

CLINICAL SIGNIFICANCE OF PATHOPHYSIOLOGICAL CHANGES IN THE BODY OF THE VICTIM AS A RESULT OF BLEEDING AND BLOOD LOSS

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A person's life depends on the functions of his organs and systems, and they can function normally only with good blood circulation in the body as a whole. Hemodynamics are provided by the work of the cardiovascular system and the volume of circulating blood. It is with this importance of the problem of bleeding and blood loss due to the frequency and prevalence of this pathology in all segments of the population; a medical worker of any specialization may be involved in bleeding; bleeding almost always accompanies such emergencies as traffic accidents and accidents, as well as complicates various diseases and is always present during surgery; untimely assistance in bleeding threatens life-threatening complications - a decrease in circulating blood volume, the development of hemodynamic disorders and hemorrhagic shock, and as a result of the death of the victim; a doctor of any specialty should be able to provide first aid to a patient with bleeding.

Blood is not just a transport medium that combines various organs and tissues into a single organism. In addition to transporting gases and bioactive substances, quantum information and control, it performs many compensatory functions, immune protection

and more. Like all organs and systems of the body, blood is genetically specific, its cellular and biochemical composition is constantly self-reproducing. Blood is the same "native" and irreplaceable to foreign tissues system of the body as all its other systems and organs. It, like other vital organs and systems, performs many functions. If it is damaged, a decrease in its volume cannot be an isolated damage to any of the functions of the blood. That is, the artificial restoration of blood gas transport function is better than nothing, but still not full compensation of all blood functions.

Like other body systems, blood has its own mechanisms of autocompensation, as well as compensatory mechanisms that are inherent in other systems. Damage or reduction in blood volume that is not compensated by its own mechanisms leads to changes in the function of the cardiovascular system, metabolism, etc., aimed at compensating for blood loss. The initial reactions of the body to acute blood loss depend on the rate at which blood volume decreases, and on the volume of blood loss, and on the previous state of the body.

Compensatory reactions that begin under autoregulation are aimed at rapidly restoring blood volume and quality. Compensatory mechanisms are activated in all functional systems of the body, starting with the blood system itself. The amount of reserves of compensatory capabilities of each organism in response to blood loss differs in individual features, which depend not only on the previous functional state of systems and organs, but also on constitutionally established and which must be taken into account when providing care.

High-grade blood loss leads to significant dysfunction of organs and systems, as well as disturbances in the body's immune system, causing significant changes in the state of cytokines. The most active role is played by cytokines of pro-inflammatory and anti-inflammatory direction. To date, most diseases are associated with immune system disorders. Gastrointestinal bleeding, which is a consequence of damage to blood tissue and reflects its severity, is no exception. As a result, an inflammatory reaction on the part of the damaged tissue is formed in response. In this case, at all stages of the formation of a specific immune response of the organism, the dominant role belongs to cytokines. Increased levels of cytokines are an important component of the body's adequate response during inflammation. At the same time, overexpression of these mediators causes changes in physiological processes in the body. Studying these changes can help predict the severity of homeostasis, the development of complications and disease outbreaks, and possible changes in treatment tactics.

Bleeding is not a simple pathological condition, a complication of a certain group of diseases and injuries. Bleeding is one of the most dramatic situations in medicine, and therefore occupies a special place in surgery, in the life of the surgeon. This value of bleeding is determined by the following features:

- * Bleeding - a complication of many, even minor diseases and injuries, as well as a consequence of the surgeon's actions.
- * Unstoppable bleeding - a direct threat to the patient's life.
- * During bleeding, the speed of decision-making and assistance becomes extremely important.
- * The surgeon's ability to deal with bleeding is an indicator of his professionalism.

Compensatory reactions, which begin in the conditions of autoregulation, are intended to immediately restore the volume and then the amount of blood. Compensatory mechanisms are activated in all functional systems of the body, starting with the blood system itself.

Blood system. Under conditions when the amount of hemoglobin has decreased, the shape of the oxyhemoglobin dissociation curve changes, due to which the efficiency of O₂ transfer to tissues increases. It is known that sufficient oxygen extraction to tissues is maintained even at a hematocrit of 15%. At the same time, the mechanisms of erythropoiesis are activated, and new blood cells, including immature erythrocytes and other blood cells, enter the bloodstream. The blood coagulation system responds with hypercoagulation to stop the bleeding more quickly, and if this compensatory hypercoagulation does not stop, it can (and usually does) lead to the development of widespread intravascular coagulation syndrome, which increases the bleeding. The possibility of its development should always be taken into account when developing tactics for the management of patients with blood loss.

