Ministry of Health of the Republic of Belarus
Educational Institution
“Gomel State Medical University”

Department of Pathological Anatomy

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METHODICAL GUIDELINES

for conducting a practical class
in the academic discipline “Pathological Anatomy”
for students
of the 3rd year, Faculty of International Students (instruction in English),
enrolled in the specialty
7-07-0911-01 “General Medicine”

Topic: “Circulatory disorders: hyperemia, stasis, hemorrhage, edema”

Duration: 3 hours

Approved at the meeting of the Department of Pathological Anatomy
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EDUCATIONAL AND INSTRUCTIONAL GOALS, OBJECTIVES, AND MOTIVATION FOR MASTERING THE TOPIC

Educational Goal:

- To identify various types of general and local circulatory disorders based on macroscopic and microscopic features, explain their causes and mechanisms of development, evaluate their likely outcomes, and determine the significance of these processes for the organism.
- To emphasize the importance of microcirculation in maintaining tissue homeostasis. When studying individual types of circulatory disorders, attention should be paid to the causes, morphological manifestations, and outcomes of these processes. In the analysis of anemias (local ischemia), the main focus is on local oligemia (angiospastic, obturation, compression), indicating the importance of collateral circulation for the condition of the organ. Among the causes of venous congestion (hyperemia), the significance of cardiac decompensation as a cause of general circulatory disturbance should be particularly emphasized. To analyze the changes developing in organs during local and general venous stasis (congestion, stasis, edema, diapedesis of red blood cells, hemosiderin pigmentation, parenchymal atrophy, increased collagen-forming function of fibroblasts – leading to organ sclerosis).
- To pay attention to the particular appearance of certain organs in venous hyperemia (brown induration of the lung, nutmeg liver). To define the difference between the concepts of hemorrhage and hematoma, and to examine the mechanisms of bleeding. When defining stasis, to emphasize the role of angioneurotic factors; to indicate that the circulatory disturbance at this level occurs in the microcirculatory bed.

Instructional Goal:

- Within the framework of the educational process for this academic discipline, the student must acquire not only theoretical knowledge, practical skills and abilities in the specialty but also develop their personal potential. They should cultivate qualities of responsibility and patriotism, be prepared to actively participate in the economic, socio-cultural, and public life of the country, recognize the social significance of their future professional activity, understand the norms of medical ethics and deontology, and learn to observe academic and labor discipline. In the course of studying the educational material, students should realize the importance of maintaining a healthy lifestyle and, as an example in the future, when performing professional duties, set an example for those around them and their patients.

Objectives: Upon completion of the practical session, the student must:

***Know*:**

- The causes, mechanisms, and morphological features of typical general pathological processes;
- The etiology, pathogenesis, and morphology of diseases at different stages of their development (morphogenesis), the structural basis of recovery,

complications, outcomes, and long-term consequences of diseases, as well as the causes and mechanisms of dying (thanatogenesis).

Be able to:

- Define arterial and venous hyperemia (congestion), name their types, and mechanisms of development;
- Diagnose venous congestion of various organs based on their macroscopic and microscopic appearance;
- Explain the significance and outcome of venous congestion in various organs;
- Define stasis, explain its causes, mechanisms of development, and functional significance;
- Define hemorrhage (bleeding), name its causes, mechanisms of development, and functional significance;
- Define hematoma (blood extravasation), name its types, and significance for the organism;
- Define oligemia (local ischemia), name its types, describe its morphology, and name its outcomes;
- Explain the mechanisms of tissue fluid imbalance, including the etiology, types, and classification of edema;
- Identify major general pathological processes and diseases using histological specimens under light microscopy;
- Diagnose pathological processes and diseases based on the description of macroscopic and microscopic changes in organs and tissues of the body.

***Master*:**

- Basic techniques for working with a microscope;
- Skills in clinical-anatomical analysis;
- The fundamentals of synthetic generalization of morphological diagnostic signs of diseases and their correct interpretation in cause-and-effect relationships.

