

## LECTURE 6

### **Disorders of blood circulation:**

**hyperemia, ischemia, infarction, bleeding, hemorrhage, stasis, plasmorrhagia. Embolism. Shock. Disorders of lymph circulation. Disturbances of hemostasis. Thrombosis, DIC-syndrome.**

### **Hyperemia**

#### **plethora (arterial and venous)**

##### ■ **Arterial plethora.**

is organ or tissue intensified blood filling caused by excessive arterial flow.

- acute or chronic,
- physiological or pathological,
- general or local.

##### **General plethora.**

■ develops under circulating blood volume increase (plethora) or number of erythrocytes increase in blood (erythroemia, Vaquez's disease)

Skin, visible mucous tunics redness (plethora), blood tension increase is observed at that.

##### **Local arterial plethora.**

could be **physiological** and occur under shame, heavy manual labor, organs hyperfunction (work plethora) and **pathological**. The following kinds of local pathological arterial plethora are differentiated:

- Angioneurotic (neuroparalytic plethora)
- Collateral plethora
- Plethora after ischemia
- Vakatn plethora
- Inflammatory plethora
- Plethora based on arteriovenous fistula

##### Angioneurotic (neuroparalytic plethora)

■ is observed under vasodilatating nerves irritation or vasoconstrictor nerves paralysis. Skin, mucus tunic becomes red, slightly swollen, warm or hot by touch. This plethora could occur on certain body portions under innervation's failure, sympathetic nervous system nodes failure. For example same side face skin redness is observed under croupous pneumonia. As a rule this plethora passes without trace.

##### Collateral plethora

■ occurs because of blood flow hindrance in main artery lumen of which is closed with thrombus, embolus or artery is squeezed with tumor. Blood comes to bloodless portion by collateral vessels, lumen of which is reflex dilated. In case collaterals insufficient development tissue and ischemia or even necrosis develops.

##### Plethora after ischemia (post ischemic)

■ occurs in cases when the cause of artery squeezing (tumor, ligature, liquid accumulation in cavity) is eliminated rather quickly. Under these circumstances vascular lumen of former bloodless tissue is sharply dilated and overfilled with blood which can cause its rupture and hemorrhage. Besides that ischemia occurs in the other organs because of bloods redistribution, for example cerebrum ischemia can occur with vertigo. So ascetic liquid should be slowly released from abdominal cavity. In case vertigo caused by cerebrum ischemia occurred in the result of blood redistribution it's necessary to place patient's body in such a way to provide low position of the head.

##### Vakatn plethora

■ is caused by atmospheric pressure decline. General vacant plethora occurred with divers and pilots under fast lift from high pressure into low pressure area. In such cases it is combined with gas embolism. An example of local vakatn plethora is redness in the place of gallipots.

##### Inflammatory plethora

■ is caused by action of biologically active substances – inflammation mediators, for example, histamine, serotonin. At this in the place of injury arterioles are dilated after short time reflex spasm of them. Most of all it relates to postcapillares and venules lumens, local redness and temperature rise. Plethora facilitates metabolism intensification in inflammable zone tissue, neutrophilic leucocytes (microphages) migration in tissues, microorganisms elimination, that is of defensive character.

##### Plethora based on arteriovenous fistula

■ occurs in those cases when, for example under gunshot wound or tumor injury joint between artery and vein is formed and arterial blood overfills venous vessels because of tension difference.

## Venous plethora

■ *Venous plethora is organ or tissue blood filling of increase caused by slow (hindered) blood outflow, blood flow at that is not changed or decreased.* Venous (passive) congestion causes dilation of veins, venules, capillaries, blood flow slowing down in them causing development of hypoxia, capillaries wall penetrability increase, edema and tissue trophism disorder.

Venous plethora

could be

- general
- local
- acute
- chronic

### **General venous plethora**

■ *General venous plethora* occurs under cardiac pathology causing heart failure.

### **Acute general venous plethora**

occurs under acute cardiac insufficiency (myocardium infarction, acute cardiac decompensation)

Chronic venous plethora

■ occurs under chronic cardiac (cardiovascular) insufficiency, which develops under congenital and acquired cardiac malformations, myocarditis, cardiosclerosis.

