EduVision: Journal of Innovations in Pedagogy and Educational Advancements
Volume 01, Issue 03, March, 2025
brightmindpublishing.com
ISSN (E): 3061-6972

PATHOLOGICAL PHYSIOLOGY OF PERIPHERAL CIRCULATION

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Abstract:
The work examines the general blood circulation and studies typical peripheral disorders. It is shown that pathological arterial hyperemia can be divided by the mechanism of development into neurogenic and developing under the direct action of metabolites on the walls of blood vessels. Characteristic signs of ischemia and classification of thrombi are given.

Keywords: Blood circulation, peripheral , disorder, ischemia, pathology, hyperemia, neurogenic, metabolite, wall, vessel, signs, ischemia, thrombus.

Introduction

The blood supply of animals is provided by the circulatory system. The circulation of blood in a closed system depends on the activity of the heart and the condition of the blood vessels. According to functional characteristics, blood circulation is conventionally divided into general (central, systemic) and local (peripheral, organ).

General circulation is carried out by the heart and large vessels, and local circulation determines the blood flow through the arteries and veins of organs and tissues. Peripheral typical circulatory disorders include arterial and venous hyperemia, ischemia, stras, thrombosis, embolism.

Hyperemia (from the Greek hyper - above, haima - blood) is an increase in the amount of blood in any organ or part of an organ. An increase in the blood supply to any part of the body or organ can be caused by either an increased flow of arterial blood compared to the norm or a decrease in the outflow of venous blood. In connection with this, hyperemia is divided into two types: arterial (active) and venous (stagnant, passive).

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Arterial hyperemia develops with increased arterial blood flow and normal venous blood outflow.

By origin, physiological and pathological arterial hyperemia are distinguished. They are distinguished by two criteria - adequacy and adaptability. Adequacy is the correspondence of arterial hyperemia to changes in function and metabolism in organs and tissues.

Adaptability is the presence (or absence) of adaptive biological significance of arterial hyperemia in each specific case. The causes of physiological hyperemia are: local action of irritants (turpentine, camphor); mechanical friction, massage; local decrease in atmospheric pressure (cups); warming (hot-water bottles, compresses); stress (general hyperemia).

The causes of pathological arterial hyperemia are: lack of oxygen; acidosis; paralysis of vasoconstrictor nerves or their centers; excitation of vasodilator nerves or their centers; decreased tone or paralysis of the muscles of the vascular wall.

Physiological arterial hyperemia is adequate to the effect of the irritant and has an adaptive value. It can be functional and protective-adaptive.

1) Functional develops in organs and tissues due to an increase in the level of their functional activity (for example, hyperemia in a contracting muscle or in an intensively working organ).

2) Protective-adaptive arterial hyperemia develops during the implementation of protective reactions and processes (for example, in the inflammation focus or around a foreign transplant, necrosis zone or hemorrhage). In these cases, arterial hyperemia facilitates the delivery of oxygen, metabolic substrates, immunoglobulins, phagocytes, lymphocytes, other cells and agents to the tissues, which are necessary for the implementation of local protective and restorative reactions.

Pathological arterial hyperemia develops as a result of inadequate action of irritants of various origins on tissues, regardless of organ function. Pathological arterial hyperemia is divided by the mechanism of development into neurogenic and developing under the direct action of metabolites on the walls of blood vessels, i.e. vasomotor and destructive.

I. Vasomotor (angioneurotic) hyperemia, arising as a result of irritation of the vasodilator nerves and (often) simultaneous paralysis of the vasoconstrictor nerves, include:

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1) neurogenic,
2) myoparalytic ,
3) reflex.

Neurogenic arterial hyperemia has two types:

a) Neurotonic arterial hyperemia, which occurs reflexively as a result of irritation of extero- and interoreceptors and the direct action of the pathogen on the vasomotor centers.

b) Neuroparalytic arterial hyperemia may develop when the vasoconstrictor vasomotor center is damaged under the influence of chemical or physical effects. In this case, the vasomotor mechanism of arterial hyperemia, characteristic of the inflammatory process, is to a certain extent associated with paralysis of the vasoconstrictor apparatus.