Motivation for Mastering the Topic:

The cardiovascular system enables the functioning of various organs under continuously changing conditions of the internal and external environment of the body. In a healthy organism, normal circulation is ensured by the activity of the heart, blood vessels, and the neurohumoral system, which maintain homeostasis – normal levels of arterial, venous, and capillary pressure. Therefore, normal circulation depends on the state of the heart, blood vessels, and neurohumoral mechanisms.

In pathology (e.g., heart disease, vascular disease, blood disorders, infectious diseases, intoxications, oxygen deprivation), the heart muscle, vessel walls, and the nerve cells that regulate the activity of the cardiovascular system are damaged. The heart loses its ability to adapt to changes in the external environment or responds with inadequate reactions, leading to circulatory disturbances, which are conventionally divided into general and local. The boundary between them is difficult to draw, as general and local circulatory disorders are closely interrelated.

Therefore, knowledge of general and local circulatory disorders is of great importance in the training of a physician, as it substantiates the material essence of pathological processes and thereby the rationale for pathogenetic therapy of various diseases.

QUESTIONS

1. Arterial hyperemia. Definition. Classification. Etiology. Clinical examples. Outcomes.
2. Venous congestion. Definition. Classification. Etiology. Clinical examples. Outcomes.
3. Ischemia. Definition. Classification. Etiology. Clinical examples. Outcomes.
4. Bleeding and hemorrhage. Definition. Classification. Etiology. Clinical examples. Outcomes.
5. Edema and dehydration. Definition. Classification. Etiology. Clinical examples. Outcomes.

COURSE OF THE SESSION

Arterial hyperemia occurs when there is an increased volume of blood entering the arterial system without a corresponding change in blood outflow.

- **General**: Involves the whole body.
- **Local**: Limited to a specific area.
- **Physiological**: Normal response to stimuli (e.g., exercise).
- **Pathological**: Abnormal and associated with disease.

General arterial hyperemia

General arterial hyperemia is an increase in red blood cells (RBCs) and, in some cases, a rise in circulating blood volume.

- Occurs at high altitudes (climbers, mountain dwellers).
- Seen in people with lung diseases.
- In newborns after the umbilical cord is tied off.

Clinical Features:

- Reddening of the skin and mucous membranes.
- Increased blood pressure.
- **Erythrocythemia** (**True Polycythemia**): Overproduction of RBCs leads to general arterial hyperemia, making it clinically significant.

Local arterial hyperemia

1) **Physiological**: Normal response. Example: a "blush of embarrassment" or redness at the site of heat or mechanical irritation.

2) **Pathological**: Abnormal and caused by various factors.

- *Angioneurotic Hyperemia*: Caused by nervous system dysfunction affecting blood vessels.
- *Collateral Hyperemia*: Develops when blood is rerouted due to blockage in nearby vessels.
- *Postischemic Hyperemia*: Occurs after blood flow is restored following a period of ischemia (restricted blood supply).
- *Vacuum Hyperemia*: Due to reduced external pressure, leading to increased blood flow.
- *Inflammatory Hyperemia*: Associated with inflammation, where blood flow increases to an affected area.
- *Hyperemia Due to Arteriovenous Fistula*: Results from abnormal connections between arteries and veins.

Angioneurotic hyperemia

It is a form of hyperemia caused by vasomotor disorders, where either the vasodilating nerves are irritated or the vasoconstrictor nerves are paralyzed.

- Blood flow accelerates in both normal and reserve capillaries.
- Skin and mucous membranes appear red, slightly swollen, and feel warm or hot.

- Typically resolves quickly without lasting effects.

Causes:

- Irritation of vasodilating nerves or paralysis of vasoconstrictor nerves.
- Irritation of sympathetic ganglia.

Clinical Examples:

- *Systemic Lupus Erythematosus*: Characterized by areas of facial hyperemia in a butterfly pattern.
- *Hyperemia in Extremities*: Due to damage to nerve plexuses.
- *Facial Hyperemia*: Trigeminal nerve irritation (neuralgia).

Collateral Hyperemia

Occurs when a **main artery is blocked** (e.g., by an atherosclerotic plaque), causing blood to divert through collateral vessels, which expand to compensate.

Mechanism:

- Blood flow is rerouted via collateral vessels around the blockage.
- These vessels dilate to allow sufficient blood supply.