Restoration of blood volume is an immediate reaction of the body, which is carried out through hormonal and nervous regulation. Under conditions of prolonged bleeding (hemorrhagic shock), the blood as an organ is damaged: its transport function is disrupted, the coagulation system, anticoagulant system and fibrinolysis system suffer (coagulopathy occurs), the function of the reticuloendothelial system, immune defense, buffer, etc.

Circulatory system. Sudden hypovolaemia associated with blood loss results in the migration of extracellular fluid into the vascular bed. The physiological mechanism of this migration is the spasm of arterioles, the reduction of hydrostatic capillary pressure and the transition of pericapillary fluid into the capillary. During the first 5 minutes after blood loss, the amount of fluid that corresponds to 10-15% of normal BCC can pass into the vessels. Increased secretion of pituitary antidiuretic hormone and aldosterone, which increase water reabsorption in the renal tubules, if glomerular filtration is not significantly impaired. Due to this, the BCC does not decrease or even increase. The reflex that provides this mechanism begins with the volume receptors of the heart and large vessels and closes through the hypothalamus in the pituitary gland and adrenal glands. Decreased cardiac output leads to an increase in vascular resistance in some organs and tissues to direct the main blood flow to the brain and myocardium. This reflex response begins with afferent neurons that engage the baroreceptor reflex, which is stimulated by a decrease in mean arterial and pulse pressure. Reflexes from chemoreceptors are also involved in the reaction, which are stimulated by a decrease in local blood flow and changes in PaO₂ and PACO₂. First of all, the vessels-containers react - veins, which contain up to 2/3 of BCC, due to which the emptiness of the vein is one of the most important signs that require active intervention in case of blood loss.

Lowering blood pressure can also be an adaptive response of the body to stop bleeding. Premature use of vasopressors on the background of unstoppable bleeding to reach the standard of 120/70 mm Hg. Art. - this is a frequent, unfortunately, consequence of instructed medical thinking. If cardiac output continues to decline, arterioles spasm, which should centralize blood flow. However, arteriolospasm leads to a decrease in the volumetric velocity of blood flow in the capillaries, where due to

changes in blood rheology, cell aggregation with sludge phenomena occurs. This ends with the sequestration of blood in the affected capillaries, which further reduces BCC, disrupts venous inflow and exacerbates hypovolemia.

Stimulation of sympathetic-adrenal activity in hypovolemia not only increases peripheral vascular resistance, but also accelerates heart rate, increases heart rate, increases oxygen demand due to increased basal metabolism.

When assessing the physiological effects of hypovolemia, it should be borne in mind that a decrease in BCC by 10% is manifested by nothing but some tachycardia and reduction of vascular capacity. Loss of 15% BCC leads to moderate rheological disorders, which are compensated by the inflow of vascular tissue fluid for the next 2-3 hours. Hypovolemia with a reduction in BCC by 20% reduces cardiac output and creates a defective rheological circuit.

Respiratory system. There are changes in gas exchange, which have different nature. Initially, adaptive hyperventilation, aimed at increasing venous inflow by the suction action of the thorax, leads to respiratory alkalosis. Oxygen saturation of hemoglobin and oxygen content in arterial blood at the same time increase insignificantly.

Due to the fact that in hypovolemia blood flow in most organs is reduced, and their need for oxygen is not changed or even increased (stimulation of the sympathetic system), venous blood flowing from such organs contains little oxygen and arteriovenous difference in the hypoxic organ is increased. Thus, a decrease in the oxygen content in the mixed venous blood in hypovolemia indicates that the patient suffers from hypoxia, even if the oxygen content in the arterial blood is satisfactory.

But the normal oxygen content in mixed venous blood with blood loss does not mean that there is no hypoxia. Just in extreme cases of hypovolemia, the capillaries of many tissues are completely excluded from the microcirculation and the most severe hypoxia of these tissues has no imprint in the figures of the total oxygen content in the mixed venous blood. It should be emphasized that in most cases of blood loss, including massive (more than 30% BCC), oxygen starvation of tissues occurs not only due to hemic hypoxia, but also due to circulatory. That is, the first and main task is not to add hemoglobin, but to normalize microcirculation.

Blood loss, like any hypovolemia, always damages the lungs because the pulmonary capillary filter is clogged with aggressive mediators and metabolites that come from the tissue structures of the microcirculation. Together with them from fabrics units which block the pulmonary capillary filter arrive, and this combined blow of physiological consequences of a hypovolemia on lungs leads to a syndrome of acute damage of lungs which essence consists in the interstitial hypostasis expressed by an alveolar shunt, disturbance of an alveophatocapillar. increases the oxygen cost of respiration, etc. All bleeding can be acute or chronic. In acute bleeding, bleeding occurs over a short period of time, and in chronic bleeding occurs gradually, in small portions. Sometimes for several days there is a slight, sometimes periodic bleeding. Chronic bleeding can be observed in gastric and duodenal ulcers, malignant tumors, hemorrhoids, uterine fibroids and the like.

