VASCULAR DISORDERS

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HEMORRHAGE

Hemorrhage

- It is the extravasation of erythrocytes.
- **By tissue vessel type**: arterial, venous, capillary
- **By location:** internal, external, interstitial, parenchymatous
- According to its pathogenesis, it occurs either in the form of **Haemorrhagia per rhexis with the deterioration of the vascular integrity** or as **haemorrhagia per diapedesis with the deterioration of the vessel permeability.**

Haemorrhagia per rhexis

- It occurs as a result of the deterioration of the integrity of the vessels, their rupture.
- **Traumatic hemorrhages:** Caused by impacts such as hitting, hitting, cutting, or by parasites. Bleeding that occurs in the veins on the opposite side of the struck area as a result of hitting the skull with a blunt object is called Contre-coup bleeding.
- **Erosion hemorrhages:** As in human pulmonary tuberculosis, it occurs as a result of the lesion spreading towards the vessels and eroding the vessel wall. In such patients, bleeding from the mouth is a clinical finding.
- **Bleeding in the form of rupture:** It is caused by the rupture of the vessels as a result of excessive increase in intravascular blood pressure.

Haemorrhagia per diapedesis

- It is the bleeding that occurs due to the increase in permeability without deterioration of the vascular integrity.
- Shock
- Hemorrhagic inflammations formed by infectious effects (such as pasteurellosis, anthrax, blackleg)
- Hemophilia and leukosis
- Toxic-chemical effects
- Vitamin C and K deficiency
- Neurotic effects

Types of hemorrhage

- **Neurotic hemorrhages:**
  - It is formed as a vegetative reflector.
  - It usually occurs in a place other than the lesion site.
  - For example, stomach bleeding in rabies.
- **Agonal or asphyxia-related bleeding:**
  - Such as bleeding in pleura, pericardium, endocardium.

Hemorrhagic diathesis

- Developing due to vascular permeability and blood production disorder; It is bleeding that occurs as **severe haemorrhagia per diapedesis.**
  - Vascular permeability disorder (vasopathies)
  - Blood clotting disorder (coagulopathies)
  - Platelet disorder (thrombocytopathy)
- They are bleedings that can easily develop on the skin and mucous membranes, usually in the form of purpura.
- **Nonthrombocytopenic purpura:**
  - Only vascular permeability is impaired.
- **Thrombocytopenic purpura:**
  - It is associated with decreased platelet production or increased destruction.

Terminology

- **Epistaxis:** Bleeding from nose
- **Hemoptysis:** Bleeding from the lungs, bleeding from the mouth
- **Hematomesis:** Stomach bleeding, bleeding from the mouth
- **Hematuria:** Blood in the urine, bloody urine
- **Melena:** The presence of blood in the feces
- **Othematoma:** Ear bleeding

Terminology

- **Hemothorax:** Collection of blood in the thoracic cavity
- **Hemoperitoneum:** Collection of blood in the abdominal cavity
- **Hemopericardium:** Collection of blood in the heart sac
- **Hemometra:** Bleeding in the uterus
- **Hemarthrosis:** Joint bleeding
- **Hemorrhagic infarction:** Bleeding due to strangulation

Hematoma

- It is a tumor-like collection of blood filled with blood, confined to the tissue.
- Initially, it is filled with blood, then the blood coagulates and is surrounded by a capsule.
- It varies according to its location and occupies a large area in loose areas such as connective tissue.
- By putting pressure on blood-lymph vessels and nerves, it impairs the function of the organ they are in.