Clinical Importance:

- Understanding collateral circulation is crucial for surgeons.
- Allows safe ligation (surgical tying off) of major arteries like the femoral, popliteal, or carotid without causing tissue necrosis.

Postischemic hyperemia

Occurs when **blood flow is rapidly restored after a period of local ischemia** (restricted blood supply), such as after removal of a tumor or fluid buildup.

Mechanism:

- Previously ischemic tissues experience sudden dilation of microcirculatory vessels, which fill with blood.

Clinical Risk:

- Overfilled vessels, especially in the elderly, may rupture, leading to bleeding or hemorrhage.

Local arterial hyperemia

Vacuum hyperemia is caused by a decrease in barometric pressure.

It is commonly observed in traditional Chinese medicine practices.

Inflammatory hyperemia is an important clinical sign of inflammation, where increased blood flow to the affected area is a key part of the inflammatory response.

Hyperemia due to arteriovenous fistula occurs when an abnormal connection (anastomosis) forms between an artery and a vein, allowing arterial blood to flow directly into the vein. The primary danger is the potential rupture of the fistula, leading to bleeding.

General venous congestion

Pathophysiology:

- In left ventricular failure, the heart's ability to pump blood effectively is reduced, leading to impaired forward flow. This causes blood to back up in the venous system.
- Blood pools in large veins (such as the vena cava and its branches), while the arterial circulation experiences decreased blood volume and pressure.
- As venous pressure increases, fluid can leak into tissues, causing edema, particularly in dependent areas like the legs and feet.

Causes:

- Heart failure: Most common cause, especially left ventricular failure.
- Chronic lung diseases: Can contribute to right-sided heart failure (cor pulmonale), worsening venous congestion.
- Venous obstruction: Conditions such as deep vein thrombosis (DVT) can cause local venous congestion but may contribute to generalized symptoms if severe.

General venous congestion

Clinical Manifestations:

- Edema: Common in the lower extremities (peripheral edema) due to increased venous pressure.
- Cyanosis: Reduced oxygen delivery may result in a bluish tint to the skin, especially in extremities.
- Jugular Venous Distension: Often seen in severe cases due to increased pressure in the superior vena cava.
- Hepatomegaly: Blood congestion in the liver, causing enlargement and possible pain.
- Ascites: Accumulation of fluid in the abdominal cavity, a result of prolonged venous congestion.

Complications:

- Organ damage: Prolonged venous congestion can lead to liver dysfunction (congestive hepatopathy), kidney impairment, and gut edema, impairing nutrient absorption.
- Thrombosis: Stasis of blood in the veins increases the risk of clot formation.

Acute general venous congestion

Causes:

- Myocardial Infarction: Heart muscle damage resulting from lack of blood flow.
- Acute Myocarditis: Inflammation of the heart muscle.
- Acute Exudative Pleuritis: Inflammation of the pleura leading to fluid accumulation.
- Pulmonary Embolism: Blockage of a pulmonary artery by a blood clot.

Pathophysiological Effects:

- Plasmorrhagia: Leakage of plasma from the capillaries into surrounding tissues.
- Edema: Fluid accumulation in tissues, leading to swelling.

- Stasis: Slowed or stagnant blood flow in the venous system.
- Diapedesis: Passage of blood cells, especially leukocytes, through capillary walls.

Acute general venous congestion

Morphological Changes:

- Lungs: The most affected organ due to left ventricular failure.
- Alveolar Capillary Expansion: Increased pressure leads to dilation of capillaries in the lungs.
- Pulmonary Edema: Fluid extravasation into the alveoli, causing impaired gas exchange and respiratory distress.
- Alveolar Hemorrhages: Possible bleeding into the alveoli due to rupture of capillaries.

Clinical Consequences:

- Respiratory Symptoms: Patients may experience shortness of breath, cough, and wheezing due to pulmonary edema.
- Hypoxemia: Reduced oxygen levels in the blood due to impaired gas exchange.
- Potential for Acute Respiratory Distress Syndrome (ARDS): Severe pulmonary edema can lead to ARDS, requiring intensive medical management.

