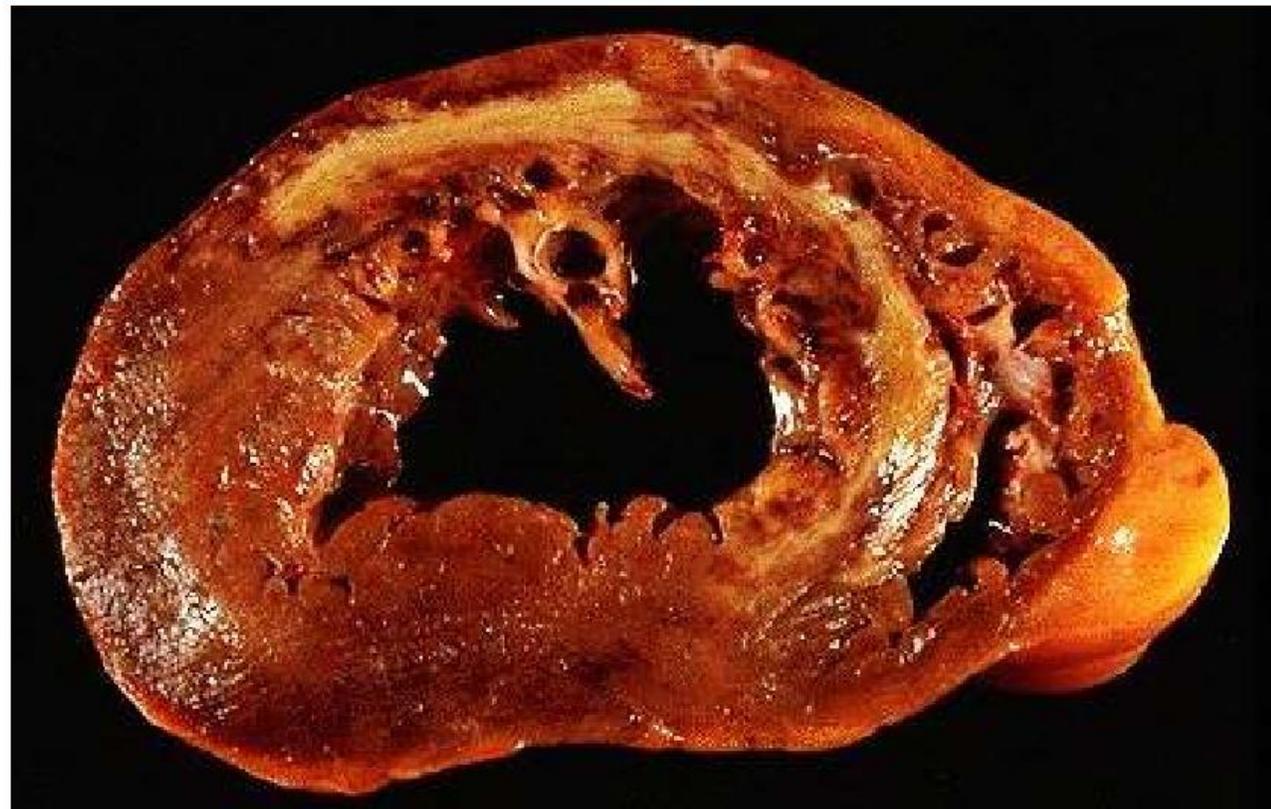
- ❑ The form of acute ischemic heart disease, characterized by the development of ischemic necrosis of the myocardium.
- ❑ It develops 18 to 24 hours after the onset of ischemia, when the necrosis zone becomes visible micro- and macroscopically.
- ❑ Has a characteristic change on the ECG.
- ❑ In the blood, pronounced enzyme is found: the level of CK peak to 24 hours, the AST content to 48 hours, the LDH level on the 2nd - 3rd day.
- ❑ By the 10th day, the enzyme level is normalized.

MYOCARDIAL INFARCTION

□ A macroscopic picture:

- a yellow-white focus surrounded by a dark red corolla,
- localized more often in the anterior wall of the left ventricle,
- irregular shape,
- flabby consistency.