Hemorrhage

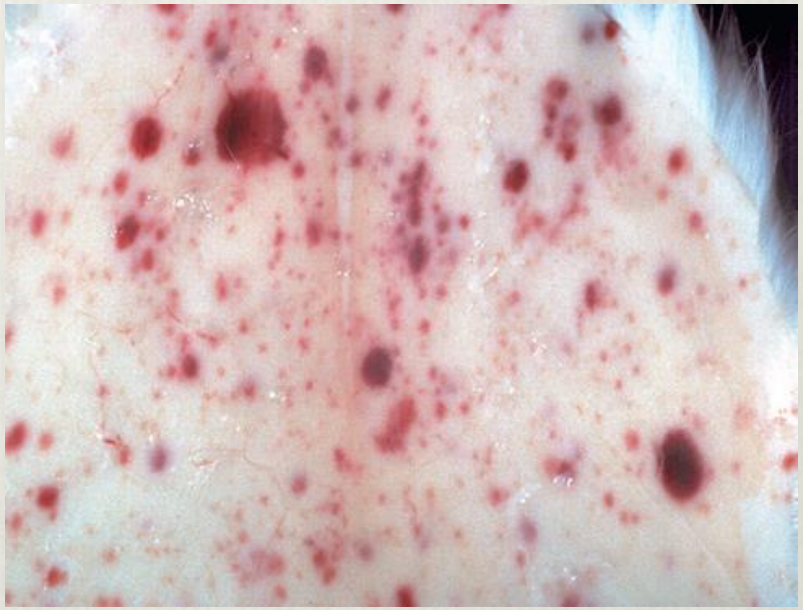
Petechie

- Spot-shaped, pinpoint-sized (1-2 mm in diameter) hemorrhages

Hemorrhage

Ecchymose

- It is a dark purplish blood blister that is widespread under the skin, mucous membranes, and serosa, with irregular borders, larger than 0.5 cm, and can reach the size of the palm of the hand.



Subcutis, rabbit

Mosier DA. Vascular Disorders and Thrombosis. Pathologic Basis of Veterinary Disease. 2017:44-72.

Hemorrhage

- **Vibex (Vibices):** a linear subcutaneous extravasation of blood
- **Purpura:** Similar to petechiae and ecchymotic hemorrhages. However, very frequent and numerous bleedings that look like they are spattered with a brush. It manifests itself as petechial, ecchymotic hemorrhages on the skin and mucous membranes. Its cause is hemorrhagic diathesis.
- **Blood aspiration:** Blood is seen in the lungs by aspirating blood from the trachea into the lungs during slaughter. It's not real hemorrhage.

THROMBOSIS

Hemostasis and Thrombosis

- H**EMOSTASIS** to normal blood coagulation to stop bleeding after vessel injury; The entry of the coagulation mechanism into the pathological process is called **THROMBOSIS.**
- hemostasis; primary-secondary
- In both, stopping the blood after vascular injury occurs through the **interaction between the three systems.**
  - Vascular wall
  - Platelets
  - Plasma coagulation proteins

Primary hemostasis

- It is achieved by **adhesion, activation and aggregation** of platelets.
- As a result of damage to the vascular endothelium, platelets (thrombocytes) adhere to the wall. **(adhesion)**
- Thrombin-Antithrombin III accelerates the **activation** of platelets.
- **Aggregation** increases with the entry of Ca into the platelets. (antithrombin III normally suppresses "thrombin", Heparin is antithrombin III cofactor)

Secondary hemostasis

- It is provided by the **activation of the plasma coagulation system and the formation of fibrin.**

Extrinsic pathway

Intrinsic pathway

Common pathway

Thrombosis

- The clotting of blood in the blood vessels and heart cavities by collapsing in the living organism is called **thrombosis,** and the clot formed as a result of this event is called **thrombus.**
- Thrombosis occurs **mostly in veins,** less in arteries due to rapid blood flow.
- The most important cause is **damage to the vascular endothelium**. However, not every injury results in thrombosis. (Ex: Arteriosclerosis)
- **Stagnation of blood** also promotes coagulation, but this alone is not enough.

Pathogenesis

Slowing of blood flow

Increased tendency of blood to clot

Changes in the vessel wall

Slowing of blood flow

a) In normal blood flow, there are leukocytes in the middle, erythrocytes around them and platelets in the outermost part.

- In the case of slowing down (stasis) blood flow, the order is disturbed and platelets collapse into the vessel wall.
- At the same time, endothelial cells cannot be fed due to blood stagnation and cannot secrete antithrombin.
- As a result, fibrinogen turns into fibrin and shaped elements precipitate to form a clot.

Slowing of blood flow

b) The blood returns to the valves (valvules) in the veins, hitting the heart valves.

- As a result of striking and returning in these regions, a vortex is formed.
- When blood flow accelerates, injury occurs due to hitting the valves.
- This, in turn, is a cause of thrombosis and also explains why thrombosis is more common in veins and cardiac cavities.

Slowing of blood flow

c) Thrombosis related to stagnation of blood flow:

- Aneurysm, varicose
- Heart failure
- Shock
- Prolonged immobility of the extremities
- Factors leading to local blood stagnation
- Sometimes in pregnancy, thrombosis develops in the extremities due to the pressure of the uterus on the V. iliaca.

Increased tendency of blood to clot

- Disorders in the structure of the blood develop in the direction of preventing or increasing coagulation. These are called **coagulopathy.**
  - Thrombosis occurs when such changes develop in the direction of increasing coagulation.
- The increase in the number of platelets and the increase in the **adhesion of platelets** to each other are the causes of coagulation.
  - For example, **inflammation, malignant neoplasms, myocardial infarction, polycythemia, loss of water and electrolytes in the blood, sickle cell anemia** cause platelet adhesion.