Assessment of the severity of blood loss is extremely important, because it determines the nature of circulatory disorders in the patient's body and, ultimately, the

risk of bleeding for the patient's life. Death from bleeding occurs due to circulatory disorders (acute cardiovascular failure), and, much less frequently, due to loss of functional properties of the blood (oxygen, carbon dioxide, nutrients and metabolic products). Two factors are crucial for the development of bleeding: the volume and rate of blood loss. Simultaneous loss of up to 40% of circulating blood volume (BCV) is considered incompatible with life. At the same time, there are situations when, against the background of chronic or periodic bleeding, patients lose much more blood, significantly reduced red blood cell count, but the patient gets out of bed, walks, and sometimes works. The general condition of the patient is also of some importance - the background on which the bleeding occurs: the presence of shock (traumatic), previous anemia, exhaustion, cardiovascular insufficiency, as well as the sex and age of the patient.

There are different classifications of the severity of blood loss. It is most convenient to determine 4 degrees of severity of blood loss: *mild, moderate, severe and massive*.

* Mild degree - loss of up to 10-12% BCC (500-700 ml).

* Medium degree - loss of up to 15-20% of BCC (1000-1400 ml).

* Severe - loss of 20-30% of BCC (1500-2000 ml).

* Massive blood loss - loss of more than 30% of BCC (more than 2000 ml).

Determining the severity of blood loss is extremely important to address the tactics of treatment, as well as determines the nature of transfusion therapy.

The relationship between blood circulation and respiration in hypovolemia is always complex. Hyperventilation as an adaptive reaction of hypovolemia, aimed at increasing venous inflow, is accompanied by different effects of inhalation (as well as exhalation) on the large and small circulation. Thus, when inhaling, the filling of the right ventricle, pulmonary vessels, and also the right ventricular stroke volume increases, while for the left ventricle the same indicators decrease. Because each breath has several heartbeats, the pressure in the aorta and pulmonary artery changes in the opposite direction during the breath. Baroreceptors and lung stretch receptors cause the heart rate to increase or slow with each breath.

Such physiological relationships have no clinical manifestations in health, but with blood loss, pericarditis, increased elastic or inelastic lung resistance, hemodynamic differences with each inhalation or exhalation become quite clear, and the pulsus paradoxus, if sought, is always determined.

Other systems. Blood loss is accompanied by damage to organ blood flow, which primarily impairs kidney and liver function. CNS insufficiency occurs last, because the centralization of blood flow in hypovolemia allows for a long time to maintain adequate blood supply to the brain.

Metabolism. Reduction of tissue blood flow leads to metabolic disorders, which due to lack of oxygen becomes anaerobic. In addition, there is an accumulation of lactic acid, anaerobic glycolysis gives 15 times less energy than aerobic. Metabolic acidosis occurs, which has a detrimental effect primarily on the circulatory system itself. It suppresses the myocardium, reduces its reactivity to sympathetic stimulation and promotes the growth of hypovolemia, which in turn exacerbates disturbances in the microcirculation system. Acidosis shifts the oxyhemoglobin dissociation curve down and to the right, causing pulmonary capillary blood to receive less oxygen than at

normal pH. Similarly, in the tissues, the transfer of oxygen through the blood is facilitated. If the pulmonary effect predominates, acidosis leads to severe arterial and venous hypoxemia, and if tissue, then venous hypoxemia may be less pronounced, although arterial remains at the previous level.

Acidosis increases the permeability of membranes, the transudation of fluid from the vascular bed increases, resulting in BCC decreases even more. For the same reason, the level of electrolytes changes and hemodynamics suffers from additional disorders of myocardial contractility.

Hemorrhagic shock. In the later stages of massive blood loss, hemorrhagic shock occurs, which is essentially a multiple organ failure (PON), the trigger of which is blood loss. The components of PON in hemorrhagic shock can be systematized as follows.

First, there is hypovolemia with a violation of the rheological properties of blood and its sequestration in the capillary systems. Due to the sequestration of blood in hemorrhagic shock, the ascending BCC of 5 l after blood loss in 1 l will not be equal to 4 l, but only 3-3.5 l, because some blood volume is sequestered in the capillary systems.

Secondly, generalized metabolic disorders develop - redox processes are disturbed, metabolic acidosis occurs, electrolyte composition of tissues, oncotic pressure, etc. change.

