



ZAPOROZHZHIAN STATE MEDICAL UNIVERSITY
the department of pathological anatomy and forensic
medicine with basis of law

Cerebral-Vascular Diseases (CVD)

Ischemic Heart Diseases (IHD)

Lecture on pathomorphology for the 3-rd year
students

CVD - it is a cerebral variant of atherosclerosis
IHD - this is a cardiac variant of atherosclerosis

Common pathological changes of vessels at CVD and IHD

- ? atheromatosis and athero-calcification of arteries with stenosis
- ? circular hyalinosis with the critical narrowing of heart or brain vessels at the patients with hypertension disease

VASCULAR DISEASE OF THE NERVOUS SYSTEM

1. Vascular-discirculation encephalopathy:

Ischemic

Hypertensive

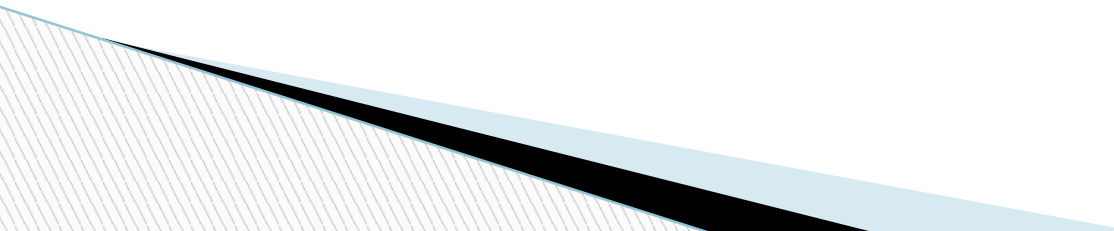
2. Cerebral haemorrhage:

? Intracerebral

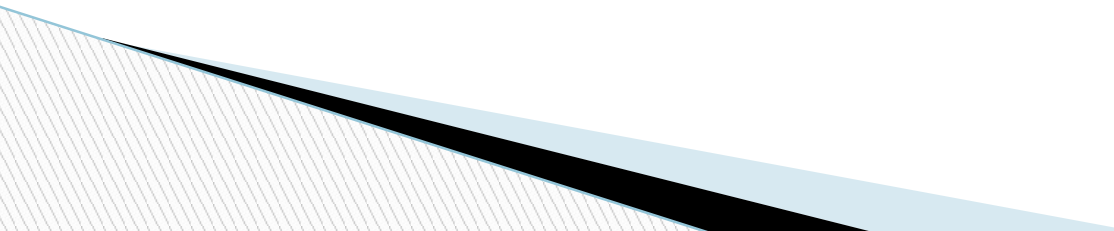
? subarachnoidal

3. Brain stroke (ischemic, hemorrhagic, ischemic infarction with haemorrhages)

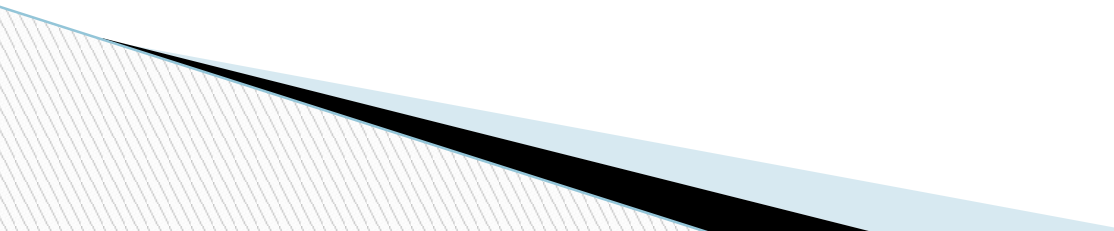
The ischemic encephalopathy (IE)

- ? It is a diffuse defeat of brain neurons with diffuse small-part character necrosis of neurons and hyalinosis of vessels.
 - ? IE develops at the decrease of cerebral blood-volume less than 25-10 ml on 100gr of tissue.
 - ? At the decreasing of cerebral blood-volume in 2 times the ischemic damage of neurons is observed.
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Reasons of the decreasing of cerebral blood-volume