TRANSMURAL MYOCARDIAL INFARCTION



MYOCARDIAL INFARCTION

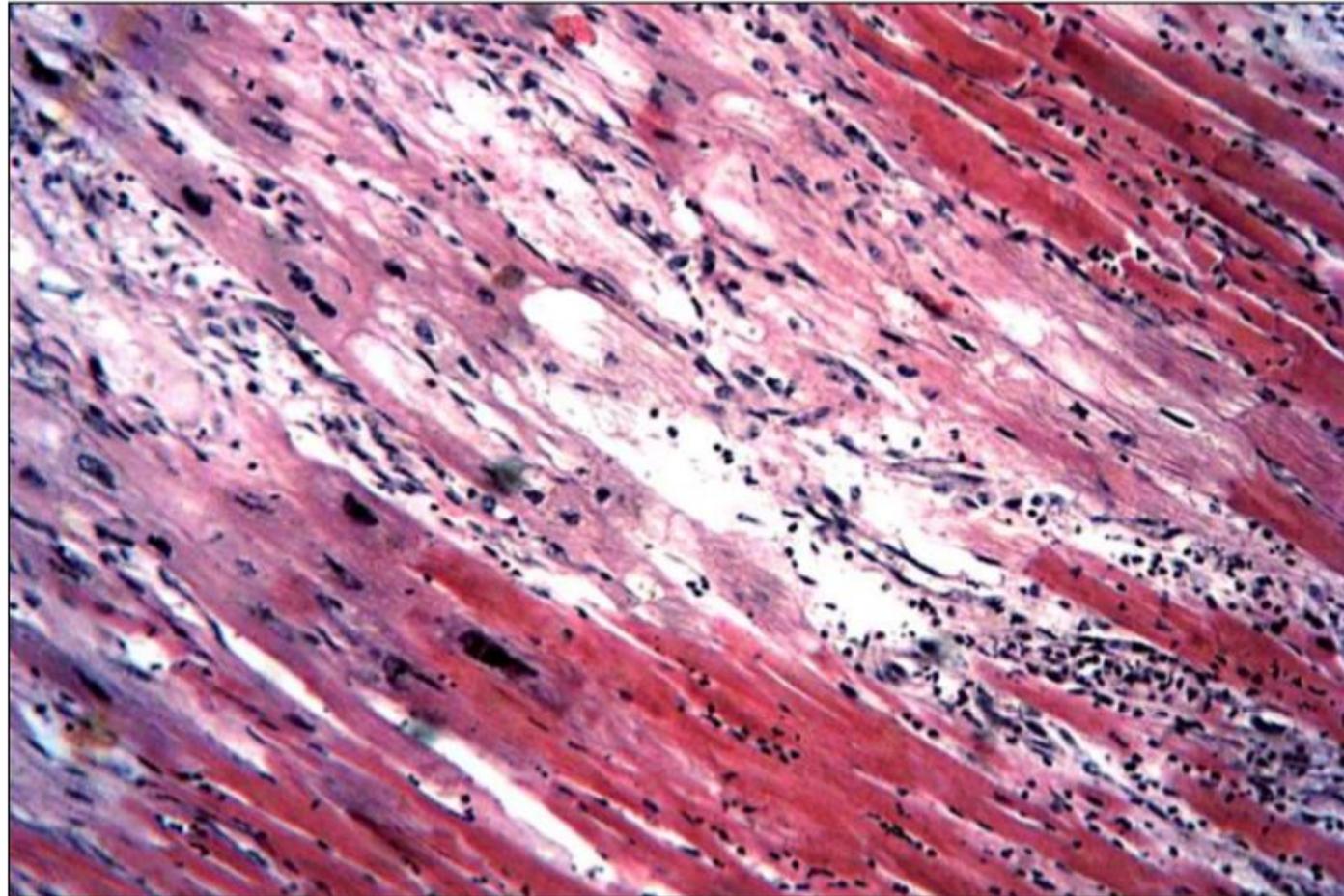


MYOCARDIAL INFARCTION

□ Microscopic picture:

- A zone of necrosis, surrounded by a zone of demarcation inflammation, separating the first from the preserved tissue of the myocardium is determined.
- In the necrosis zone - cardiomyocytes, devoid of nuclei (karyolysis), with lumpy decomposition of the cytoplasm (plasmorexis);
- On the periphery of necrosis - expansion and fullness of the vessels, congestion of PLN (demarcation inflammation);
- In the surviving parts of the myocardium - dystrophic changes in cardiomyocytes.

MICROSCOPIC PICTURE OF MI



MYOCARDIAL INFARCTION

- ❑ From the 7th to the 10th day, a young connective tissue (granulation tissue) appears in the zone of demarcation inflammation, gradually replacing the necrosis zone.
- ❑ Scarring of the heart attack occurs by the 6th week.
- ❑ During the infarction, the following stages are distinguished:
 - pre-inecrotic (ischemic),
 - necrotic,
 - scarring.

