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Modern Aspects of The Effectiveness of Providing Resuscitation Care in Acute Blood Loss

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Abstract

Massive blood loss remains one of the most pressing problems for the entire field of critical care medicine. This pathology is complex both in terms of approaches to intensive therapy and in terms of complications that arise during this therapy. Given the available capabilities to ensure conservative and surgical hemostasis, replenish hypovolemia, and ensure adequate oxygen transport, it is extremely important to determine not only the list of therapeutic measures, but also the pace of care. This review is devoted to modern aspects of the effectiveness of resuscitation care for acute blood loss.

Key words: effectiveness, resuscitation, acute blood loss, treatment.

Relevance of the study. Acute blood loss is a pathological process that develops as a result of rapid loss of blood from a damaged vessel(s), leading to tension in the adaptation mechanisms and characterized by hypovolemia, a decrease in the oxygen capacity of the blood and impaired hemocoagulation [V.N. Lapshin, 2017]. Massive blood loss is defined as the loss of one or more circulating blood volume (CBV) within 24 hours, or the loss of 50% of CBV within 3 hours, or bleeding at a rate of more than 150 ml/min. Clinically, such blood loss is manifested by a decrease in systolic blood pressure (BP) less than 90 mmHg. and an increase in heart rate of more than 110 beats per minute [Klein AA, Arnold P, Bingham RM, 2016]. The loss of more than 20% of the circulating blood volume (CBV) causes the development of pathological and compensatory reactions in the body [1, 2]. Macro- and microhemodynamics are disrupted, hypoxia develops, followed by multiple organ failure [3, 4, 5]. A decrease in the level of tissue blood flow below threshold values causes metabolic, biochemical and enzymatic disorders, which leads to death [3, 6]. The

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consequences of acute blood loss depend on its volume, the rate of bleeding, the duration of the post-hemorrhagic period and uncompensated condition, the timing of eliminating the cause of bleeding and other factors (combined effects of damaging factors, hypothermia or overheating of the body, dehydration, etc.), as well as on the individual reaction of the body to decrease in BCC [6, 7]. In case of acute blood loss, compensatory mechanisms are activated, in particular the centralization of blood circulation, aimed at maintaining perfusion of the tissues of the brain, lungs, and heart. Vasospasm of peripheral tissues and organs (skin, muscles, kidneys) as the main mechanism of centralization of blood circulation leads to a decrease in the volume of the peripheral vascular bed and an increase in total peripheral vascular resistance [8]. Maintaining effective perfusion occurs by enhancing the function of the cardiorespiratory system, including the organs of the cardiovascular and respiratory systems. The close interaction of mechanisms regulating blood circulation and respiration is aimed at maintaining the constancy of regulated constants: partial pressure of oxygen, carbon dioxide and blood pH, which ensures compliance with the metabolic needs of the body [9]. The search for effective methods of treating acute blood loss is based on knowledge of emerging disorders, compensation mechanisms and their capabilities. Acute massive blood loss (AMB) during intensive therapy from the point of view of pathophysiology can be considered as a combination of shock, acute coagulopathy and massive transfusion syndrome, which ultimately leads to the formation of multiple organ failure (MOF) in a patient in critical condition [Cannon J.W. , 2018]. Massive blood loss continues to be one of the most pressing problems for the entire field of critical care medicine: for the work of emergency medical teams, for emergency surgery doctors of various profiles, for blood service specialists and for doctors in the departments of anesthesiology, resuscitation and intensive care [Moroz V.V. , Ryzhkov I.A., 2016]. This cohort of patients poses a challenge with respect to the risk of developing coagulopathy against the background of ongoing bleeding, which ultimately leads to the formation of massive transfusion syndrome and the risk of MOF, independent of other factors, during treatment [White N.J., Ward K.R., Pati S., Strandenes G., Cap A.P., 2017]. The causes of AMB leading to shock vary widely: obstetric and gynecological pathology, bleeding from the gastrointestinal tract, perioperative bleeding, aneurysm rupture, erosive bleeding, vascular pathology, etc. [Harrois A, Baudry N, Huet O, 2015]. The adverse effects of AMC are based on a number of pathophysiological mechanisms, the main of which is hemorrhagic shock associated with insufficient oxygen delivery. It has been proven that independent predictors of death are acute hypovolemia, hypofibrinogenemia, acidosis and hypothermia [Chang R., Cardenas J.C., Wade C.E.,