Morphology of acute *venous plethora*

(for example in lungs under left ventricle infarction)

- plasm extravasation (plasmorrhagia),
- edema,
- punctuated diapedesis bleeding,
- degenerative and necrotic changes in parenchymatous elements,

Morphology of chronic *venous plethora*

- not only plasmorrhagia,
- edema and punctuated diapedesis bleeding
- but also tissues and organs atrophy and sclerosis. Sclerotic changes are caused by the fact that **hypoxia** stimulates collagen synthesis by fibroblasts; simultaneously parenchymatous elements atrophy occurs. In such a way parenchyma is substituted with conjunctive tissue, organs and tissues thicken – their induration occurs.

Morphology change in skin by chronic *venous plethora*

- -especially legs' skin becomes cold and of bluish color (cyanosis).
- (Blue color is caused by the reduced hemoglobin (without oxygen) which is of bluish color).
- -veins and cutis lymphatic vessels are dilated, overfilled with blood,
- -derma and subcutaneous fat are edematous.
- -Conjunctive tissue enlargement is manifested with skin induration.
- -Inflammation pyogenic abscesses and trophic ulcers

Morphology change in Liver by chronic *venous plethora*

- - Liver is enlarged
- -Liver is Hard.
- -Section surface is striped – dark red spots are seen on grey-yellow background,
- - looking similar to nutmeg section - nutmeg liver.

Morphogenesis of the nutmeg liver

- -Under general venous plethora blood outflow from the liver is hindered, hepatic veins are dilated.
- -Central veins of the parts and central sections of sinusoids supplying blood to the central veins also dilates.
- -Dilated central veins and central sections of sinusoids create “bloody lakes” in the center of the parts causing dark-red spots.
- -In case plethora intensification hemorrhages occur in the center of the parts.
- -Hepatocytes situated in the center of the lobules (centroclinal) atrophy because of dilated vessels' compression, degenerative changes and necrosis develop in them.
- -At this parts periphery hepatocytes compensatory hypertrophy.
- -In the result of hypoxia adipose degeneration occurs in hepatocytes, causing grayish-yellow color of liver.
- -Hypoxia facilitates conjunctive tissue excrescence, due to that sinusoids walls thicken causing hepatocytes hypoxia extension.

■- Venous plethora intensification causes hepatic sclerosis (fibrosis) progress which is finalized with congestive (nutmeg) hepatic cirrhosis. In such a way as time passes hepatic insufficiency joins cardiac insufficiency.

Nutmeg liver. Parenchyma at section is striped has nutmeg appearance: dark brown spots on light yellow background. Light yellow color of liver is caused by hepatocytes' adipose degeneration, dark brown specks – blood lakes (dilated central veins and sinusoids).

Morphology change in lungs by chronic *venous plethora*

■-brown hardening (induration) develops

■Aetiology- mitral or aortic valves failure or left ventricle cardiomyocytes injury

. Lung hyperemia. Colored with hematoxylin and eosin. Arteries' and capillaries' lumens are dilated, plethoric. Erythrocytes situates also in alveoli's lumen.

Morphogenesis

■- Pulmonary venous blood congestion occurs on condition that right ventricle of heart pumps blood into lungs and left ventricle can not provide this blood pumping from the lungs into aorta. **Blood accumulates in pulmonary artery** pond, hypertension occurs in lesser (pulmonary) circulation. As a result of hypertension **microcirculation channel vessels dilate and capillary walls permeability increases**. Besides that capillary walls permeability is caused by intensifying **hypoxia**. Blood liquid phase sweats from capillaries accumulating in alveoli's lumen, pulmonary edema develops. As hypoxia and hypertension intensify in lesser circulation capillary walls permeability becomes more expressed - numerous **diapedetic hemorrhages** occur, meaning erythrocytes' sweating from vessels lumen into surrounding tissues. Out of vessels they are treated by tissues as foreign and are absorbed by macrophages. **Hemoglobin transforms in them into hemosiderin** (ferrum containing pigment). Further on macrophages are destroyed and hemosiderin under insufficient lymph flow deposits in stromal tissues. Lungs obtain brown color. Macrophages in which hemosiderin forms are called siderophages. Alveolocytes also have macrophage function and those of them which are found in patients' with cardiac decompensation sputum are called cardiac failures' cells. Thus, rusty-brown color of lungs under chronic venous plethora is caused by hemosiderin which situates in macrophages as well as in interalveolar partitions, alveolar lumens, bronchi' walls and lumens, lymphatic vessels and lymph nodes.

Morphogenesis Lungs (induration) under chronic venous plethora

■1. Tissue hypoxia activates fibroblasts, latter actively fissure, synthesize collagen fiber and intracellular substance causing conjunctive tissue growth leading lungs' thickening.