CLASSIFICATION OF MI

□ By the time of onset:

- primary infarction,
- recurrent
 - develops within 6 weeks after the previous myocardial infarction,
- repeated
 - develops 6 weeks after the previous myocardial infarction.

CLASSIFICATION OF MI

□ Localization:

- infarction of the anterior wall of the left ventricle, apex and anterior divisions of the interventricular septum (40-50%),
- posterior wall of the left ventricle (30-40%), lateral wall of the left ventricle (15-20%),
- isolated infarction of the interventricular septum (7 - 17%),
- extensive heart attack.

□ In relation to the membranes of the heart:

- subendocardial,
- subepicardial,
- intramural,
- transmural (affecting the entire thickness of the myocardium)

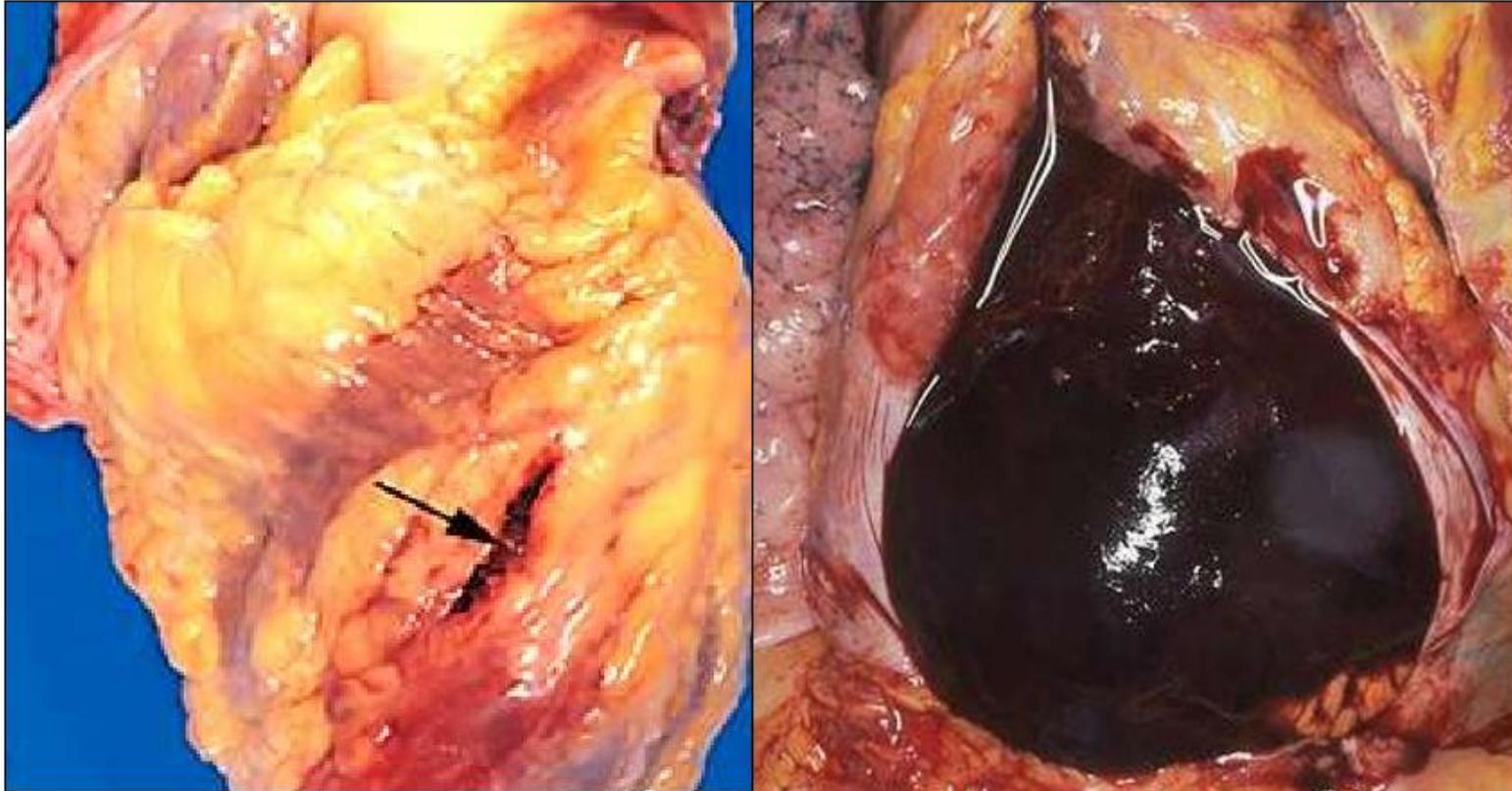
COMPLICATIONS OF MYOCARDIAL INFARCTION

- ❑ Cardiogenic shock.
- ❑ Ventricular fibrillation.
- ❑ Asystole.
- ❑ Acute congestive heart failure.
- ❑ Myomalacia and rupture of the heart.
- ❑ Acute aneurysm of the heart.
- ❑ parietal thrombosis with thromboembolic complications.
Pericarditis, pleurisy (Dressler's syndrome).