Chronic general venous congestion

Causes:

- Congenital Heart Defects: Structural heart issues present from birth.
- Chronic Ischemic Heart Disease: Long-term reduction in blood supply to the heart muscle.
- Chronic Myocarditis: Persistent inflammation of the heart muscle.
- Cardiomyopathy: Diseases of the heart muscle affecting its size, shape, and function.

Organ and Tissue Changes:

- Enlargement: Affected organs and tissues may become larger (organomegaly) due to fluid accumulation.
- Cyanosis and Density: Tissues may appear cyanotic and become denser as a result of chronic venous congestion.

Chronic general venous congestion

Clinical Manifestations:

- Cyanosis: Distinctive bluish discoloration of the skin, especially noticeable in the extremities.
- Cold and Sweaty Skin: Particularly in the lower extremities due to poor blood circulation.
- Fluid Accumulation in Cavities:
  - **Ascites**: Fluid buildup in the abdominal cavity.
  - **Hydrothorax**: Fluid accumulation in the pleural cavity.
  - **Hydropericardium**: Fluid accumulation in the pericardial sac surrounding the heart.

Anasarca: A severe form of generalized edema characterized by widespread swelling in subcutaneous tissues and fluid accumulation in body cavities.

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Congestive hepatopathy

The cut surface of liver has specific appearance ("nutmeg liver") due to anatomical peculiarity on the venous system of the liver. The **liver has two venous systems**. One adductor is a **portal vein** that collects blood from unpaired abdominal organs. Parallel to this, arterial blood enters the liver through the **hepatic artery**. Branching out, **both the artery and the vein**, together with the bile duct, form the well-known "**triads**" between the lobules of the liver. Inside the lobule, the branches of the vein and artery merge, forming a single small vessel –**intralobular sinusoids**. The terminal sections of these sinusoids form the central veins in the center of the lobule – this is the beginning of the efferent venous system of the liver. **Blood from the central veins** is sent to the collecting hepatic veins, and then **to the inferior vena cava**. With general venous congestion, the developing venous congestion in the inferior vena cava spreads, respectively, **first to the hepatic veins**, then to the collecting and **central veins**, and partially to the sinusoids of the hepatic lobule.

Further expansion is not observed, since in the capillary branches of the hepatic artery flowing into the sinusoids, the pressure is always higher than in the sinusoids. The **full-blooded central sections of the lobules** are visible not only microscopically, but also macroscopically. The central sections of the lobule on the section of the liver **look dark red**. On the periphery of the lobules, the liver cells are in a state of dystrophy, often fatty, which explains the gray-yellow color of the liver tissue. Prolonged oxygen starvation in venous hyperemia leads to coarsening and growth of connective tissue in the organ and the formation of progressive **congestive fibrosis and cirrhosis of the liver**. This nutmeg cirrhosis is also called cardiac, since it usually occurs in chronic heart failure.

General venous congestion

General venous congestion can be a reversible condition if the underlying cause is addressed promptly. For instance, managing heart failure or resolving obstructions can restore normal blood flow and alleviate symptoms.

Chronic general venous congestion can lead to significant tissue hypoxia and various pathological changes, which may become irreversible:

- **Plasmorrhagia**: Leakage of plasma from capillaries into surrounding tissues.
- **Edema**: Fluid accumulation in tissues.
- **Stasis**: Slowed blood flow, leading to stagnant blood in veins.
- **Hemorrhages**: Bleeding due to vessel rupture or leakage.
- **Dystrophy**: Alterations in tissue nutrition and metabolism.
- **Necrosis**: Tissue death due to prolonged lack of blood supply.
- **Atrophic Changes**: Reduction in organ size and function due to chronic hypoxia.
- **Sclerotic Changes**: Increased collagen synthesis by fibroblasts, leading to tissue fibrosis and scarring.

Local venous congestion

Local venous congestion occurs when venous blood outflow from a specific organ or body part is obstructed. This obstruction can be due to:

- **Thrombus**: A blood clot blocking a vein.
- **Embolus**: A clot or debris traveling through the bloodstream and lodging in a vein.
- **External Compression**: Pressure from tumors or growing tissues that constrict the vein.

