



**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-calcinosis 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

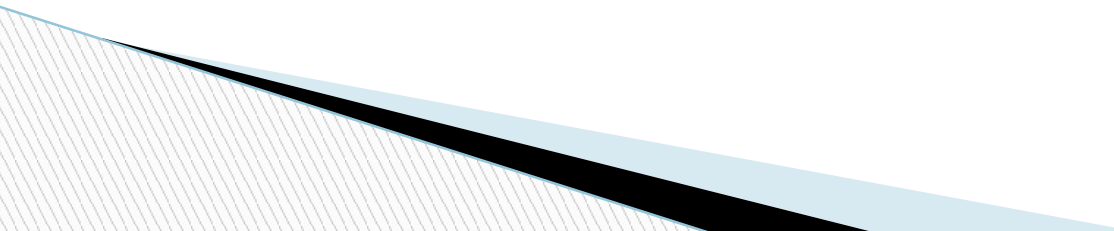
2. Cerebral haemorrhage:

□ Intracerebral

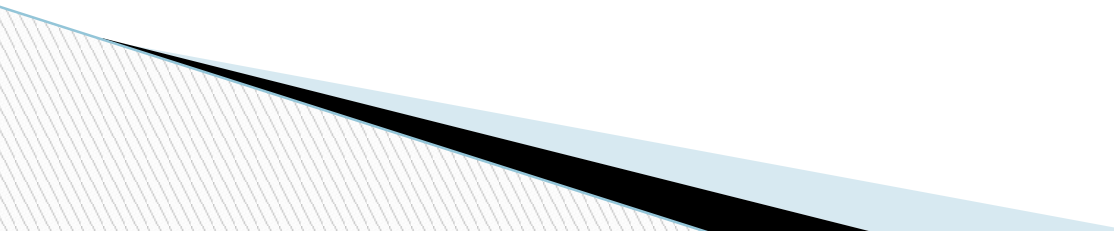
□ subarachnoidal

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

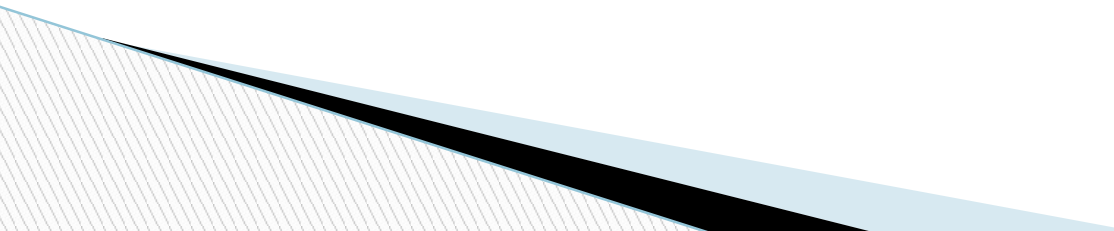
# The ischemic encephalopathy (IE)

- 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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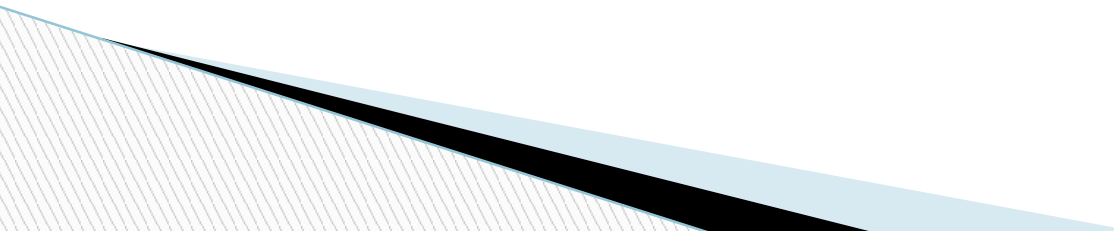
# The ischemic encephalopathy (IE)

- 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

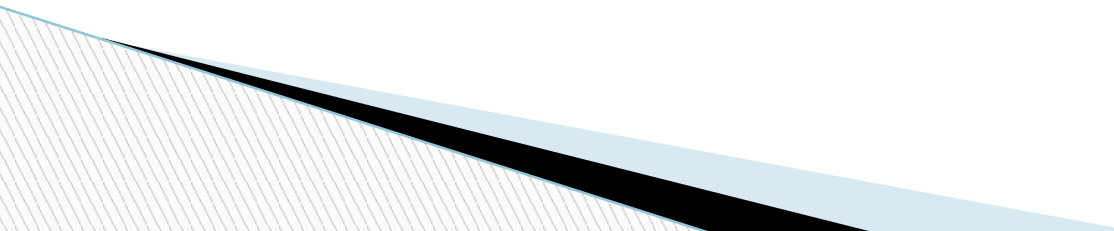
- violations of sensitiveness
  - violations of motions
  - violations of memory
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# The hypertensive encephalopathy