CAUSES OF DEATH

- ❑ Mortality with myocardial infarction is 35%.
- ❑ In the first few hours:
 - from fatal arrhythmias (ventricular fibrillation),
 - cardiogenic shock,
 - acute heart failure.
- ❑ In a later period:
 - from thromboembolism,
 - heart rupture, often in the area of acute aneurysm with a tamponade of the pericardial cavity (4 - 10th day).

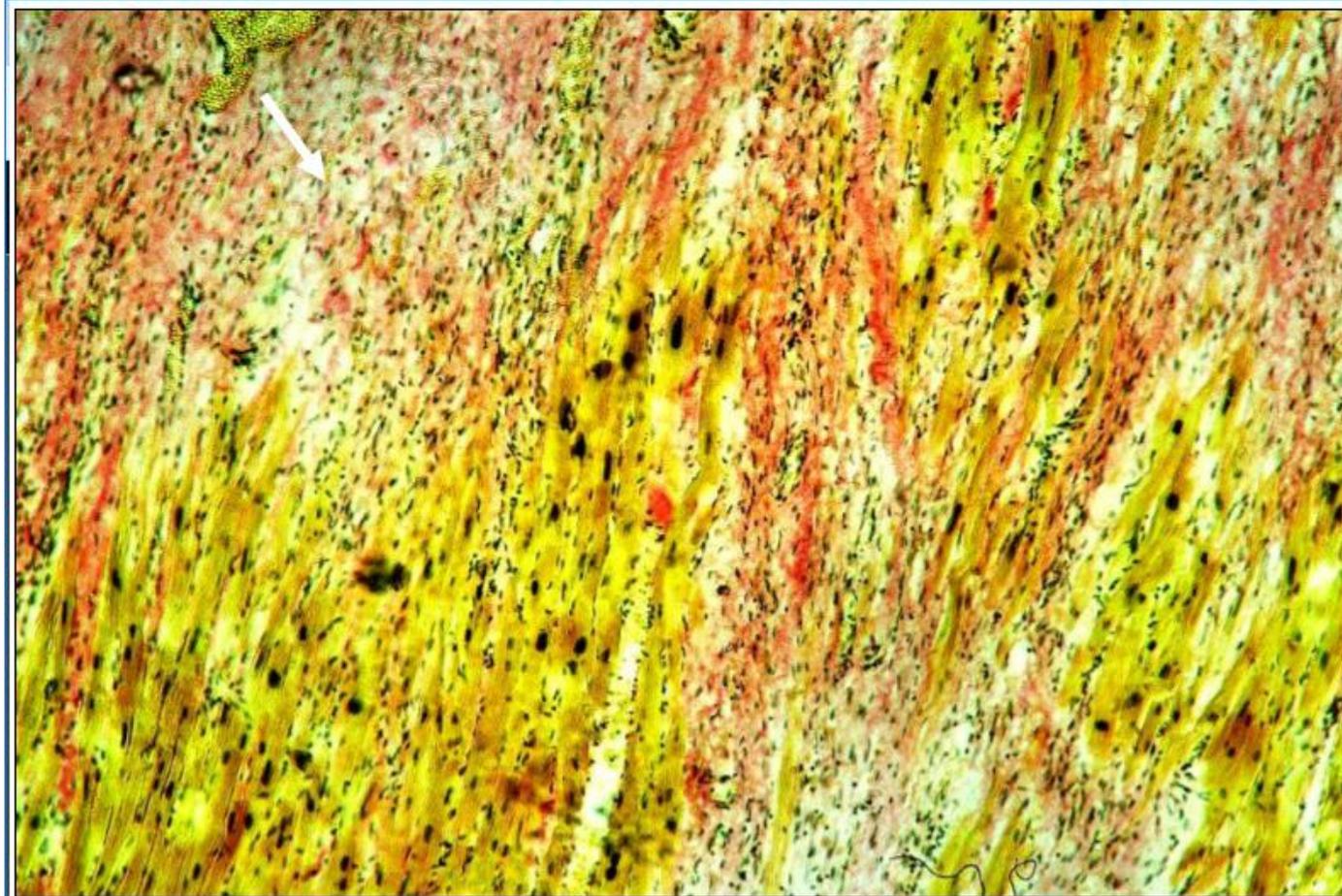
HEART RUPTURE, HEMOPERICARD WITH TAMPONADE