2) Myoparalytic arterial hyperemia develops with predominant damage to the vascular wall itself, under exposure of tissue to cold, heat (compresses), mechanical irritation, chemical compounds, and is accompanied by a decrease in the tone of the smooth muscle elements of the vascular wall.

This type of arterial hyperemia may include vacant hyperemia (from the Latin vacuus - empty), which develops due to a decrease in barometric pressure above the tissue, for example, after removing a medical cup, a limited area of hyperemia appears on the skin.

3) Reflex arterial hyperemia: a) P -postemic arterial hyperemia can be observed when fluid is quickly removed from the pleural (hydrothorax) or abdominal (ascites) cavities during the development of dropsy. Due to the large capacity of the vessels supplying the abdominal organs, especially the mesenteric vessels, with the rapid removal of gases from the rumen in cattle, a complication in the form of collapse developing as a result of cerebral ischemia is possible. Therefore, the removal of gases and fluid from the cavities of sick animals should be done very carefully, slowly, in order to prevent blood deposition and cerebral ischemia.

b) Collateral arterial hyperemia develops in cases where the blood flow in the central vessel of the arterial system stops as a result of thrombosis or embolism. In this case, the blood passes through the lateral branches of the arterial system, which are called collateral vessels. This type of hyperemia has a great positive effect on the body, since, despite the thrombus or embolus that has closed the lumen of the

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main arterial trunk, the blood supply to the organ does not stop and its vital functions are not disrupted.

c) Inflammatory arterial hyperemia always accompanies the inflammatory process.

II. Destructive (traumatic) arterial hyperemia occurs when the connective tissue base of the vessel wall is damaged.

Arterial hyperemia is manifested by a number of characteristic signs: - bright red coloration of non-pigmented organs, tissues, areas due to increased flow of oxygenated blood);

- an increase in local temperature due to the influx of blood and the intensification of metabolic processes;

- an increase in the magnitude of pulsation of arterioles and small arteries due to the spread of the pulse wave from large main vessels to small-caliber vessels.

- an increase in the organ's volume due to increased blood filling, excess accumulation of interstitial fluid and increased tissue turgor;

- an increase in the number of visible vessels as a result of previously non-functioning vessels filling with blood;

-increased lymph formation and lymph flow ; -increased functional activity of an organ, tissue, or part of an organ.

Venous (stagnant, passive) hyperemia is characterized by an increased blood content in any area of the body due to obstructed outflow through the veins. Obstructed outflow also affects the inflow of arterial blood, the amount of which decreases somewhat. At the same time, the amount of circulating blood in a given area or organ slows down. That is why venous hyperemia is stagnant.

The etiology of venous hyperemia may be different. In the case of a local process, the most common cause is compression of venous vessels by bandages, tourniquets, tight harness, tumor, scar tissue, pregnant uterus, tympany of the scar and intestinal flatulence, narrowing of the lumen of venous vessels and thickening of their walls in phlebitis. The causes of extensive venous hyperemia are associated with weakening of cardiac activity (especially its right half) and congestion in the systemic circulation.

Based on the etiology and mechanism of development, the following types of venous hyperemia are distinguished:

1) obstructive venous hyperemia caused by blockage of the lumen of the vein by a thrombus or embolus ;