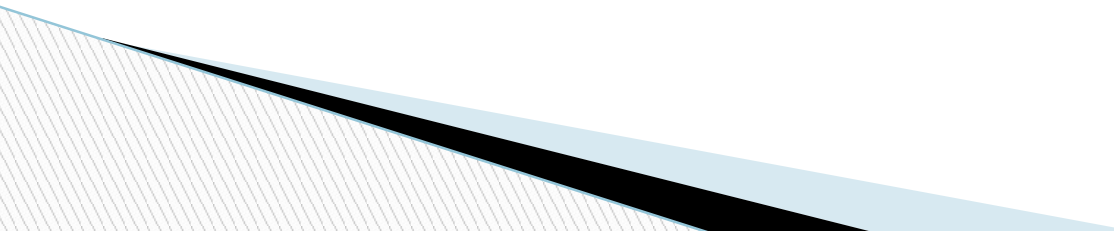
- ▣ It is hypertensive hyaline arteriolar sclerosis. At the moment of crisis a fibrinoid necrosis of the arteriole walls of brain is observed, it leads to vascular-genic edema of brain (acute swelling).
- ▣ The dislocation (herniation) of brain begins into the natural opening (foramen magnum);
- ▣ The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



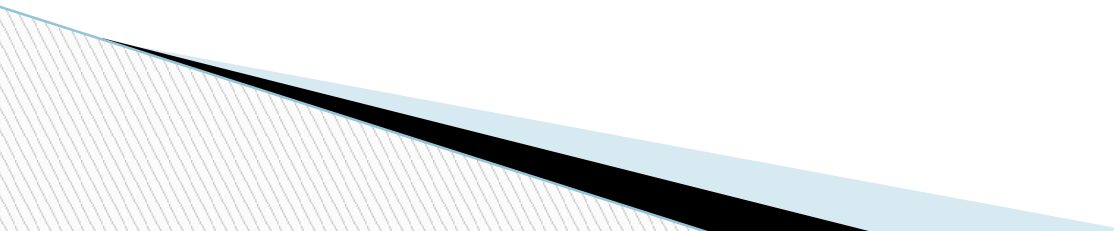
# The hypertensive encephalopathy

- 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
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## “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

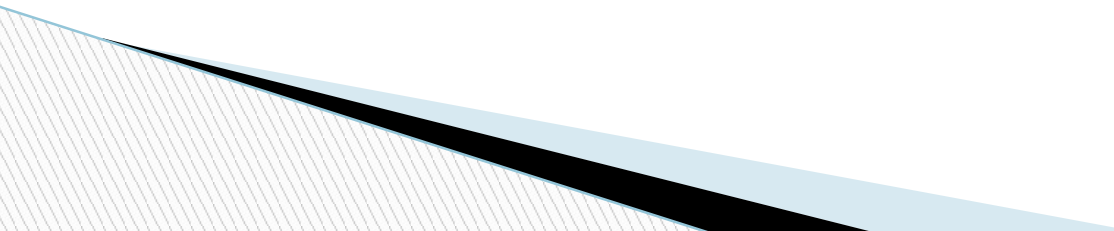
- Thrombotic infarcts
- Embolic infarcts
- Subclavian steal syndrome (Robin Hood syndrome), in which a patient with occlusive atherosclerosis of a proximal subclavian artery suffers brainstem syndromes upon exercising the arm on the involved
- Granulomatous angiitis of the CNS
- Moyamoya disease - the 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)

- Ischemic (often 2-5) with signs of anoxia and
- necrosis, liquefactive necrosis, swollen glial cells, macrophages
- necrosis, formation of cyst

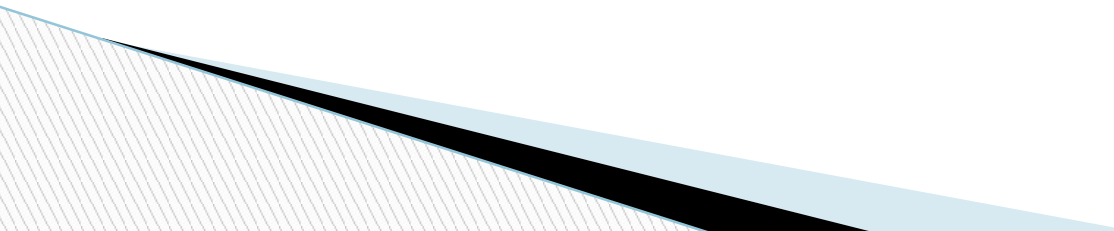
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# Brain hemorrhage