LARGE-SCALE CARDIOSCLEROSIS

- ❑ Large-focal cardiosclerosis develops in the outcome of a heart attack.
- ❑ **Macroscopic picture:**
 - in the wall of the left ventricle, a dense foci of irregularly shaped grayish-white color is defined, myocardium is hypertrophied.
- ❑ **Microscopic picture:**
 - a lesion of a sclerosis of the wrong form, expressed hypertrophy of cardiomyocytes on periphery.
 - When staining the connective tissue (van Gieson), the scar turns red, the cardiomyocytes turn into yellow-green.
- ❑ Sometimes it is complicated by the development of chronic heart aneurysm.

LARGE-SCALE CARDIOSCLEROSIS

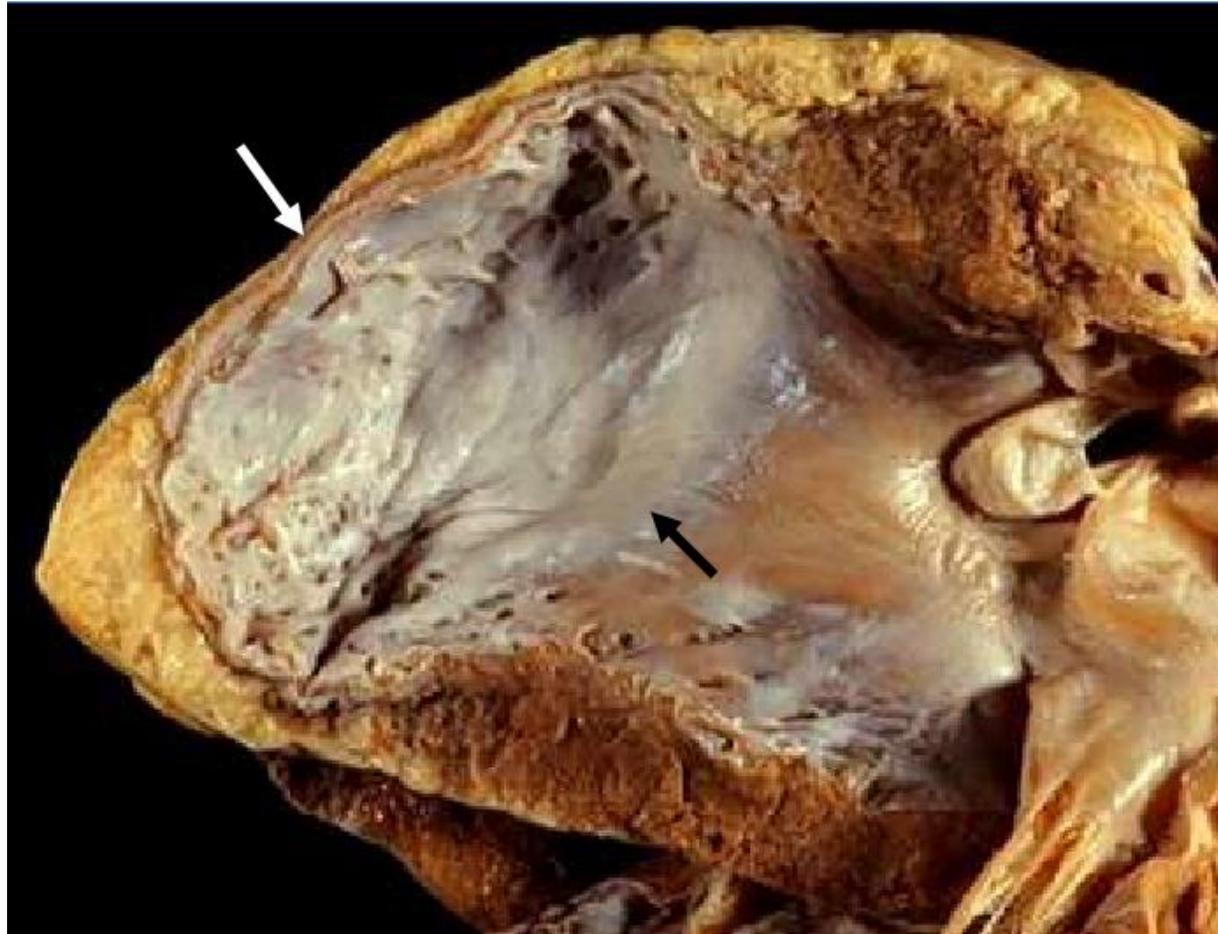


CHRONIC ANEURYSM OF THE HEART

□ **A macroscopic picture:**

- The heart is enlarged in size.
- The wall of the left ventricle in the region of the apex (anterior, posterior wall, interventricular septum) is thinned, whitish gray, represented by scar tissue, swells.
- The myocardium around the bulging is hypertrophic.
- Often in the cavity of the aneurysm, it is possible to detect parietal thrombi.

CHRONIC ANEURYSM OF THE HEART



FINESCALE CARDIOSCLEROSIS

- ❑ Diffuse small-focal cardiosclerosis develops as a result of relative coronary insufficiency with the development of small foci of ischemia.
- ❑ Clinically accompanied by angina attacks.
- ❑ Often occurs with rhythm disturbances.

CAUSES OF DEATH IN CIHD

- ❑ Chronic cardiovascular failure.
- ❑ Thromboembolic complications.

CEREBROVASCULAR DISEASES

- ❑ A group of diseases characterized by acute disorders of cerebral circulation, the background for which are atherosclerosis and hypertension.
- ❑ Separated into an independent group in 1977 due to the great social importance.

CLASSIFICATION OF CD

- Diseases of the brain associated with ischemic damage:
 - ischemic encephalopathy,
 - ischemic cerebral infarction,
 - hemorrhagic cerebral infarction.
- Intracranial hemorrhage.
- Hypertensive cerebrovascular diseases.

CEREBROVASCULAR DISEASES

□ The clinic uses the terms "stroke", or "brain blow".

□ **Ischemic stroke:**

- Ischemic cerebral infarction,
- Hemorrhagic cerebral infarction.