- ? stenosis of cerebral arteries
 - ? thrombosis of the atherosclerotic plaque
 - ? protracted spasm of vessels
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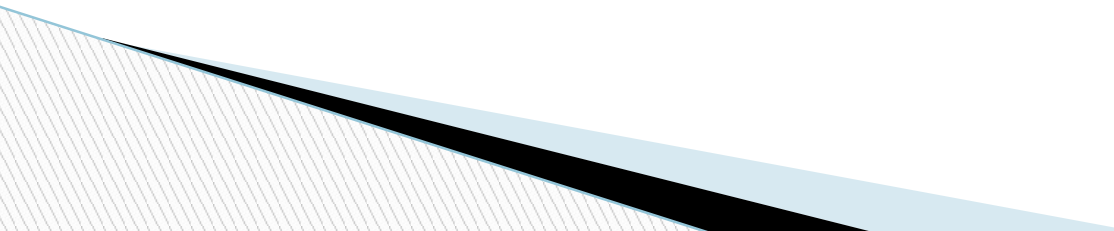
The ischemic encephalopathy (IE)

- ? Laminar necrosis - ischemic changes of pyramidal cell layers of the cerebral cortex.
 - ? Adaptive (around-neuronal) satellitosis - glial cells are gathered round neurons.
 - ? Zones of gliofibrosis are observed in the place of necrotic changes.
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The ischemic encephalopathy (IE)

- ? Acute
- ? Sub acute
- ? Chronic with relapses (at seniors with the expressed atherosclerosis)

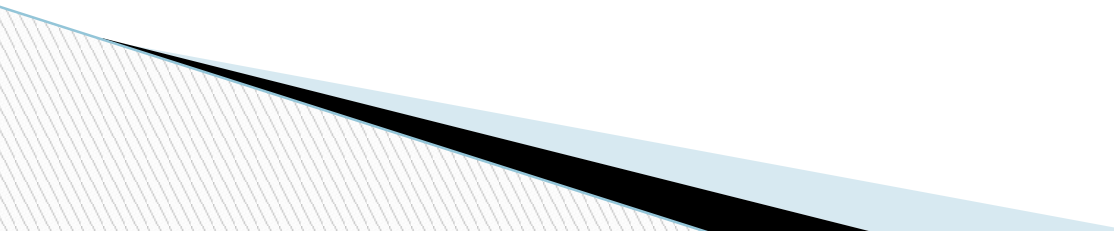
Outcomes of IE

- violations of sensitiveness
 - violations of motions
 - violations of memory
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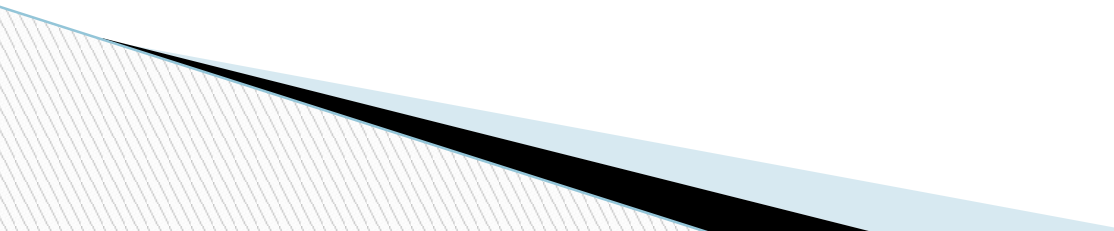
The hypertensive encephalopathy

- ? It is hypertensive hyaline arteriolar sclerosis. At the moment of crisis a fibrinoid necrosis of the arteriole walls of brain is observed, it leads to vascular-genic edema of brain (acute swelling).
- ? The dislocation (herniation) of brain begins into the natural opening (foramen magnum);
- ? The cortex layer of brain stake is hurt in the process of dislocation;

The hypertensive encephalopathy

- ? Haemorrhage begins in the upper 1/3 of Pons (in the zone of cardio-respiratory centers).
 - ? Displacement of cerebellum in foramen magnum leads to compression of basal artery and ischemia of cardio-respiratory center.
 - ? The diapedesis haemorrhage arises up round vessels, so the cavities with haemosiderophages are formed. They are named - lacunar infarcts.
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The hypertensive encephalopathy

- ? Lacunar infarcts ("lacunae") are little infarcts, a few mm across, typically in the deep structures of the brain
 - ? In fatal cases, necrotic changes of blood vessels are seen, much like in the kidney at "malignant hypertension".
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Outcomes of HE

- ? death in the acute period
- ? the progressive disorders of memory, sensitiveness, motions and etc

“Brain Stroke“ -

- ? it is a sudden onset of a permanent, localized neurologic deficit, may result either from hemorrhage (1) or infarction (2), and has a multitude of specific causes.