■2. Under lungs' venous plethora lymphatic system's absorption and dynamic insufficiency causing congestion of fluid in tissues and tissues proteins accumulation. Tissue fluid accumulation enhance hypoxia, that in its turn leads to sclerosing.

■3. Free hemosiderin also contributes tissues sclerosing.

■In such a way lungs become large, thick, of rusty-brown color on surface and in section. Thus lungs insufficiency joins cardiac decompensation.

Morphology change in kidneys by chronic *venous plethora*

■kidneys become large and cyanotic (cyanotic induration), the most plethoric are cerebral layer veins and intermediate area veins. Cyanotic color is caused by organ's overfilling with venous blood. Enhancing hypoxia causes

■-parenchymatous elements degeneration and

■conjunctive tissue excrescence, leading to organ's hardening.

■Similar changes develop in spleen, cerebrum and other organs. Skin, especially legs' skin, becomes cyanotic, cold to touch, hard.

■Fig.2. Cyanotic induration of kidney. Dark-blue color dominates on section, it is the most full-blown in medullar layer). Cyanosis is caused by the fact that kidney is overfilled with venous blood. In conditions of hypoxia the process of collagen formation by fibroblasts activates, so sclerosis occurs (induration, thickening).

*Local venous plethora*

- develops in case hindrance of blood outflow from specific organs or parts of the body,

Aetiology:

-vein obstruction with clot, embolus

- vein contraction by tumor, enlarged neighbor organ.

For example,

■acute venous plethora of gastrointestinal tract occurs under portal vein thrombosis. Under hepatic veins' thrombosis or in case their obliteration caused by thrombophlebitis nutmeg liver disease (Budd-Chiari syndrome) develops.

■Kidneys' venous plethora can develop under thrombosis of their veins. Under local venous plethora venous blood outflow partially goes through collaterals.

Result venous plethora

■Sometimes collateral veins are so much overfilled with blood that their varicose develops. Such varicose nodes (knots) can burst because of their wall atrophy, causing hemorrhage, sometimes fatal. For example, under portal vein blood congestion at hepatocirrhosis port-canal anastomosis develop causing varicose of low one-third of esophagus veins. Varicose node burst causes significant hemorrhage, sometimes fatal.

### Ischemia

■ischemia (from Lat. ischo – block ) is organ, tissue or part of the body blood filling' reduction caused by insufficient blood inflow.

■*Morphology* change in organs by ischemia

■Ischemic tissue becomes pale,

■ flaccid,

■organ decrease in size,

■its capsule shrinks.

Morphogenesis change in organs by ischemia

■tissue' oxygen shortage (hypoxia) occurs, metabolism slows down, reductive-oxidative ferments activity decreases, mitochondrion destroy, glycogen disappears, degenerative and necrobiotic changes develop, in first turn of parenchymatous elements. Tempo of described changes depends on ischemia development (acute and chronic ischemia). Under complete blood supply cessation ischemised portion necrosis occurs (infarction). Under chronic ischemia parenchymatous elements degeneration and atrophy develops as well as conjunctive tissue enhanced excrescence (sclerosis).

Classification of ischemia

■There are acute and chronic ischemia.

■ *acute ischemia* Under complete blood supply cessation ischemised portion necrosis occurs (infarction).

■ *chronic ischemia* parenchymatous elements degeneration and atrophy develops as well as conjunctive tissue enhanced excrescence (sclerosis).

**Depending on courses and conditions of origination:**

■1. Spastic (reflex),

■Obstruction,

■Compressive,

■ Ischemia caused by blood redistribution

### Ischemia

■1. Spastic (reflex) – arteriospasm under painful stimulation, negative emotions.

■2. Obstruction – partial or complete obstruction of artery with thrombus, embolus, spalled atherosclerotic plaque, conjunctive tissue grew after arterial wall inflammation (obliterating endarteritis).

■3. Compressive – artery contraction with tumor, exudates, ligature, tourniquet.

■4. Ischemia caused by blood redistribution. Under ascitic fluid drain blood outflows to abdominal cavity and brain ischemia develops. Blood outflows in lower situated portions of the body in cases person tries to stand up quickly, brain ischemia occurs with giddiness, orthostatic shock develops, that is loss of consciousness.

### Stasis

■Stasis (from Latin stasis – arrest) – blood circulation arrest in microcirculation channel vessels, mainly in capillaries.

■Aetiology *stasis*:

■-blood clotting under capillary walls increased permeability,

■- under plethora,

■- hypoxia,

■- vasculitis,

■- high and low temperature's action,

■- allergic diseases.