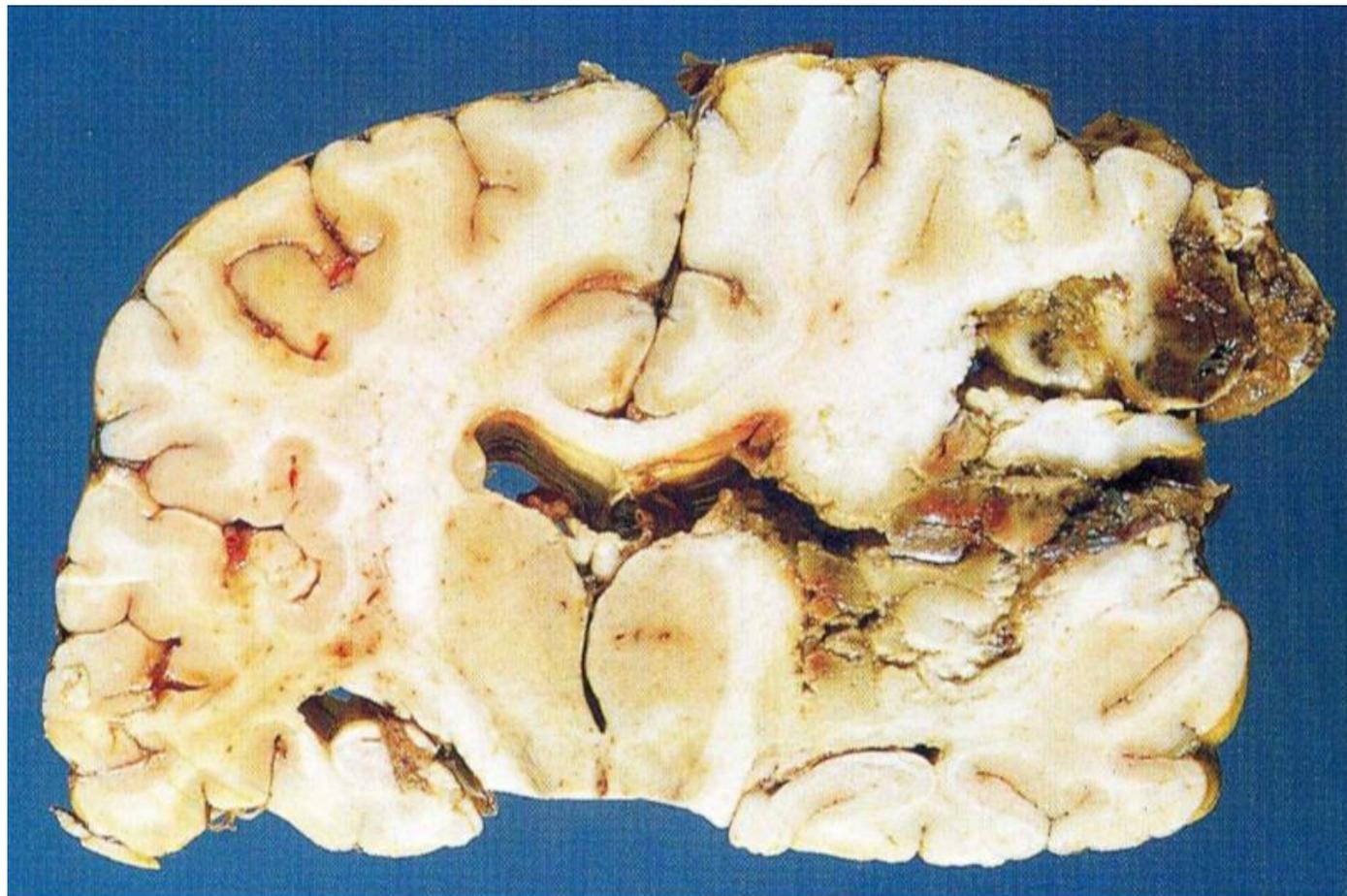
□ **Hemorrhagic stroke:**

- Hematoma,
- Hemorrhagic impregnation
- Subarachnoid hemorrhage.