Increased tendency of blood to clot

- The increase in blood clotting factors (factor V, VII, VIII), especially **fibrinogen (factor I),** shapes thrombosis.
- In addition, **diabetes,** the effect of the dead fetus remaining in the uterus and not excreted, **amyloidosis in the kidneys, contraceptive drugs, long-term cortisone treatment, high cholesterol and other lipids in the blood** impair the composition of the blood.
- **Activation of the complement system, antigen-antibody complexes, and lymphokines** play an important role in the formation of thrombosis in this way.

Changes in the vessel wall

- The main cause is **damage to vascular endothelial cells.**
- Damage to the endocardium in the heart also leads to this.
- Because endothelial cells **prevent platelets from sticking, they neutralize thromboplastin by removing antithrombin.**
- Besides anticoagulant, it also has **fibrinolytic properties.**
- When damaged, this feature disappears.

Changes in the vessel wall

- Physical trauma, cold, burning, radiation, electric current, chemical-toxic substances (such as arsenic, snake venom), **parasitic (Strongylus vulgaris larvae in A.mesenterica cranialis)-infectious (virus, bacteria and toxins),** various Thrombosis also develops in **inflammatory events** (arteritis, phlebitis, endocarditis), other changes related to arteriosclerosis.

- However, endothelial cells are most affected by lack of oxygen (hypoxia, anoxia).

- Irregularity in blood flow in the vessels is shaped by effects that reduce oxygen.

Types of thrombosis

I. According to pathogenesis

1. **Coagulation thrombosis:**

- The slowing of blood flow is formed by an increased tendency to coagulate.
- As a result of these (such as slowing of the blood) the vessel wall is subsequently affected (eg in relation to hypoxia).
- However, the main reason is slowing of blood and coagulation disorder.

Types of thrombosis

I. According to pathogenesis

2. Conglutination thrombosis (Thrombosis related to vascular disorder):

- It is formed by damage to vascular endothelial cells.
- It is more common in arteries.

3. Mixed thrombosis:

- As a result of this type of vessel wall defect, conglutination begins as thrombosis. Then the coagulation continues as thrombosis and completely occlude the vessel lumen.

4. Hyaline thrombosis:

- It stands out both as a thrombosis and as a result of thrombosis. It is formed as a result of hyalinization of the thrombus.

Coagulation thrombosis

- The clot is in the vessel lumen, filling the lumen.
- It does not adhere to the vessel wall.
- When it is removed, there is not much disruption in the endothelium.
- It is not stratified, laminar.
- It is redder because it is rich in erythrocytes.
- It is not elastic. It crumbles easily.
- The upper face is rough (not smooth) according to the degree of stasis.
- It can be broken down by fibrinolysis.

Conglutination thrombosis

- It develops from the region of vascular disorder towards the lumen.
- It is attached to the vessel wall.
- When it is lifted, the vessel wall is endothelial defected, rough, raised in places.
- It is stratified (as the endothelial cells deteriorate and blood cells collapse over a period of time)
- It is generally lighter in color because there are fewer erythrocytes.
- It is not broken down by fibrinolysis.
- Its surface is uneven.
- It may not completely cover the vessel lumen.

Types of thrombosis

II. According to morphology

Plug thrombosis: It completely closes the vessel lumen.

Channeled or perforated thrombosis: The clot has been punctured from several directions. Blood flow is provided through the channels where these holes are formed.

Tailed thrombosis: The tip is elongated like the tail. Conglutination is found in thrombosis. It occurs in the direction of blood flow in veins and in the opposite direction of blood flow in arteries.

Types of thrombosis

III. According to localization

Arterial thrombosis: It is a dry, friable, smooth gray mass. It is more common in coronary, cerebral, iliac and mesenterial arteries. Mostly, conglutination is in the form of thrombosis.

Venous thrombosis (Phlebothrombosis): As a result of blood stagnation, it is mostly seen as coagulation thrombosis.

Cardiac thrombosis: It follows as a result of cessation of blood flow or endocarditis, endocardium, valve disorders. It is more common in heart valves.

The fate of thrombosis

Fibrinolysis

Softening

Organization

Recanalization

Reendothelialization

Hyalinization

Infection

Breakdown

Fibrinolysis

- It is formed when plasmin activated by plasminogen dissolves the clot (fibrin). This happens in two ways:

a) Endogenous:

- Because of the endothelial defect, **factor XII** affects **kallikrein** formation via **prekallikrein.**
- This creates **plasmin** over plasminogen. As a result, the clot lyses and melts.
- It occurs in cases that do not completely close the lumen.