The infarction of brain

- ? ischemic infarction (75%) - develops at the obstructive thrombosis or thrombi-emboli
- ? ischemic infarction with hemorrhages (5-10%) - at embolism of vessels
- ? hemorrhagic infarction - "anemic infarcts" complicated by dissolution of an embolus or backflow of blood from the margins.

Clinic: hemiplegia and disorders of sensitiveness on the other part of defeat, and disorders of speech at the involving of cortex of brain.

Reasons of brain infarcts

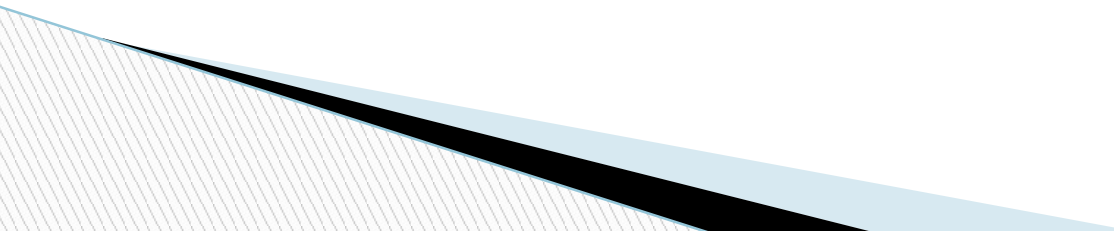
- ? Thrombotic infarcts
- ? Embolic infarcts
- ? Subclavian steal syndrome (Robin Hood syndrome), in which a patient with occlusive atherosclerosis of a proximal subclavian artery suffers brainstem syndromes upon exercising the arm on the involved
- ? Granulomatous angiitis of the CNS
- ? Moyamoya disease - the process in which the vessels of the Willis circle and nearby become narrowed (fibrosis of the intima) and may bleed.

The evolution of brain infarction (stages)

- ? Ischemia (after 2-3h) with bright structure and
- ? hyperdense features, swollen sulci and gyri
- ? necrosis, swelling, necrosis, liquefactive
- ? necrosis, formation of cyst

?

Brain hemorrhage

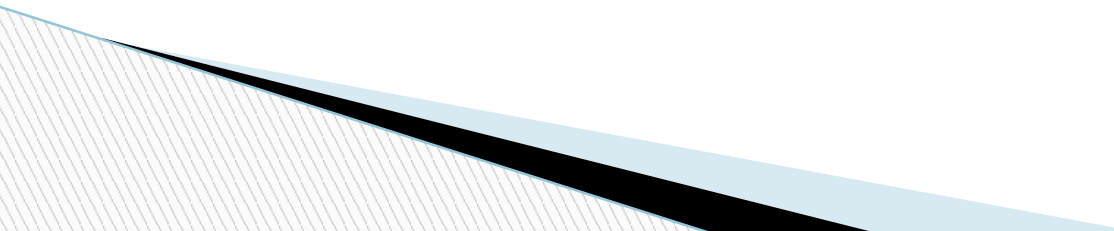
- ? Sudden arising up of the volume in one hemisphere of brain brings to the rapid dislocation of brain & death.
 - ? The haemorrhage mass can break through into the ventricles of brain on any stage that leads to coma. The second trunk syndrome develops (defeat of reticular structure).
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Brain hemorrhage. Reasons.

- ? "Hypertension" - arterial pressure higher than 180mmHg item
- ? the break of artery, or aneurism, or vascular malformations ("angiomas")
- ? bleeding disorders
- ? hemorrhage into brain tumors (primary, metastatic)
- ? Congo-philic (amyloid) angiopathy (hereditary, idiopathic; "Alzheimer's amyloid angiopathy")

Brain hemorrhage. Classification.