Pathological Changes: Similar to general venous congestion, local venous congestion can lead to:

- **Edema**: Localized swelling in the affected area.
- **Stasis** and **Hypoxia**: Reduced blood flow leading to tissue hypoxia and subsequent damage.
- **Hemorrhages**: Possible bleeding due to vessel rupture.
- **Dystrophic** and **Sclerotic Changes**: Chronic local hypoxia can stimulate fibroblast activity, resulting in fibrosis and structural changes in the affected organ.

Ischemia

Ischemia refers to any reduction in blood flow that leads to decreased oxygen and nutrient supply to tissues. This condition can be classified as:

1) **Reversible Ischemia**: Tissue can recover if blood flow is restored promptly.

2) **Irreversible Ischemia**: Prolonged lack of blood flow leads to tissue death (necrosis).

Types of Ischemia

1) **Angiospastic (Reflex) Ischemia**: Caused by spasms of blood vessels that temporarily reduce blood flow.

2) **Obstructive Ischemia**: Resulting from physical blockages in blood vessels (e.g., thrombus, embolus).

3) **Compressive Ischemia**: Occurs when external pressure compresses blood vessels, impeding blood flow.

4) **Redistributive Ischemia**: Due to the diversion of blood flow from one area to another, affecting the supply to certain tissues.

Angiospastic ischemia

Angiospastic ischemia results from the spasm of arteries, leading to temporary reductions in blood flow.

1) **Injury**: Angiospasms can occur following any type of injury (e.g., household accidents, surgical procedures, gunshot wounds), particularly when accompanied by pain or fear.

2) **Drug Administration**: Certain medications, such as adrenaline (epinephrine), can induce arterial spasms.

3) **Allergic Reactions**: Angiospastic processes may be triggered by allergic responses, causing vasoconstriction and reduced blood flow.

Obstructive ischemia

Obstructive ischemia occurs when the lumen of the arteries is blocked, leading to a significant reduction or cessation of blood flow.

1) **Thrombosis**: Formation of a blood clot within a blood vessel, obstructing normal blood flow.

2) **Embolism**: A traveling clot or debris that lodges in a blood vessel, causing an obstruction.

3) **Inflammation**: Conditions such as obliterating endarteritis can cause the growth of connective tissue within the artery lumen, leading to obstruction.

4) **Atherosclerosis**: The buildup of fatty plaques within the arterial walls can narrow the lumen and restrict blood flow.

Compressive ischemia

Compressive ischemia occurs when an artery is compressed, leading to restricted blood flow and potential tissue damage.

1) **Tourniquet Application**: The use of a tourniquet during medical procedures can compress arteries, temporarily halting blood flow to distal tissues.

2) **Surgical Ligature**: Arteries may be intentionally ligated (tied off) during surgical procedures to control bleeding, leading to ischemia in the areas supplied by the affected artery.

3) **Inflammatory Effusion**: Accumulation of exudate from inflammation can exert pressure on nearby arteries, restricting blood flow.

4) **Tumors**: Growths can compress surrounding arteries, impeding circulation to tissues.

5) **Scarring**: Scar tissue from previous injuries or surgeries may also compress arteries.

6) **Enlarged Organs**: Conditions that cause organ enlargement (e.g., hepatomegaly, splenomegaly) can lead to compression of adjacent blood vessels.

Ischemia due to redistribution of blood

Ischemia caused by the redistribution of blood occurs when a significant volume of blood shifts from one area of the body to another, leading to reduced blood flow in certain tissues.

Example: Cerebral ischemia can occur after the rapid removal of ascitic fluid from the abdominal cavity. The sudden decrease in abdominal pressure allows a large volume of blood to rush towards the upper body, potentially reducing blood flow to the brain and resulting in cerebral ischemia.

Morphological Changes in Ischemia

- Oxygen Starvation: All types of ischemia lead to morphological changes in organs and tissues primarily due to hypoxia (reduced oxygen) or anoxia (complete lack of oxygen).
- Tissue Damage: Prolonged ischemia can cause cellular injury, necrosis, and various pathological changes depending on the affected tissues.