## Stasis

- Blood circulation arrest is preceded by blood circulation slowing down which is *pre-stasis* condition.
- In *stasis* development mechanism changes of blood flow characteristics expressed with enhanced erythrocytes' intracapillary aggregation are of main importance. It leads blood capillary flow hindrance, slowing down and arrest. Under stasis hemolysis and blood coagulation doesn't occur. Erythrocytes aggregation is called *sludge-phenomenon*. Erythrocytes stick together forming so called coin columns causing blood viscosity increase.
- Stasis is *reversible* phenomenon.
- Condition after its release is called *post-stasis*.
- Irreversible condition leads to *dystrophy* and tissue and organ cells' *necrosis*.
- Stasis in cerebrum capillaries. Erythrocytes are of black color. Find the portion where cerebrum capillaries are clearly seen filled with erythrocytes in the form of black erythrocytes chain – stasis.

## Plasmorrhagia

- Plasmorrhagia is plasma going out blood circulatory channel, causing plasma leakage of vessel wall and degenerative changes development in it up to fibrinoid necrosis.

■ Epithelium edema and hardening takes place, choroids fissure dilates, basal membrane integrity is crippled.

- Plasmorrhagia consequence is transcapillary metabolism failure and fibrinoid necrosis development or vessels' hyalinosis.

## Aetiology Plasmorrhagia

- -nerve-vascular failures (spasm) – hypertension disease,
- -tissue hypoxia – decompensated cardiac diseases,
- -immunopathologic reactions – autoimmune reactions,
- -vasoactive substances (serotonin, histamine) amount increase in blood
- -infection, infection-allergic diseases, coarsely dispersed proteins,
- -lipoproteins – atherosclerosis.

## Hemorrhage

- Hemorrhage (*haemorrhagia*) is blood outcome from vessels lumen or heart into environment (*external*) or into body cavities (*internal*).

### Terminology

- External hemorrhages from lung (hemoptysis) – haemoptoe, External hemorrhages from nose – epistaxis,
- blood vomiting– haematemesis,
- blood in excrements – maelena,
- External hemorrhages from uterus – metrorrhagia.
- Internal hemorrhages:
  - blood accumulation in heart cavity hemopericardium,
  - blood accumulation in pleura – hemothorax,
  - blood accumulation in abdominal cavity – hemoperitoneum.

■ *Extravasations are accumulation of blood run out from vessels in tissues.*

### Kinds of extravasations

- hematoma,
- haemorrhage,
- petechia,
- echymosis,
- hemorrhagic infiltration.

### Kinds of extravasations

- Hematoma is clotted blood accumulation in previously damaged tissue. They are the most dangerous in cerebrum, adrenal glands. Hemorrhage – flat hemorrhages in skin and mucous tunics.
- Petechias, echymosis are small spot hemorrhages.
- Hemorrhagic infiltration – it is massive infiltration of tissue without basic and structural components destruction.

### *The causes of blood outgo from blood circulatory system*

- break (haemorrhagia per rhexin),
- erosion (haemorrhagia per diabrosin),

■vascular walls' permeability increase (haemorrhagia per diapedesis).

**Haemorrhagia per rexin** *Hemorrhages caused by vascular' wall or heart rupture.*

Aetiology:

■traumatic (mechanical origin) or pathological origin

(mostly by necrosis, inflammation, tumor). For example, under myocardial infarction, rupture of aorta's outgoing portion (over valve), under hypertension disease, necrosis of mid layer of aorta wall (medionecrosis), syphilitic mesaortitis.

■Sometimes rupture of cardiac aneurysm or aorta or other organs is observed caused by considerable increase and overdilatation of their capsule (enlarged spleen rupture under leucosis). Such ruptures occur even with minor trauma, for example, rough palpation.

**heart rupture**

**Haemorrhagia per diabrosin** *Hemorrhages caused by vascular walls erosion*

■Aetiology:

■inflammation, malignant tumors, necrosis. For example, proteolytic ferments action under inflammation, gastric juice, chorion villous growing-in under chorioepithelioma.

**Haemorrhahia per diapedesis**

Mostly shows up under arterioles, capillaries, venules injury.

■Aetiology:

■- hypoxia (cardiac, pulmonary insufficiency, ischemia);

■-vascular walls inflammation (vasculitis) under flu, measles, epidemic typhus, meningococcosis secondary syphilis, sepsis, scarlatina ,

■-avitaminosis – deficiency of vitamin – scorbutus.

■-blood flow features and blood coagulability characteristics change,

■- haematogenic organs failure (thrombocytopenia or Werlhof's disease, hemophilia, leucosis, ischemia).