Fibrinolysis

b) Exogenous:

- **Plasmin** is activated by activators released from tissue and blood.
- Such activators (such as lysokinase, urokinase, streptokinases) are either released from tissues in certain regions or plasmin is activated by being released from cells such as endothelium, leukocytes, erythrocytes, and platelets under special conditions such as stress and oxygen deficiency.

Softening

- **It is formed by proteolytic enzymes.**
- Such enzymes, which are generally released from leukocytes, soften the clot and **the clot dissolves and disappears.**
- However, in this case, the fragments from the thrombus cause **embolism.**
- If the thrombosis is caused by bacteria, the effect of neutrophils is greater, and in this case, **septic emboli** develop as bacteria spread by emboli.
- This softening of the thrombosis is called **purulent softening.**

Organization

- It occurs in thrombus that completely fills the lumen.
- Connective tissue precursors originating from the vascular subendothelium first **surround the thrombus from above**, and then **migrate into the connective tissue cells to form granulation tissue.**

Recanalization

- It is formed in areas with high blood pressure.
- With the gradual enlargement of the spaces formed by the melting of the fibrin or the entry of endothelium into these regions as a result of organization, channels and secondary vessel lumens are formed.
- It develops in three main ways.

Reendothelialization

- When adhered to the vessel lumen, endothelial cells surround the surface of the thrombus.

Hyalinization

- It occurs especially in organized thrombosis.
- Calcification is also seen.
- If the calcified areas lithify, phlebolite (vein lith) is formed.

Infection

- Thrombus is conducive to the growth of microorganisms.
- When they come and multiply, they become infected, and **septic embolisms** are formed.

Breakdown

- As a result, **thrombo-embolism** occurs.

Alaka

- It is a **postmortal blood coagulation**, found only in cadavers and confused with thrombosis in necropsy.
- **Features;** It is a postmortal blood clot observed in many vessels, with a soft flexible consistency, dark red color, gelatinous appearance, shiny outer surface, vessel-shaped and not adhered to the vessel wall.

Alaka

- **It appears in two ways:**
- **Currant jelly:** It is a homogeneous mass of red color.
- **Chicken fat:** It occurs when it forms slowly.
  - It is stratified. Its stratification causes it to be confused with this form of thrombosis, but the relevance does not adhere to the vessel wall and takes its shape.
  - A red layer with a collection of erythrocytes at the bottom; On the top, a second layer, similar to chicken fat, is formed, containing fibrin, blood plasma, leukocytes.
  - This is seen in the vascular and cardiac cavities, the animal died at the end of long agony; indicates a defect in blood coagulation.

EMBOLISM

Embolism

- The dissemination of fragments from the thrombus with blood is understood. This is called a **thromboembolism.**
- Embolism is an insoluble, solid (single), solid or gaseous foreign masses that move from one place to another via blood in the vessel, causing occlusion of the vessel where it goes, and this is called **embolism.**

Types of embolisms according to pathways

- **Direct embolism:**
  - It follows the direction of blood flow.
  - For example, embolism in the veins passes to the right heart, and from there to the lungs.
  - Thrombosis in the left heart and aorta is carried to organs such as the brain and kidney by arteries.
- **Retrograde embolism:**
  - It occurs in the opposite direction of blood flow.
  - For example, in right heart failure, the emboli coming from the superior V. cava to the right atrium may move against the blood flow and go to the liver.
- **Paradoxal embolism:**
  - It is an embolism that passes from vein to artery and from artery to vein.
  - Especially when the foramen ovale is open in the heart, it passes to the left heart without passing from the right heart to the lungs.

Types of embolism according to structure

Solid embolism	• Thromboembolism, cell and tissue embolism, parasite embolism, pigment embolism, Atheroma embolism, foreign body embolism
Fluid embolism	• Fat embolism, Bile embolism, Amniotic fluid embolism
Air embolism	• Air and nitrogen gas embolism

Thromboembolism

- It is the **most common** form. It is formed by fragments that break off from thrombi.

  a) Venous thromboembolism:

  - More often, it comes to the lungs.
  - For example, it localises through the leg veins to the right heart and then to the lungs. It varies according to the size of the clogged vessel in the lung. **Sudden death occurs when the truncus pulmonalis or a large vessel is occluded. Hemorrhagic infarction is formed in the occlusion of medium-sized vessels.**
  - This type of embolism can originate from veins, as well as from fragments of thrombosis in the right heart valves. If the foramen ovale is open, the embolus can travel to other organs without reaching the lungs.