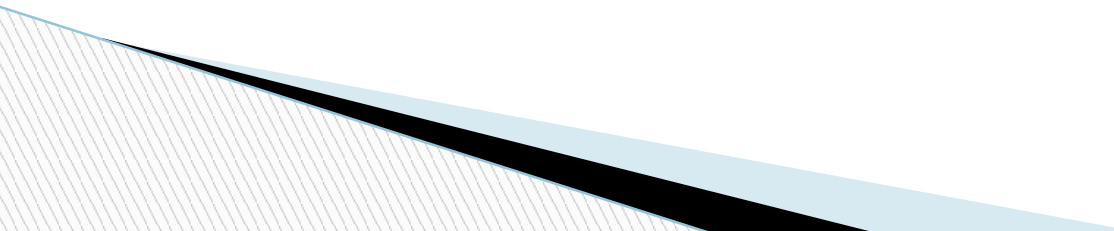
- ? Intra-brain - in area of under-cortex ganglier and visual hillock, rarely in the cerebellum and trunk of the brain
 - ? Sub-arachnoidal hemorrhage.

 - ? **According to morphology features**
 - ? hematoma - massive bleeding
 - ? hemorrhagic infiltration.
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The sub-arahnoidal hemorrhage - reasons of development

- ? Break off innate or acquired aneurism.
- ? Vascular malformations - may bleed into the subarachnoid space, the brain substance, or both. Arteriovenous malformations (masses of large blood vessels) tend to be located in the hemispheres
- ? Germinal plate hemorrhages in premature babies - bleeds into the ventricles, rather than the subarachnoid space.
- ? Atherosclerotic aneurysms in the head are typically fusiform dilatations of the basilar

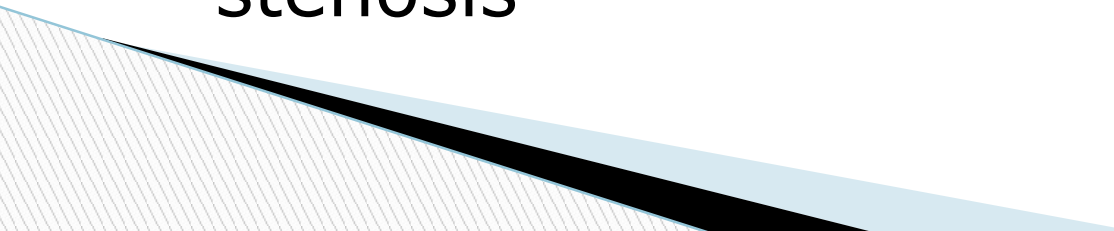
Classification of Ischemic heart disease

- ? **Acute IHD:** angina pectoris, acute coronal insufficiency, acute myocardial infarction, repeated myocardial infarction, Sudden cardiac death
 - ? **Chronic IHD:** stenosis and occlusion of coronary arteries, postinfarction cardiosclerosis, chronic aneurism of heart wall.
- 

Ischemic heart disease

- ? It is disease that is conditioned by the relative or absolute insufficiency of coronal blood supplying that is secondary leads to irreversible changes of myocardium.

CAUSES

- ? Atherosclerosis of coronal arteries
 - ? Concentric hyalinosis and circulation stenosis
- 

Angina pectoris

? It is disparity between necessities of oxygen and its supplying to myocardium.

Reasons of development:

1. Prolonged spasm of coronal arteries at hypertension disease. Spasm that is longer than 60 minutes leads to myocardial infarction.
2. Coronal stenosis at atherosclerosis
3. Circular hypoxia at: cardiomyopathies, arrhythmias, heart vices, heart de-compensation

Angina pectoris

- ? **Stable** ("classic") **angina** - results from increased work in a patient with coronary atherosclerosis, and relieved by rest.
- ? **Unstable** ("acute coronary insufficiency") **angina** - due to a thrombus developing, by fits and starts, over a ruptured plaque. In duration less than 60 minutes.

Prinzmetal's angina - primarily attributable to vasospasm. Sudden cardiac death can be observed at this patients.