■Diapedetic hemorrhages taken systemic character it's called *hemorrhagic syndrome*. Multiple dot hemorrhages are called hemorrhagic purpura or hemorrhagic diathesis.

***Consequences of bleeding, hemorrhages***

■– blood resolves more often, sometimes cysts are formed (cerebrum). Their content and walls are of chocolate color (chocolate cysts), the color is caused by hematogenous pigments. Sometimes blood coagulates and grow with conjunctive tissue – organization.

**Cerebral hemorrhage.** Hematoma with brain tissue destruction is seen in the right-brain.

***Hemorrhages significance.***

■In case aorta wall rupture death comes fast of heart ventricles filling deficiency caused by intracardial pressure sharp drop, even under minor blood loss. The condition of cardiac systole is sufficient intracardial pressure, as it is not made, heart stops in diastole. Autopsy shows in blood sags in endocardium (Minakov's spots), which occurs because of adhere by suction heart action (like after cupping glasses). In cases cardiac rupture its pressurization with blood comes - cardiac tamponade. Under considerable hemorrhage up to half mass of blood (2-2,5l) death comes from loss of blood. Long term hemorrhages repeating periodically under gastric ulcer disease, ulcerative colitis, menstrual period's failures, etc. lead to chronic ischemia, posthemorrhagic ischemia. The most dangerous is cerebral haemorrhage, and pulmonary hemorrhage at which death comes because of asphyxia as lumens of bronchi and trachea are obturated with blood.

Thrombosis

■**Thrombosis** is antemortem blood coagulation in lumens of vessels or heart. Formed grume is called thromb. Intravascular grume of lymph is also called thrombus.

***Aetiology of thrombosis***

■***Local factors***

■-endothelium damage,

■- blood flow laminarity slowing down and abnormality.

■***General factors:***

■imbalance between coagulative and anticoagulative blood systems

■ change of its composition.

***Pathogenesis of thrombosis***

■thrombocytes agglutination,

- fibrin formation,
- erythrocytes agglutination,
- blood plasma proteins' precipitation.
- Thrombocytes agglutination and their coagulation is one of important stages of thrombs formation.
- Under thrombocytes denaturation thromboplastic substances are segregated: active thromboplastin or thromboplastin which in the presence of calcium ions activate prothrombin which transforms into trombin.
- Fibrin formation goes on caused by coagulation or protein (fibrinogen) coagulation.
- Thrombin influences fibrinogen and fibrin-polymer forms. The process of blood coagulation proceeds in the form of cascade reactions.

### **Thromb morphology.**

- Thrombus consists of head, body and tail.
- head is fixed to vascular wall in the place of its damage, exactly where the process of thromb formation started.
- Thrombus is thick unlike postmortem grume, its surface is striped (Tsan's transverse lines) because of thrombo- cytes and fibrin rhythmic precipitation. Postmortem grume's surface is smooth, shining.
- There are white, red, mixed and hyaline thrombus - depending on regular elements of blood domination.

- *White Thrombus* – dominate leucocytes, thrombocytes and fibrin, it forms slowly under fast blood flow in arteries.
- Red Thrombus*, contain bigger of erythrocytes, forms fast under blood slow flow, more often in veins.
- *Mixed thrombus*, in which leucocytes are alternated with erythrocytes and fibrin layer-by-layer occur in heart cavities, aneurisms, varicose veins.
- Hyaline thrombus* does not contain fibrin, consists of destroyed erythrocytes, leucocytes, blood plasma proteins, forms in microcirculation channel vessels.
- In respect to vessels lumen thrombus could be mural (parietal) and obturating.

■Thrombi can form in arteries, veins, cardiac cavities, in heart's and vessels'в aneurisms. The most important practical meaning has thrombus appearance in cardiac cavities and venous network. The causes of thrombus formation in veins are progressive cardiac insufficiency, immovability after complicated surgeries, severe oncology pathology, serious infections, veins inflammation (phlebitis), veins catheterization. Thrombus formation in cardiac cavities occurs more often in atriums, in atrial auricle portion, in chronic aneurisms, on cardiac valves. The cause is: cardiac insufficiency and cavities dilation, myocardial infarction with endocrdium damage, valves injury under endocarditis. Thrombi formation in arteries are observed under atherosclerosis plaques' ulceration, arterial aneurisms, vasculitis. Thrombi growth goes on by thrombosis masses stratification in the direction of blood flow or against blood flow direction.