Thromboembolism

b) Arterial thromboembolism:

- When thrombosis occurs in the left heart, it occurs in areas such as the brain, kidney, spleen, leg arteries.
- Thrombosis resulting from aneurysm and arteriosclerosis in the aorta, great vessels also has the same result.
- In the other arteries, such as thrombosis caused by **strongylus larvae in A. mesenterica cranialis**, it is seen in the intestines, that is, in the organs where that vein goes.

Thromboembolism

- Embolus containing infectious agents are **called septic embolisms.** Embolus that do not contain infectious agents are called **aseptic (sterile) embolisms.**
- **Septic embolism** results from a septic thrombus in the vessel or from bacterial endocarditis.
- It is caused by inflammation (**especially purulent**) in any part of the body.
- For example, **purulent omphalophlebitis** that occurs in the umbilical cord in newborns spreads to many organs through the blood.
- As a result of **septic valvular endocarditis**, the agents spread through the blood.
- The spread of purulent microorganisms in the blood in this way and their presence in the blood is called **pyemia.**

Cell and tissue embolism

- It is mainly observed in **tumor metastases.**
- After bone fractures and traumatic liver injuries, bone marrow tissue or liver cells are found in the pulmonary vessels.

Fat embolism

- It occurs when the adipose tissue breaks down and passes into the torn veins as a result of trauma.
- It may cause infarction in the organs to which it goes by mixing with circulation.
- Bone marrow can also cause embolism.
- It can be seen after bone and adipose tissue surgeries.

Air embolism

- It occurs as a result of **air leakage into V.jugularis during injection.**
- Especially in the heart cavities, air and gas are encountered.
- In necropsy, if the vessels of the heart are ligated and then opened in water, gas bubbles appear when opening, confirming this.
- In neck operations, air can pass into the veins due to **negative pressure as a result of accidental cutting of great veins,** pneumothorax, lung disorder in costal fractures.

Air embolism

- **In Caisson's disease**, when divers go deep, N2 (nitrogen) gas melts more than normal.
- When it suddenly comes to the surface, there is no opportunity to melt again and it collects in the veins in gaseous form.
- A similar situation occurs in pilots **(Decompression sickness).**
- This time, the normally molten nitrogen turns into gas when you suddenly go up.

Results of embolism

- It varies depending on **the size of the occluded vessel,** whether it is **completely occluded or not**, whether there is a **collateral connection or not.**
- **Sudden death** results from sudden occlusion of the great vessels by fragments from the thrombus.
- If the collateral connection of the occluded vessel is not complete or absent, **infarction** occurs.

INFARCTION

Infarction

- It is an area of **ischemic coagulation necrosis.**
- It is usually formed in organs with **insufficient collateral connections and insufficient vessels.**
- As a result of complete occlusion of the vessel due to a cause such as **embolism, thrombosis or spasm**, blood does not go to the area fed by the vessel and ischemia (local anemia) develops.
- As a result, coagulation necrosis occurs on the side of the occluded vessel, which is called **infarct** and the event is called **infarction.**

Pathogenesis

- **Organ's resistance to lack of oxygen** (For example: kidneys are weak, infarction is more common)
- Insufficient blood circulation and vascularization,
- Whether there are collateral connections or not,
- Whether it is adequately nourished or not fed through other vessels (For example**, infarction is not seen because of vascular anastomoses in skeletal muscles, skin, uterus, thyroid).**

According to morphology

Anemic infarction

- It is formed as a result of arterial occlusion in organs such as the heart, kidney, spleen, and brain.
- It appears pale.

Hemorrhagic infarction

- It is caused by obstruction of the venous system.
- It is most common in the lung.
- It also occurs in the testicles and ovaries.
- It is red in color due to stagnation of blood.

- It is divided into **acute and chronic according to duration.**

Morphology

- It is in the form of **coagulation necrosis.**
- Those that settle in organs such as the **lungs and heart** do not have a specific shape. It is like a map and its borders are **irregular.**
- Those located in the **kidneys and spleen** are **triangular** in shape. The top of this triangle is on the side of the occluded vessel, and the base is in the direction of the capsule of the organ.
- However, just below the capsule is not affected much as it is fed by the blood vessels in the capsule.

Morphology and progress

- Infarct area according to the affected organ;
- Initially, the infarct area is **hyperemic and slightly protruding**.
- After a day or two, **the necrotic part becomes pale**, acquires a grayish color, its borders become clear and there is a **hyperemic zone around it.**
- In the following days, it takes a **gray-yellow color and softens.**
- In this way, the **necrotic region is cleared and organized by macrophage**s. In this situation; It is closed by **granulation tissue** consisting of connective tissue and capillaries developing from the environment.
- Later, the vessels decrease, the collagen threads increase and **scar tissue** is formed. It is also hard, grayish and sunken.