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2) compression venous hyperemia, observed when a vein is compressed from the outside by inflammatory edema, a tumor, a ligature, or proliferating connective tissue;
3) collateral venous congestion, which can be observed when a large main venous vessel is closed;
4) destructive venous hyperemia, which occurs when the connective tissue base of the vessel wall is damaged.
Signs of venous hyperemia are caused by decreased blood flow, overfilling of the organ with venous blood. This leads to hypoxia of circulatory origin, accumulation of metabolic products, and an increase in the content of reduced hemoglobin in the blood.
One of the cardinal signs of venous congestion appears - cyanosis (blueness) of the congested area. Slowing down of metabolic processes, accumulation of venous blood in the tissue are manifested by a decrease in the temperature of superficially located organs. Increased pressure in the capillary network, postcapillaries and venules leads to the removal of transuding fluid beyond their limits , stretching the tissue gaps and accumulating there in significant quantities. All this leads to an increase in the organ's volume - edema.
Ischemia (from the Greek ischein - I delay, I stop and haima - blood) is a condition of complete cessation or reduction of arterial blood flow in any part of the body, organ or tissue.
Depending on the causes that cause narrowing of the lumen of the arterial vessels and difficulty in blood flow to a certain area, the following types of ischemia are distinguished:
1) Compression ischemia, which occurs as a result of compression of a vessel or area of tissue by accumulated fluid, a growing tumor, foreign bodies, etc. , i.e. the cause of the narrowing of the lumen of the artery lies outside the vessel.
2) Angiospastic ischemia, which can be caused by the action of various factors on the vasoconstrictor nerve elements of the arterial wall, including trauma, cold, chemical substances of hormonal nature (catecholamines, vasopressin, etc.), pharmacological agents, substances of plant, animal, mineral origin, bacterial toxins. These factors act directly on the vasomotor centers and the nervous apparatus of the vessels, as well as reflexively.

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3) Obstructive ischemia, which occurs as a result of complete or partial closure of a vessel due to thrombosis, embolism, pathological thickening of the inner wall of an arterial vessel due to atherosclerosis, etc.
4) Reflex ischemia, which occurs when the receptors of internal organs are irritated, when the effect of irritation is transmitted reflexively to the vessels of other organs. For example, when the angioreceptors of the bile ducts and intestines are irritated, a spasm of the heart vessels occurs.
5) Collateral ischemia, which develops as a result of a sharp shift of blood into the vessels of a certain area of the body and, accordingly, depletion of blood in other areas. For example, local collateral anemia of the brain, lungs or extremities may occur with a rapid shift of blood into the vessels of the abdominal cavity after a sharp removal of gases from the rumen in tympania, or rapid pumping of fluid out of the abdominal cavity in ascites.
6) Paralytic ischemia, observed in long-term paralysis of the limbs due to loss of their function. Dilatins , which under normal conditions contribute to the expansion of arteries, are not formed in the vessels, as a result of which the organ gradually " dries up". During ischemia, the volume of the microcirculatory bed decreases, which causes a decrease in the formation of tissue fluid in the ischemic area and a disruption of lymph flow .
Metabolic disorders develop due to a lack of oxygen and nutrients, and in the affected area, products of under-oxidized metabolism accumulate, causing intoxication.
In addition, nutritional deficiency leads to “local tissue starvation,” which leads to disruption of the organ’s morphology (granular, fatty degeneration, atrophy, necrosis).
The following signs are characteristic of ischemia:
- blanching of the area, the severity of which depends on the degree of reduction in blood flow to it;
- a decrease in the temperature of the superficially located ischemic area due to the prevalence of heat transfer over heat production;
- a decrease in the volume of the organ with a decrease in tissue turgor;
- disappearance of normally visible small vessels (veins and arteries);
- deterioration of organ function, perversion of sensitivity - paresthesia due to disruption of the nutrition of nerve endings;