### **Form of thrombi depending (on) pathogenesis**

■Thrombi which grows fast is called *progressive*. There is also a concept of "*migrating thrombosis*", when many thrombi in various places of human body form in case blood ability to coagulate is increased. Thrombi in aneurism are called *dilatative*. Thrombi formed under blood flow general slowing down, under cardiac insufficiency are called *marantic* or *congestive*. Thrombi formed in the place of the vessels branching are called *thrombus-riders*.

### ***Thrombosis consequences***

- favorable
- unfavorable.
- favorable*
- Aseptic autolysis
- organization .
- Thrombi dissolve owing to blood anticoagulative system activation and leucocytes' proteolytic ferments which are destroyed in the thrombi. Thrombi disappear without a trace. Big thrombi are rare to dissolve, more often they grow with conjunctive tissue, that is called organization. Conjunctive tissue growing in starts from the head. Cracks (channels) form in it in which blood circulation can recommence - recanalization of vessels. Surface of such channels is paved with endothelium. Later on they convert into vessels containing blood – "thrombus vascularization". Besides that vessels can grow in from intima side. Sometimes thrombi could carbonize (phlebolits).

Organized thrombus. Vascular lumen is filled with thrombus masses on the stage of organization.

recanalization of vessel

### ***Unfavorable consequence***

■is septic autolysis under pyogenic infection influence. In such cases thrombi disintegration into parts is observed, these parts are carried with the blood in various organs and tissues causing inflammation generalization and sepsis development.

### **Thrombosis significance**

■The defensive one is determined by hemorrhage stop from damages vessel.

- Unfavorable – development of necrosis, thromboembolism, thrombophlebitis.

## Embolism

—***Embolism*** is circulation in blood or lymph particles which are not met there as normal.

### ***Classification of embolism***

- *orthograde* - Emboli mostly moves in blood or lymph flow direction),
- *retrograde* - (against the flow), for example in case veins (lymphatic vessels) valves insufficiency under their lumen dilation (venous stagnation, lymphostasis).
- *paradoxical* embolism is possible when under defects presence in interatrial septum or interventricular septum, embolus, passing lungs, comes from left half of heart to the right one.
- *kinds of embolism on emboli nature*
- thromboembolism, fat, air, gas, tissue (cellular), microbial embolism, embolism by foreign objects.

### ***Thromboembolism***

- – is the most often kind of embolism.
- The most often thrombi of greater circulation veins' become emboli or those formed on valves under endocarditis.
- From greater circulation veins they come into small branches of pulmonary artery. and hemorrhagic infarction of lungs occurs.
- Under thromboembolism of pulmonary artery large branches sudden death comes caused by **pulmocardiac shock** development.

### ***pulmocardiac shock***

■ The essence of shock lays in the fact that as the result of pulmonary artery intima irritation by embolus, which is rich with nerve receptors, especially in the place of its branching, sudden spasm of bronchi, pulmonary artery branches and cardiac coronary vessels occurs. Thromboemboli from lungs, mitral and aortic valves comes to aorta and through its branches – into various organs, where they obstruct vessels and contribute infarctions development. Thromboemboli from intestines veins migrate in liver portal vein system. Under migrating thrombosis thromboembolism is diversified, in such cases we speak of thromboembolism syndrome.

***Pulmonary embolism.*** In the opened pulmonary artery lumen foreign object is seen, which is thromboembolus not fixed to vascular wall. It is of red color with smooth surface.

Thromboembolus of pulmonary artery

### ***Fat embolism***

- – emboli are fat drops.
- Aetiology
- - traumatic injury of subcutaneous fatty tissue,
- - tubular bones fracture,
- -massive fermentative necrosis of fatty tissue (pancreonecrosis),
- -mistaken injection in vessels oily medicines.
- Oil, as a rule, comes into veins and pulmonary artery branches. Death comes in case two third of its branches are obstructed, from acute pulmonary-cardiac insufficiency. In case less amount of vessels are obstructed, fat emulsifies, lathers and resolves with lymphatics, sometimes pneumonias' development is observed.

***Pulmonary vessels fat embolism.*** Colored with sudan III. Sudanefill substance in lungs microcirculatory channel lumen is of yealaw-red color.