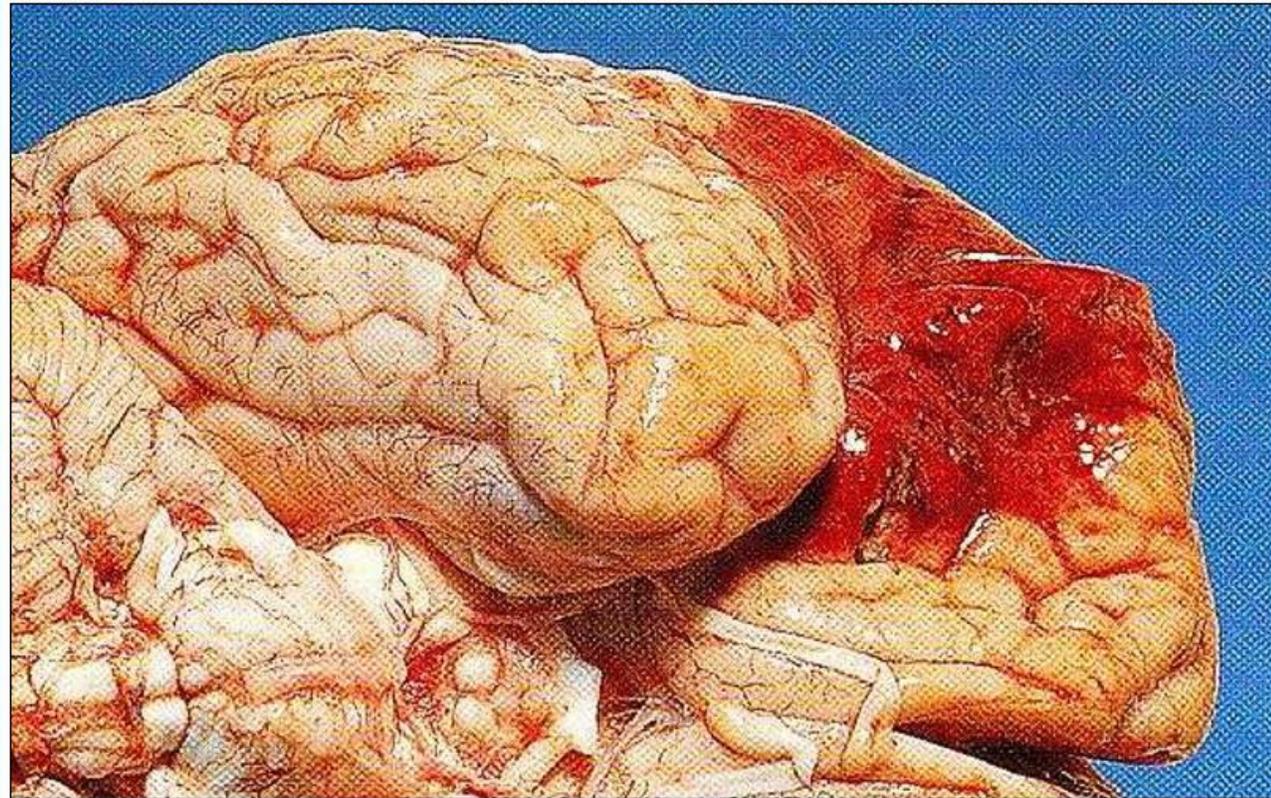
ISCHEMIC ENCEPHALOPATHY

- ❑ It is associated with chronic ischemia caused by stenosing arteriosclerosis of the cerebral arteries.
- ❑ Ischemic damage to neurons (primarily pyramidal cortical cells and Purkinje cerebellum cells) is characterized by the development of coagulation necrosis and apoptosis.