SHOCK

Shock

- Shock (cardiovascular collapse) is a circulatory dyshomeostasis associated with loss of circulating blood volume, reduced cardiac output, and/or inappropriate peripheral vascular resistance.
- However, **collapse** is circulatory failure caused by dilation of peripheral vessels. It results in **fainting.**
- It is generally adapted and recovered with a short rest. Therefore, collapse refers to the collapse of the body, falling, fainting and **cannot be used as a substitute for shock today.**

Shock

- Shock is caused by **acute circulatory failure.**
- In cases of severe pain or psychological stress such as excitement and sadness, neural vasodilation occurs.
- On the other hand, microcirculation is disrupted and blood is collected in the terminal areas formed by arteriolar capillaries and venules, especially in the abdominal cavity organs.
- As a result, venous blood cannot return to the heart.
- In the meantime, since the brain is left without blood, loss of consciousness and fainting occurs.

Clinical features

- Clinical features of shock are **rapidly progressive;**
  - hypotension
  - weak pulse
  - tachycardia
  - hyperventilation with pulmonary rales
  - reduced urine output
  - hypothermia

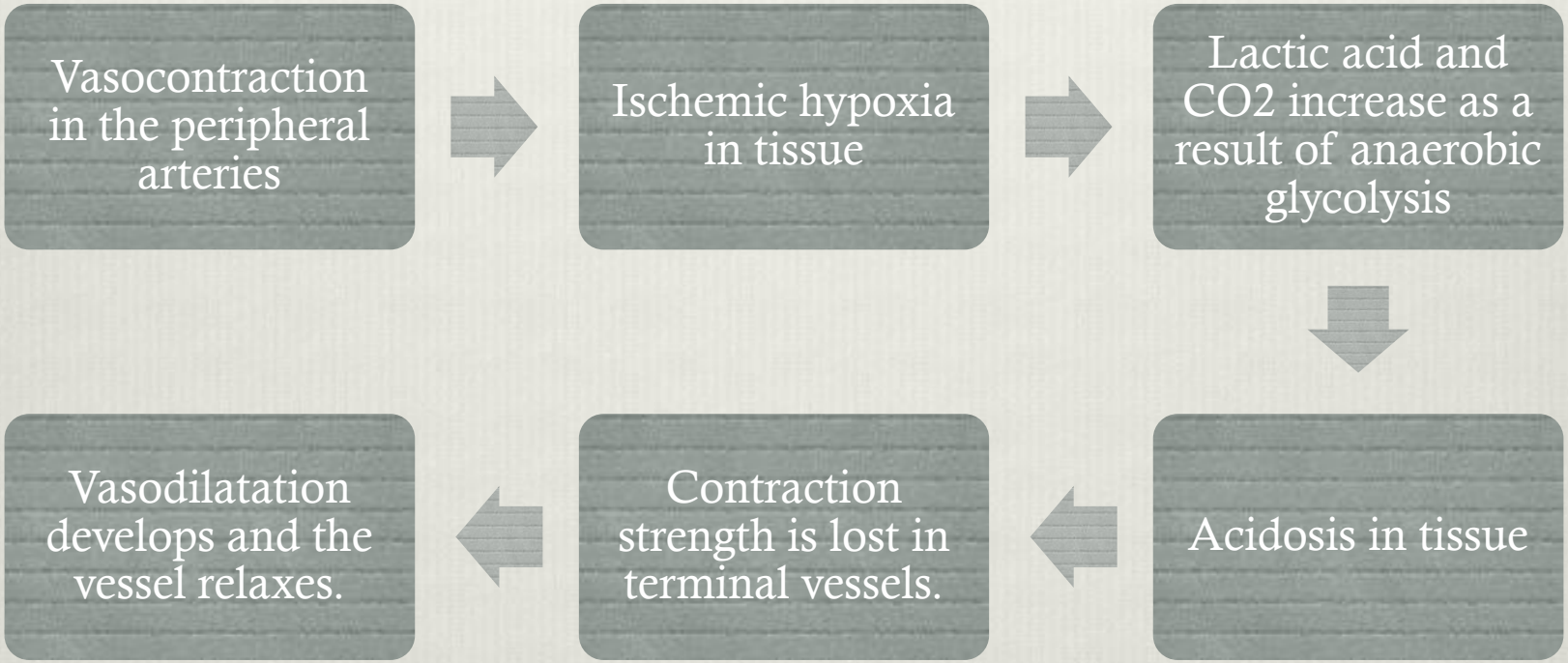
Pathogenesis

- At the onset of shock, **baroreceptors** (pressure receptors in the heart) and **vasomotor centers in the nervous system are disrupted.**
- **Neuro-humoral mechanisms** are activated to maintain the increased heart rate and the blood volume and pressure.
- These mechanisms occur **by stimulating the renin-angiotensin system**, especially by increasing **catecholamine release** and affecting vascular tone.