Ischemia

Ischemia can be classified based on its causes, onset, duration, and the degree of reduction in arterial blood flow:

1) **Acute ischemia** is characterized by a complete and sudden cessation of arterial blood flow to an organ or tissue.

It is considered a pre-necrotic condition, meaning that if blood flow is not restored quickly, it can lead to irreversible damage or necrosis.

Often caused by conditions such as thrombosis, embolism, or acute trauma.

2) **Chronic ischemia** refers to a long-term, gradual decrease in arterial blood flow, which leads to:

Atrophy of Parenchymal Cells: Decreased size and function of the functional cells of the organ.

Stromal Sclerosis: Increased collagen synthesis by fibroblasts, resulting in the hardening and scarring of the connective tissue, e.g. the development of cardiosclerosis in chronic coronary heart disease is a classic example of chronic ischemia leading to structural changes in the heart.

Factors influencing the outcome of ischemia

1) **Degree of Development of Collaterals**: Well-developed collateral circulation can help compensate for reduced blood flow, mitigating damage.

2) **Condition of Collateral Arteries**: Healthy and functional collateral vessels enhance blood supply to ischemic areas.

3) **Efficiency of the Cardiovascular System**: A well-functioning heart and vascular system can better maintain blood flow and oxygen delivery.

4) **Rate of Occurrence of the Obstacle**: Sudden obstructions can lead to more severe ischemia compared to gradual blockages, which may allow time for collateral circulation to develop.

5) **Susceptibility of Tissue to Ischemia**: Different tissues have varying tolerance to ischemia; for example, cardiac and neural tissues are more sensitive and susceptible to damage from reduced blood flow.

The outcome of ischemia

The effects and importance of ischemia vary based on its severity and duration, as well as the following factors:

1) **Development of Collaterals**: Tissues with a robust network of collateral arteries maintain blood flow even if one artery is blocked. For instance, if the radial artery is occluded, ischemia in the arm may not occur due to compensatory blood flow through the ulnar artery.

2) **Status of Collateral Arteries**: If the collateral arteries are narrowed, their ability to compensate diminishes. For example, in younger individuals, a blockage in the internal carotid artery may be offset by increased flow in the collateral vessels of the circle of Willis. In contrast, older adults with atherosclerotic narrowing of these arteries often experience cerebral ischemia when one internal carotid artery is occluded.

3) **Cardiovascular System Efficiency**: Adequate functioning of the heart and normal blood pressure are necessary for effective collateral circulation, as blood must navigate through relatively narrow collateral vessels.

4) **Speed of Obstruction**: Rapid arterial occlusion leads to more severe ischemic effects compared to gradual occlusion, as there is less opportunity for collateral vessels to develop. For example, a sudden blockage of a healthy coronary artery can result in a myocardial infarction, while a gradual blockage allows for the development of collateral vessels, reducing ischemic damage.

5) **Tissue Sensitivity to Ischemia**: Different tissues have varying levels of tolerance to ischemia. The brain is highly vulnerable, often experiencing infarction within minutes of arterial blockage, while skeletal muscles, bones, and some other tissues can endure ischemia for several hours before damage occurs.

Bleeding and hemorrhage

Bleeding is the release of blood from a blood vessel or the heart. It can be categorized as external bleeding, where blood exits the body, or internal bleeding, where blood accumulates within body cavities.

Hemorrhage refers specifically to blood accumulating in tissues.

1) **Hematoma**: This is an accumulation of coagulated blood within tissues. It occurs when blood vessels are damaged, leading to a collection of blood that may cause necrosis in the center of the hematoma.

2) **Hemorrhagic Infiltration**: In this type, blood seeps into tissues while maintaining the structural integrity of tissue elements. It often results in localized swelling and discoloration without significant tissue death.

3) **Bruises** (Contusions): These are planar hemorrhages that occur in the subcutaneous tissue and muscles. Bruises are typically characterized by discoloration of the skin due to blood pooling beneath the surface.

4) **Petechiae**: These are small, pinpoint hemorrhages that appear on the skin and mucous membranes. They often indicate underlying conditions like thrombocytopenia or vascular abnormalities.