- ❑ Gliosis develops on the site of dead cells.
- ❑ With prolonged existence, cortical atrophy may develop with the development of dementia.

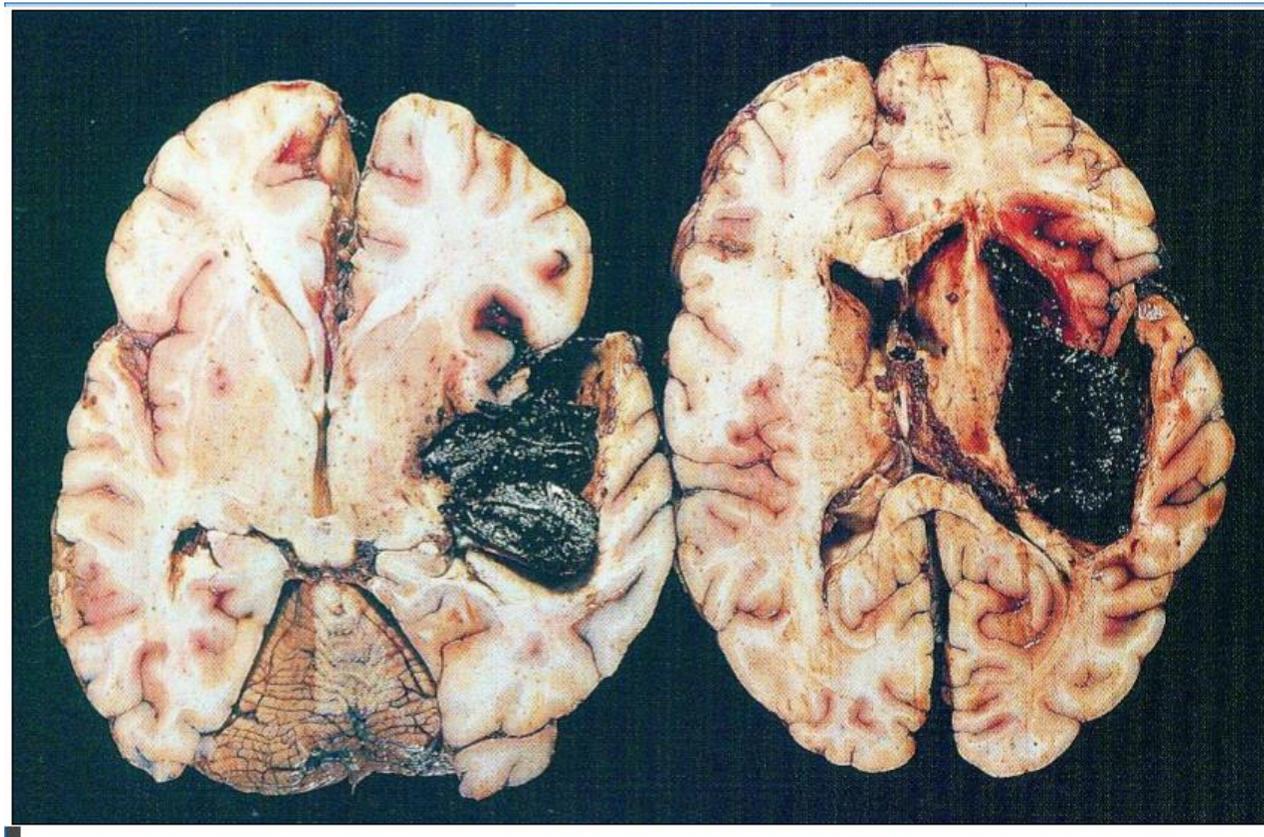
ISCHEMIC INFARCTION OF BRAIN



HEMORRHAGIC INFARCTION OF BRAIN



BRAIN HEMATOMA



HEMORRHAGIC IMPREGNATION WITH TAMPONADE OF THE VENTRICLES OF THE BRAIN



SUBARACHNOID HAEMORRHAGE