### ***Air embolism***

- Aetiology
- in case neck veins injury in which negative pressure exists
- in case uterus veins are not diminished in its postnatal atony,
- -pneumothorax,
- accidental air injection in vein together with medicines.
- Massive air embolism of lesser circulation vessels causes sudden death. At that air accumulates in right heart cavities. With the aim of its preliminary diagnosis right heart is subject to stuck submerged in the water. First pericardium should be dissected and filled with water, after right ventricle of the heart sticking air bubbles are coming out. Blood in right heart cavities is foamy

### ***Gas embolism***

- Aetiology
- -fast change of high atmospheric pressure to the low one (fast depressurization, of airplane cabin, space vehicle, pneumatic work).
- Pathogenesis
- Under fast decompression nitrogen dissolved in blood could not be taken out by lungs and its bubbles occur in blood - "blood boils". Gas emboli appears in arterial blood, obstruct capillaries of all organs and tissues, especially in capillary vascular network structure. The most affected are cerebrum and spinal cord., kidneys, knee joints, eye retina. The portions of ischemia and necrosis

appear in organs with further multiple spot hemorrhages and microthrombi, which is characteristic for decompression (caisson) sickness

**Tissue (cell) embolism**

■ **Aetiology**

- tissue damage with trauma or pathologic process causing a piece of tissue (cells) coming into blood circulation.
- It's mostly apply to malignant tumors, cells of which penetrate into lumens of blood (veins) and lymph vessels causing metastatic disease,
- pieces of heart ventricles under ulcerable endocarditis, aorta walls under atherosclerotic plaques ulceration,
- cerebrum tissues under head trauma, as well as (neonates) under birth craniocerebral trauma.
- Embolism with amniotic water in parturient women is also refers here.

Liver metastasis. On section white color tumor nodes of various sizes are seen in liver parenchyma.

Lymphoma's metastasis in kidney

**Microbial embolism**

■ occurs when pathogens' colonies obstruct vessels lumens (capillaries). It could be fungus, protozoa, zooparasites. Quite often microbial emboli forms under thrombi' suppurative melting. In obstruction place metastatic pyogenic abscesses form.

**Embolism with foreign objects**

■ occurs in case fragments of bullets, mines and other objects come into vessels lumen. Heavy foreign objects move close, sometimes against blood flow – rethrograde embolism. Here relates also embolism with a pieces of petrificates, atherosclerotic plaques' cholesterol crystals.

**Significance of embolisms**

- - infarctions development,
- - metastatic diseases of tumors,
- - pyogenic abscesses metastatic diseases with sepsis,
- - thromboembolism syndrome development,
- - sudden death of pulmocoronary shock

**Infarction**

■ **Infarction is a necrosis, caused by blood supply stop, in other words, ischemia.**

■ It is vascular and ischemic kind of necrosis. Infarctions occurs of wedging shape in organs with mainline type of arteries branching (spleen, lungs, kidneys) and of irregular shape in organs with scatter type of arteries branching (cerebrum, heart).

■ **Classification**

- White infarction (spleen),
- white infarction with red shell (myocardium, kidneys)
- red infarction (lungs, bowel)

**White infarction**

■ is well separated from surrounding tissue necrosis portion of white-yellowish color. Occurs mostly in organs with collaterals insufficient development (spleen).

**White infarction with hemorrhagic dressing**

■ is a portion of white-yellowish color necrosis separated from surrounding tissue with dilated collateral vessels and diapedetic hemorrhages. The shell is the result of spasm conversion into paretic dilation of vessels and increase of vessels permeability.

**Hemorrhagic infarction**

■ is a portion of necrosis soaked with blood. Its development is caused by organ's angioarchitecture – dual type blood supply with anastomosis presence. For example, lungs obtain venous blood through pulmonary artery system and arterial – from bronchial artery system. In conditions of pulmonary artery branch lumen obstruction which is often facilitated with thrombi formation on venous stagnation basis, blood through anastomosis comes to necrosis portion from bronchial artery, burst capillaries and accumulates in alveoli.

**Hemorrhagic infarction of lung**

Morphology of infarctions

|            |                                 |                  |                        |
|------------|---------------------------------|------------------|------------------------|
| ■ Organ    | Type of infarction              | Type of necrosis |                        |
| ■ Heart    | White with hemorrhagic dressing | Coagulation with | secondary colliquation |
| ■ Lungs    | Red                             | Coagulation      |                        |
| ■ Kidneys  | White with hemorrhagic dressing | Coagulation      |                        |
| ■ Cerebrum | White and red                   | Colliquation     |                        |
| ■ Spleen   | White                           | Coagulation      |                        |
| ■ bowel    | Red                             | Colliquation     |                        |

**morphogenesis of infarction**

- There are three stages
- pre-necrotic (ischemic),
- necrotic
- post-necrotic (infarction healing, cicatrization).
- Pre-necrotic stage is characterized with growing degenerative changes. Tissue structure yet conserved. Glycogen disappears in ischemic portion, breath ferments activity decreases, intracellular organelles swell and destroy.
- Necrosis stage is clearly manifested in 18-24 hours from the beginning of the process development. It is characterized with tissue decay (nucleus disappear, cytoplasm dissolve) and its melting (autolysis).