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-pain syndrome;
-dystrophic processes.
Thrombosis (from the Greek trombosis – clotting) is the formation during life of clots inside a vessel that are connected to its wall, called thrombi, and that impede blood flow due to narrowing or complete closure of the lumen of the vessel.
Thrombosis is caused mainly by three factors: damage to the vessel wall, slowing and disruption of blood flow, and changes in the coagulation and fibrinolytic systems of the blood.
Classification of thrombi. According to their location in the vessel, the following types of thrombi are distinguished:
a) a mural thrombus, which forms directly at the wall at the site of damaged intima of the vessel and narrows its lumen; in this case, blood flow occurs at the free part of the wall;
b) longitudinal or continuous is a type of parietal thrombus. Its head, fixed to the vessel wall, gradually grows and is located longitudinally to the vessel, and its tail is not fixed to the wall. Such a thrombus can be of considerable length, increasing in diameter, it can become obturating ;
c) an occluding or obturating thrombus that completely closes the lumen of the vessel. This type of thrombus is more common in small diameter vessels;
c) the central thrombus is located in its main mass in the center of the vessel and is fixed to the wall by strands, while blood flow is maintained along the part of the vessel not occupied by the thrombus; d) lining or obliterating - lines the wall of the vessel, leaving only a small lumen for blood flow.
According to the mechanism of formation and structure, white, agglutination and mixed thrombi are distinguished.
Thrombus formation occurs in two phases.
The first phase is basically a physical process in which platelets are layered and glued together on a pathologically altered area of the inner surface of the vascular wall. The thrombus formed in this phase is called primary or agglutination , or white, and consists of platelets, leukocytes, and some plasma proteins. Typically, such a thrombus is parietal, tightly connected to the vessel wall, and does not cause serious complications for blood flow in large vessels.
The second phase of thrombus formation is basically an enzymatic, coagulation process and consists of the release of thromboplastin from disintegrating platelets,

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under the action of which, together with other elements of the coagulation system, the coagulation of plasma fibrinogen into fibrin occurs.

The precipitated fibrin in the form of thin threads forms a network into which erythrocytes fall, imparting their color to the thrombus, which is called red.

Red (coagulation) thrombi are a dark red mass that often closes the lumen of a vessel, most often a venous one. The main factor in the formation of a red thrombus is a sharp slowdown in blood flow, and its formation is always preceded by the formation of a primary, white thrombus.

Red thrombi are weakly attached to the vessel wall, they are loose in consistency, easily break off and, getting into the bloodstream, can cause thromboembolism; two different phenomena in genesis - thrombosis and embolism can ultimately cause vessel blockage.

Layered or mixed thrombi are also distinguished. The beginning of their formation is the appearance of a white thrombus, after some time the thrombocytes and leukocytes in it are destroyed, thrombokinase is released, fibrin precipitates, and erythrocytes begin to get stuck in its threads. Then, with a change in the physical and chemical conditions in the vessel and on the surface of the red part of the thrombus, leukocytes and thrombocytes agglutinate again, and a layer of white thrombus appears.

Embolism (from the Greek. Emboli - wedging) - the movement of foreign particles unusual for blood and lymph through blood and lymphatic vessels and their blockage of the lumen of the vessels. Particles or bodies carried by the blood and lymph flow and blocking the vessels are called emboli .

They can enter the vessels from the external environment or form in the body itself. Therefore, embolisms of exogenous and endogenous origin are distinguished.

Embolisms of exogenous origin include: bacterial, parasitic, air and gas. Embolisms of endogenous origin include: thrombotic , fatty and tissue.

Embolism by parasites and bacteria can occur when bacteria or parasite larvae enter the blood and lymphatic vessels en masse from infection sites. Air embolism occurs when air from outside enters the venous vessels, which occurs with injuries to large veins located close to the chest and heart (jugular, subclavian or vena cava).

In this case, with a sharp expansion of the chest during inhalation and at the moment of diastole of the right atrium, the pressure in these veins will be lower than atmospheric (negative) and atmospheric air, due to the difference in pressure, is

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freely sucked in through the ruptured vessels in the form of bubbles and moves with the blood flow into the right half of the heart.

There, in the form of a large bubble, it can block the right atrium, which leads to cardiac arrest, or, breaking up into small bubbles, it is pushed into the right ventricle and blocks the vessels of the pulmonary circulation.

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