Cardiac syndrome X ("microvascular angina") classical clinical angina and wide-open coronary arteries

Acute coronal insufficiency

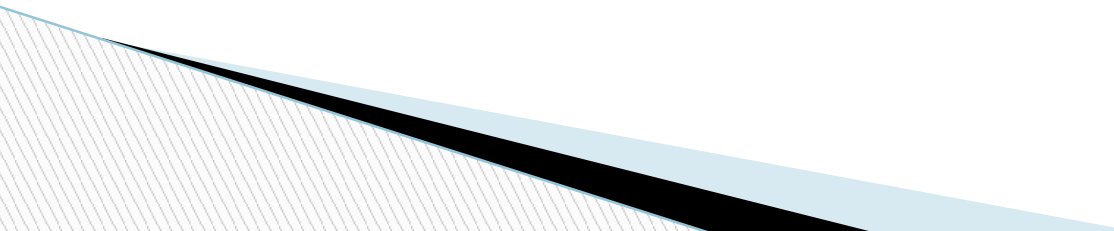
? It is inability to satisfy metabolic necessities of myocardium by coronal blood supplying.

Reasons of development:

- ? Brief spasm of coronal arteries (less than 60 minutes)
- ? Brief increasing of concentration of catecholamine at stress
- ? Physical overload at stenosis of one artery (haemodynamic disturbances)

Acute coronal insufficiency.

Complications and outcomes:

- ? Reperfusion post-ischemic damage of myocardium by free radicals, ions, ets.
 - ? Damage by mediators of platelets, toxins leucocytes and lymphocytes
 - ? Local necrosis and apoptosis of cardiomyocytes
 - ? Damage of endothelium that leads to thrombi formation
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Myocardial infarction

- ? It is ischemic partial necrosis of myocardium wall due to sudden loss of the blood supplying.

Myocardial infarction. Reasons.

- ? Atherosclerosis: ruptured plaque - often with an overlying thrombus ("coronary thrombosis"); massive haemorrhage into a plaque, ballooning its cap against the opposite wall.
- ? Prolonged spasm of coronal arteries - more than 60 minutes in duration
- ? Physical overloading of patient with critical stenosis of coronal arteries (more than 75%)
- ? Thrombosis of coronal arteries
- ? Cocaine use, Prinzmetal's coronary spasm, Vasculitis, Embolization, Syphilis, other

Myocardial infarction. Classification.

- ? **According to localization and spreading:** sub-epicardial, sub-endocardial, intramural, transmural
- ? **According to time of development:** acute primary - 4 weeks from the beginning, recidivating (relapsed) - the formation of the new necrosis during 4 weeks on the background of primary infarction, repeated - the formation of the new necrosis after 4-th week from the beginning of 1-st one.
- ? **According to the stage of development:**
 - Ischemic stage - 12-18 hours
 - Stage of necrosis - 18-24 hours up to 5 days
 - Stage of organization - 5 days - 7 weeks

Morphological characteristics:

- ? ischemic - through 60 seconds, after stopping of blood-circulation, the abbreviation of myocytes is halted, but during the 1-st days a nuclear is stored, and membranes of organell's gradually collapse (picnosis and eosinophylia of cytoplasm)
- ? necrosis - in a 24 hour from the beginning of ischemia (kariolysis, kariopiknosis) of about 5-7 days, grows myomalyatsia of heart walls (wall is yellow-green), on periphery - hemorrhagic halo.
- ? organization - into the area of necrosis vessels grow up and migrate fibroblasts - zone of cardiosclerosis. A scar is formed by the end of 2th month.

Diagnose of ischemic stage of infarction during autopsy

- ? The nitro-blue tetrazolium technique can demonstrate early myocardial infarcts.

Drop a slice of heart in the solution, and viable heart, containing an oxidizing enzyme, will stain brown, and dead heart remain pale.

Complications of myocardial infarcts

- ? **Ischemic stage:** rhythm disturbances with stopping heart work, Left-sided congestive heart failure, Cardiogenic shock, Acute coronal insufficiency
- ? **Stage of necrosis:** Rupture of the heart - occur, when the damaged heart is most soft (days 3-5), Formation of acute aneurysm, Mural thrombus formation in aneurism and embolization, Rupture of the wall of acute aneurism, Dressler's pericarditis (fibrin pericarditis)

Complications of myocardial infarcts

Stage of organization

- ? Formation of chronic aneurysm.
 - ? Near-wall mural thrombus formation in chronic aneurism and embolization
 - ? Formation of recidivating (relapsed) infarction
 - ? Progression of myocardial insufficiency
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