#### **Consequence of infarction**

- -conjunctive tissue cicatrix is formed.
- -petrification
- -cyst formation (cerebrum) also relate to favourable consequences.
- -Dangerous one is suppurative melting which is often found under bacterial embolism.

#### **Shock**

■ *Shock is generalized acute failure of hemodynamics caused by super strong irritation of organism with cardiac-vascular system neurohumoral regulation disorder manifested with acute decrease of blood supply into tissues, their hypoxia and vitally important functions of organism depression.*

#### **Shock pathogenesis**

In shock development pathogenesis erectile and torpid phases are differentiated.

- In the first phase generalized excitation of nervous system is observed, metabolism intensification, sympathoadrenal system activation, catecholamines' amount increase in blood, endocrine glands function increase, generalized spasm of the vessels, arterial-venous anastomosis opening, blood re-distribution in venous channel past capillaries, venous pressure increase, failure of blood outflow from capillaries, blood depositing in internals, hypovolemia, blood portion exclusion from general circulation, blood minute volume decrease, circulation speed decrease, hypodynamia development, energy metabolism change on anaerobic way.
- In the second phase considerable slowing-down of central nervous system functions is observed as well as cardiovascular system function failure, respiratory compromise and hypoxia development.

#### **Etiopathogenetic classification of shock**

from exogenous factors action: traumatic, burn, from electric trauma;

- - from endogenous factors action under internals diseases: abdominal, cardiogenic, nephrogenic;
- - caused by humoral failures: anaphylactic, hemotransfusion, hemolytic, endocrine, toxic (bacterial, infection-toxic).
- By endopathogenous principle shock is divided into septic, cardiogenic, anaphylactic, hypovolemic, neurogenic.

#### **Shock morphology**

- fluid condition of blood in vessels,
  - disseminated intravascular blood coagulation,
  - hemorrhagic syndrome,
  - blood depositing in microcirculatory channel,
  - blood circulation bridging,
  - glycogen mobilization in tissues' depots,
  - degenerative changes in parenchymatous organs.
- **Fluid condition** of blood occurs under instantaneous death and is caused by postmortem fibrinolysis as the result of consumption coagulopathy under **DIC-syndrome** which the most often occurs under bacterial shock.
  - **Blood depositing** macroscopically is manifested with the features of hypovolemia: there is no blood in the heart, small amount of blood is in big venous vessels.
  - **Blood circulation bridging** is manifested with kidneys cortex ischemia, juxtglomerular zone and renal pyramids plethora, interstitial edema of lungs.
  - Fast **glycogen mobilization** from depot is manifested with light (shock) hepatocytes presence: first glycogen disappears, then fatty (adipose) degeneration develops.
  - **Hemodynamic changes** at shock are as follows: venous hyperemia, sludge-syndrome, stasis, thrombosis, diapedetic hemorrhages, pulmonary edema.
  - Certain morphologic features of changes in internals depending on shock type were found.

## **Disseminated intravascular clotting (DIC) syndrome**

■ **(Disseminated intravascular blood coagulation syndrome)** or *DIC-syndromw* or *thrombohemorrhagic syndrome* or *consumption coagulopathy* is grave terminal condition characterized with thrombi (fibrin, erythrocyte, hyaline) widespread formation in microcirculatory channel with simultaneous non-coagulation of blood causing multiple hemorrhages.

### **Morphology of DIC**

- -The most often thrombi are observed in microvessels of lungs, kidneys, liver, adrenal glands, hypophysis, cerebrum, etc.
- -Simultaneously multiple hemorrhages develop in these organs,
- -degenerative and necrotic changes,
- - thrombocytopenia in blood causing pathologic bleeding disease.
- Owing to such changes multisystem insufficiency develops and patients' death

### **Aetiology of DIC**

- endotoxic shock caused by massive injury of endothelium with bacteria, virus, rickettsia, immune complexes or cytokines,
- premature detachment of placenta
- embolism with amniotic fluid
- intrauterine death of fetus;
- snake bites,
- promyelocytic leukemia,
- etc.
- Syndrome is grounded on blood coagulative and anticoagulative systems function failure.
- 

thanks