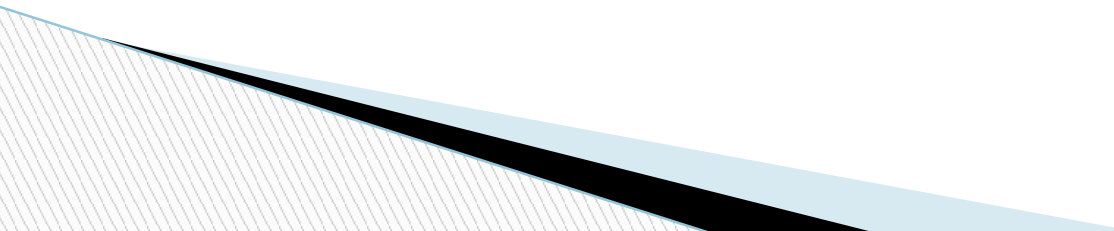
- Sudden arising up of the volume in one hemisphere of brain brings to the rapid dislocation of brain & death.
  - The haemorrhage mass can break through into the ventricles of brain on any stage that leads to coma. The second trunk syndrome develops (defeat of reticular structure).
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#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

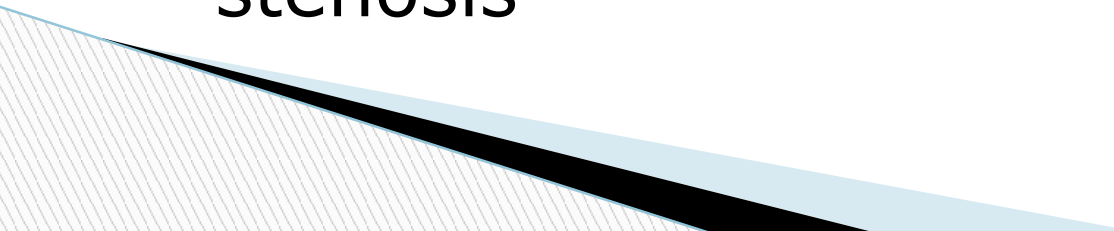
# Classification of Ischemic heart disease

- **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
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# 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.

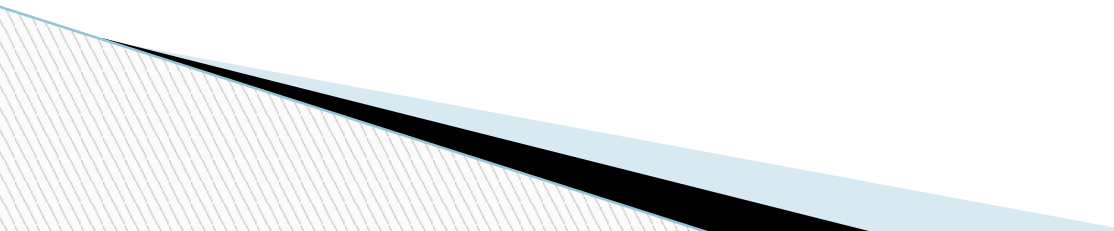
**Prinzmetal's angina** - primarily attributable to vasospasm. Sudden cardiac death can be observed at this patients.

**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.

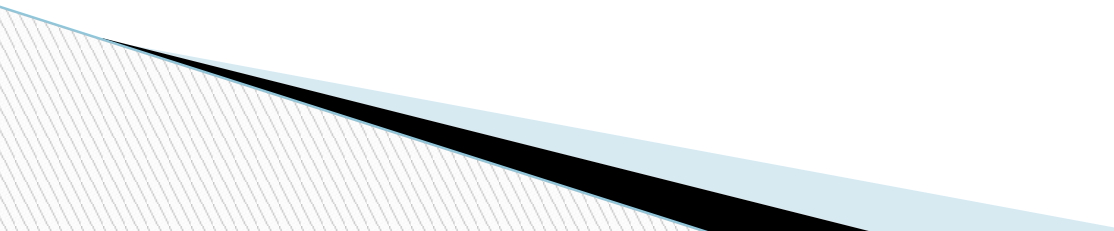
## Reasons of development:

- 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)
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# 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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