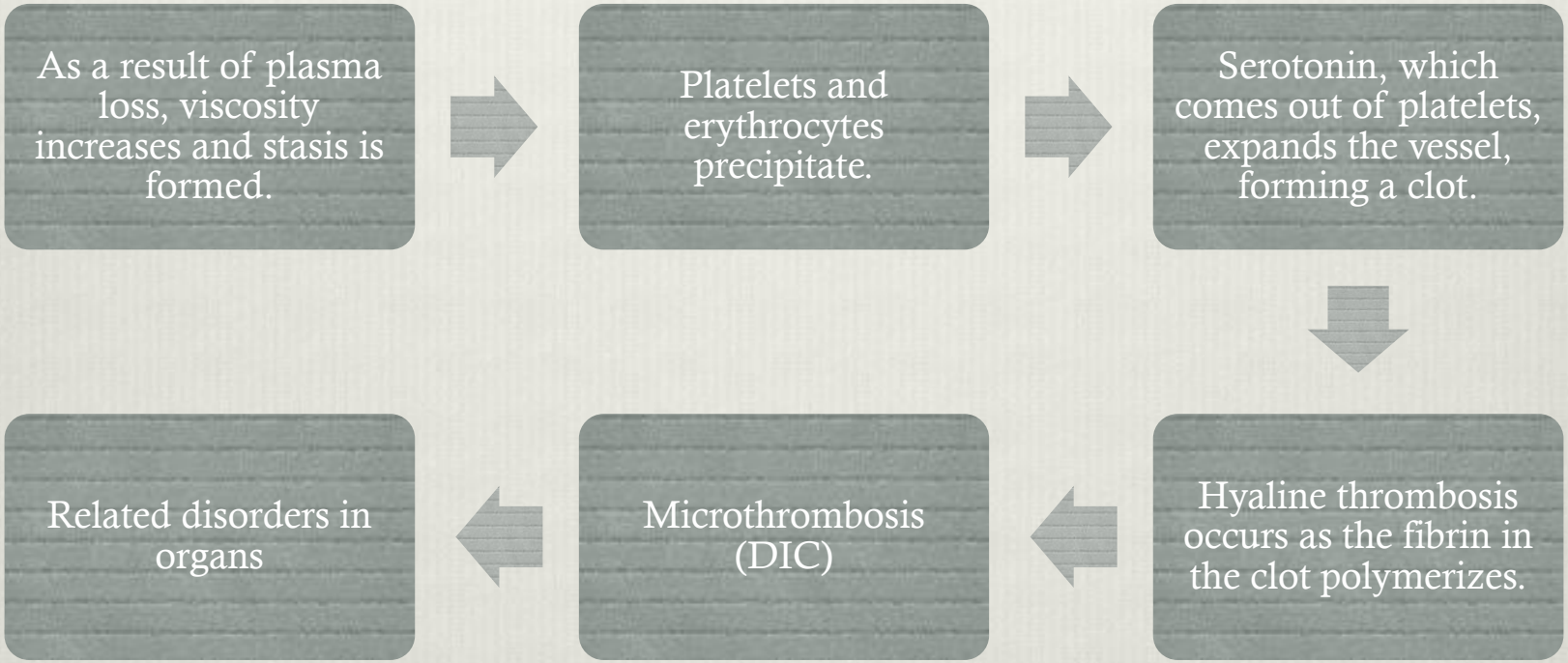
Pathogenesis

- In such a stimulation, different results are obtained from the veins, depending on its location in the body.
- Because **when □-receptors are stimulated**; While **vasoconstriction occurs on kidneys, other abdominal cavity organs, great veins, smooth muscles, skin vessels**, the same effect causes vasodilation by acting on □-receptors in tissue vessels such as skeletal and cardiac muscles that need more oxygen.

Shock


Vasocontraction in the peripheral arteries
Ischemic hypoxia in tissue
Lactic acid and CO2 increase as a result of anaerobic glycolysis
Acidosis in tissue
Contraction strength is lost in terminal vessels.
Vasodilatation develops and the vessel relaxes.