Causes of bleeding

1) **Rupture of the Vessel Wall**: This occurs when the wall of a blood vessel is injured or undergoes pathological changes, such as inflammation, necrosis, or aneurysm formation.

2) **Corrosion of the Vessel Wall**: The vessel wall can be damaged by various factors, including inflammation, necrosis, or the presence of malignant tumors, leading to compromised structural integrity.

3) **Increased Vascular Permeability**: Conditions that cause blood vessels to become more permeable can lead to red blood cell (RBC) diapedesis, where RBCs escape from the circulation into surrounding tissues.

Outcome of hemorrhage

1) **Formation of a Cyst**: Accumulated blood may become encapsulated, forming a cystic structure.

2) **Encapsulation**: The body may form a capsule around the hemorrhage, isolating it from the surrounding tissues.

3) **Organization**: The blood clot may undergo organization, leading to the development of granulation tissue and eventual healing.

4) **Petrification**: In some cases, the area of hemorrhage may calcify, leading to petrification.

5) **Aseptic Autolysis**: The tissues surrounding the hemorrhage may undergo aseptic autolysis, where they break down without infection.

6) **Suppuration with Secondary Infection**: If the hemorrhage becomes infected, it may lead to suppuration, characterized by pus formation and inflammation.

Edema

Edema is the **excess accumulation of tissue fluid**, specifically a transparent fluid known as transudate, which contains no more than 2% protein. While edema can occur in any tissue, it is most easily observed in the subcutaneous tissue.

Types of Edema

1) **Allergic Edema**:

- **Cause**: This type arises from acute allergic reactions that trigger the release of vasoactive substances like histamine.
- **Mechanism**: These substances cause dilation of blood vessels and increased permeability of capillaries.
- **Manifestation**: Allergic edema is most commonly seen in the skin, where it can present as blisters or hives (urticaria).

2) **Due to Venous Congestion**:

- **Cause**: This occurs when there is an obstruction or impairment of venous outflow, such as in patients with varicose veins (phlebeurysm).
- **Location**: It is most commonly observed in the lower extremities, leading to swelling and discomfort.

3) **Due to Lymphatic Stasis**:

- **Cause**: When lymphatic drainage is compromised, protein and fluid that normally exit through capillaries accumulate in the interstitial space.

- **Example**: A similar mechanism occurs in lymphatic filariasis, often referred to as elephantiasis, where severe swelling results from the obstruction of lymphatic vessels.

4) **Hypoproteinemic Edema**:

- **Definition**: This type of edema occurs when there is a decrease in plasma protein levels, particularly albumin, leading to reduced osmotic colloidal pressure.
- **Causes**:
  - Insufficient Dietary Protein Intake: This can result in starvation edema due to inadequate nutrition.
  - Decreased Albumin Synthesis: Conditions affecting the liver, such as cirrhosis, can lead to hepatic edema.
  - Increased Protein Loss: This can occur in:
  - Nephrotic Syndrome: Increased protein loss through the urine.
  - Protein-Losing Enteropathy: Protein loss from the intestines.

5) **Cardiac Edema**:

- **Cause**: This type is associated with chronic heart failure, where the heart's ability to pump blood effectively is compromised.
- **Mechanism**: The resultant venous congestion leads to fluid accumulation, especially in the lower extremities and sometimes in the abdominal cavity (ascites).

6) **Renal Edema**:

- **Cause**: Renal edema occurs when there is a significant reduction in glomerular filtration rate (GFR).
- **Mechanism**: This reduction leads to sodium and water retention, resulting in moderate edema. It is commonly seen in conditions affecting kidney function, such as acute kidney injury or chronic kidney disease.

INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

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NORMATIVE LEGAL ACTS AND DOCUMENTS

1. Order of the Ministry of Health of the Republic of Belarus No. 111 of 06/01/1993 "On further improvement of the pathological service of the Republic of Belarus"

Access mode: http://patan.by/menyu/administrativnyie-proczeduryi.html - The date fccess: 20.03.2022.

ELECTRONIC DATABASES

1. Physician's consultant. Electronic Medical Library = Consultant of the doctor. Electronic medical library [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". – Access mode: http://www.rosmedlib.ru/ . – Access date: 03/15/2022.

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