Third, ischemia of organs leads to organ disorders - renal, hepatic, pulmonary (emphasize, pulmonary and not just respiratory failure!), Dysfunction of the myocardium, intestines and more.

Fourth, infectious diseases and purulent-septic lesions are exacerbated or recurrent, and finally, fifth, coagulopathy is a type of disseminated intravascular coagulation syndrome.

Therefore, hemorrhagic shock is a multiorgan failure that occurs as a result of unreplaced or untimely reimbursed massive blood loss.

Hemorrhagic shock is a type of hypovolemic shock. The clinical picture of shock can occur starting with blood loss of 20-30% of BCC, which largely depends on the ascending condition of the patient.

Determine the 3 stages of hemorrhagic shock:

Stage 1 - compensated reverse shock;

Stage 2 - decompensated reverse shock;

Stage 3 - irreversible shock.

Compensated shock is characterized by a volume of blood loss that is well replenished by the compensatory and adaptive capabilities of the patient's body.

Decompensated shock is characterized by deeper circulatory disorders, provided that the spasm of the arterioles can no longer support the central hemodynamics, the normal value of blood pressure. Later, due to the accumulation of metabolites in the tissues, there is paresis of the capillary bed, decentralization of blood flow develops.

Irreversible hemorrhagic shock is characterized by prolonged (more than 12 hours) uncontrolled hypotension, ineffectiveness of transfusion therapy, the development of multiple organ failure.

To assess the severity and determine the stage of shock, it is necessary to take into account the general condition of the patient, pulse rate, blood pressure, respiration (frequency, mode), the amount of urine in milliliters per 1 hour (hourly diuresis), shock index. In assessing the state of blood circulation is important level of blood pressure, heart rate. With the development of shock, the pulse rate increases and systolic pressure decreases. With this in mind, Algover proposed a shock index, which is determined by the ratio of heart rate and systolic pressure. Normally, the Algover index is equal to: $\text{Pulse bout./min. AT120mmpt.ct.} = 0.5$ At the developed, heavy shock the Algover index is equal: $120 \text{ beats / min. } 80 \text{ mmrt. Art. } -1.5$ The larger the index, the more severe the shock and approximately the Algover index can be concluded about the amount of blood loss. At the Algover index equal to 1 blood loss is 20% BCC; at an index of 1.3-1.4 - blood loss of 30% BCC; at 1,5 - blood loss makes 40-50% of BCC.

In severe acute blood loss, treatment begins with a jet infusion of blood into 1-2 veins and after raising the systolic pressure to 80 mm Hg. switch to drip. Cardiac drugs, vitamins C, WB, vicasol, calcium gluconate, glucocorticoids, prednisolone and others are used.

Thus, when providing medical care for bleeding, the surgeon must solve three main tasks:

- * in the near future at least temporarily stop the bleeding, ie interrupt the patient's loss of blood and thus eliminate the threat to his life;
- * achieve reliable cessation of bleeding with minimal loss of function of various organs and systems of the body;
- * to restore in an organism the disturbances which have arisen owing to blood loss.

Temporary cessation of bleeding:

- a) a compressive bandage;
- b) lifting the limb;
- c) maximum bending;
- d) finger pressing - to know the points;
- e) imposition of a tourniquet;
- e) application of a hemostatic clamp in the wound.

For the final stop of bleeding use the following methods:

- a) mechanical - ligation of vessels in the wound, twisting of the bleeding vessel, ligation of the vessel during (during bleeding in a purulent wound), imposition of a vascular suture (lateral or circular);
- b) physical - cold, hot solution, diathermy;
- c) chemical - adrenaline, calcium chloride solution, aminocaproic acid;
- d) biological - tamponade with omentum, muscle, blood transfusion, administration of vitamins, local use of blood products (thrombin, hemostatic sponge, biological aseptic tampon, etc.).

A vascular suture is applied when a large vessel is injured, the ligation of which is life-threatening in general or the viability of tissues located distal to the site of injury.

Bleeding contains other threats, so when large veins are injured, air embolism is possible. At bleedings in a pericardial cavity there is a cardiac tamponade, at hemorrhages in a brain there are paralyzes and paresis, and at localization in the vital centers and death. The cause of death from bleeding - paralysis of the vascular or

respiratory centers. Blood loss is about 4- 4.5%, relative to body weight is considered fatal. Therefore, knowledge of the clinic and diagnosis of external and internal bleeding, the ability to establish their nature, timely provide first aid and medical care, be able to stop bleeding, necessary in the training of doctors of any specialty. Bleeding is a direct threat to the patient's life and the patient's fate depends on the correct actions of the doctor.

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