Shock


As a result of plasma loss, viscosity increases and stasis is formed.
Platelets and erythrocytes precipitate.
Serotonin, which comes out of platelets, expands the vessel, forming a clot.
Hyaline thrombosis occurs as the fibrin in the clot polymerizes.
Microthrombosis (DIC)
Related disorders in organs

Stages of shock

Nonprogressive stage

- Compensatory mechanisms that counteract reduced functional circulating blood volume and decreased vascular pressure.
- Notable exceptions are critical tissues, such as the heart, brain, and kidney, to which the blood flow is preserved.

Progressive stage

- Compensatory mechanisms are inadequate.
- As a result of hypoxia, liver, kidney, lung and heart disorders are seen.

Irreversible stage

- As a result of severe hypotension, disorders in organs such as the heart and kidney occur and death occurs.

Progression of shock

- **Secondary changes in the heart, brain, kidney, and lungs**
- **Electrolyte loss, metabolic acidosis, especially kidney failure**

Types of shock

Cardiogenic Shock

Hypovolemic Shock

Septic shock

Neurogenic shock

Shock

- **Cardiogenic shock:**
  - It results from **failure of the heart** to adequately pump blood. Cardiac failure can occur due to myocardial infarction, ventricular tachycardia, fibrillation or other arrhythmias, dilated or hypertrophic cardiomyopathy, obstruction of blood flow from the heart (e.g., pulmonary embolism and pulmonary or aortic stenosis), or other cardiac dysfunctions.
- **Hypovolemic shock:**
  - Arises from reduced circulating blood volume as the result of blood loss caused by hemorrhage or the result of fluid loss secondary to vomiting, diarrhea, or burns
- **Neurogenic shock**:
  - May be induced by trauma, particularly trauma to the nervous system; electrocution, such as by lightning strike; fear; or emotional stress.

Shock

- **Septic shock:**
  - It is formed by the action of bacterial **endo-exotoxins,** especially gram-negative bacteria with lipopolysaccharides (with Lipid-A moieties).
  - In this case, it starts with **microcirculation disorder.**
  - In such events; **Disseminated intravascular coagulation** occurs as a result of vasodilation and permeability disorder, activation of macrophages, mobilization of neutrophil leukocytes, activation of complement (alternative), and endothelial dysfunction.

Shock

- **Septic shock:**
  - **Histamine and serotonin** are released in mast cells.
  - By releasing **catecholamines** (adrenaline, noradrenaline), kallikrein, bradykinins, the vessels contract and thrombosis occurs.
  - It is formed in edema disease in pigs, in infections of *Clostridium, Salmonella, Pseudomonas, Enterobacter* species and especially in enterotoxemic *E. coli.*

Shock

- **Anaphylactic shock:**
  - Generalized type I hypersensitivity
  - Insect or plant allergens, drugs, or vaccines.
  - Immunoglobulin E bound to mast cells results in widespread mast cell degranulation-release of histamine and other vasoactive mediators.
  - Systemic vasodilation-increased vascular permeability-hypotension and tissue hypoperfusion

Shock

- Shock in animals mostly occurs as a result of **infection and intoxication.**
- Since such diseases are also acute and the macroscopic findings of acute shock are not evident, it is difficult to detect them in necropsy.
- It is even **more difficult**, especially in **peracute events.**
- However, **changes related to the difference in blood distribution in the organs are noticeable**.
- In some species, these are **changes in different organs in the form of increasing and decreasing blood.**
- Such organs are called **shock organs**. (heart, brain, kidney, spleen, lung, gastrointestinal and liver)

Shock

- Severe passive hyperemia is observed in the internal organs, especially the spleen is bloody.
- Severe pulmonary edema, passive hyperemia, microthrombosis
- Subepicardial and endocardial petechiae in the heart, focal necrosis of the myocardium, bleeding areas and microthrombosis
- In the gastrointestinal tract, edema of the mucosa, petechial hemorrhages, sometimes erosions and ulcers are observed.
- Central necrosis of the liver, sometimes due to lipidosis and ischemia.

Shock

- Passive hyperemia in the kidneys, dilatation of the tubules, microthrombosis in the glomerulus capillaries, necrosis in the renal cortex are seen.
- Edema in the leptomeninges of the brain, malacia due to microthrombosis, hemorrhages, and medial necrosis in the arteries are observed, especially in the brain basal.
- Hyperplasia in the adrenal glands, lipid accumulation in the cortex, medial necrosis in large vessels,
- In long-term cases, atrophy occurs in the thymus and lymph nodes.