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Holcomb J.B., 2016]. Many modern foreign recommendations on the problem of massive blood loss draw fundamental attention to the fact that this problem is multidisciplinary. For highly professional work in providing care to patients with AMC, close cooperation of the anesthesiology and resuscitation team with surgical specialists, transfusiologists, hemostasiologists and hematologists is necessary. In each medical institution that provides medical care to patients with AMC, AMC protocols must be developed and implemented into clinical practice based on evidence-based standards and with the mandatory implementation of quality control for the implementation of these protocols. Timely initiation of an adequate set of anti-shock measures for AMC has a huge impact on patient survival. Therefore, a CMB protocol of a timely and adequate multidisciplinary approach is the key to improving the treatment outcomes of patients with CMB. The consequences of acute blood loss are hypovolemia, leading to a decrease in cardiac output, a decrease in the oxygen capacity of the blood due to a decrease in the concentration of the oxygen-carrying substrate - hemoglobin, and disturbances in the hemocoagulation system, causing disorders in the microcirculatory sector. As a result of the developed disorders, mixed type hypoxia occurs (circulatory, hemic and tissue), which in case of severe blood loss can cause death. Vasoconstriction, which occurs as a result of activation of the sympathetic component of neuroregulation (adrenaline, norepinephrine), as well as humoral-hormonal factors that implement the body's stress response to mechanical damage (ADH, ACTH, glucocorticoids, hypertensin, etc.), is one of the important adaptation mechanisms with shock and acute blood loss. Vasospasm leads to a decrease in the capacity of the vascular bed and the development of centralization of blood circulation, manifested in a decrease in the volumetric velocity of blood flow in the splanchnic system (kidneys, liver, intestines) and the vessels of the extremities, which subsequently creates conditions for disruption of their function. The blood supply to the heart, brain, lungs and muscles that provide the act of breathing is maintained at a level sufficient to maintain the function of these organs in an "emergency" mode and is the last to be disrupted. Ischemia of a large tissue mass leads to the accumulation of under-oxidized products and activation of anaerobic metabolism. Disruptions occur in the energy supply system, and catabolic processes begin to predominate in most tissues. Metabolic acidosis progresses, which can also be considered an adaptation reaction, since it contributes to the development of more complete utilization of oxygen by tissues. There is a redistribution of fluid, and in particular its movement from the interstitial sector to the vascular sector. This mechanism is realized only if hemorrhage occurs slowly and its volume is small. Continued bleeding leads to decentralization of blood

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circulation, an extreme decrease in cardiac output and oxygen capacity of the blood, severe metabolic disorders and ultimately to circulatory and respiratory failure, which are leading in thanatogenesis in mechanical trauma accompanied by acute blood loss. Shock and acute blood loss are the main pathological processes of the acute period of a traumatic disease, determining its severity. In this regard, the main elements of intensive care at the stage of emergency care at the prehospital stage should be considered measures to stop bleeding, stabilize blood pressure, correct breathing disorders and adequate pain relief. It is clear that the earlier intensive therapy starts, the better the final result. It follows that the role of the prehospital stage in providing adequate emergency care is so significant that in many cases it predetermines the outcome of the injury [Yu. M. Mikhailov, 2017].

At present, the special significance of acute massive blood loss in the pathogenesis of traumatic shock, the most common clinical form of the acute period of a traumatic disease, has certainly been proven. The priority and most important task for the wounded and victims with acute massive blood loss of severe and extremely severe degrees is to eliminate the deficit of blood volume as quickly as possible, restore hemodynamic parameters to a safe level in order to eliminate circulatory failure, which determines the relevance of scientific developments devoted to further improving the tactics of infusion therapy in period of traumatic shock [Kuneev K.P., 2015].

The feasibility of using balanced infusion therapy during the period of traumatic shock in victims with acute massive blood loss of severe and extremely severe degrees is that, compared with “traditional” infusion therapy, it provides more rapid stabilization of hemodynamics and is accompanied by a lesser degree of circulatory inactivity in the second or third day of traumatic illness, helps to improve the delivery and consumption of oxygen, during the first two days it is accompanied by an improvement in microcirculation in the lungs, and also contributes to a lesser severity of hypocoagulation syndrome and metabolic acidosis and is not accompanied by an increase in plasma osmolarity. The use of “low-volume resuscitation” drugs during the period of traumatic shock in victims with extremely severe acute massive blood loss in combination with balanced infusion therapy is accompanied by an increase in systolic blood pressure by an average of 30 mm Hg. Art. for 35–45 minutes, reduces the frequency of forced inotropic support. Optimization of infusion therapy during the period of traumatic shock in victims with acute massive blood loss of severe and extremely severe degrees ensures a more adequate deployment of mechanisms of urgent and long-term compensation, which is accompanied by earlier (on average 6 hours) stabilization of hemodynamics, reducing the duration of the period of maximum likelihood of complications

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developing in on average by 3 days, reducing overall mortality by 4.3%.

The terminal condition caused by acute blood loss, hemorrhagic hypotension, asphyxia, drowning, electrical trauma, is accompanied by severe hemodynamic disorders. Studying the oxygen budget of the body in the recovery period after acute fatal blood loss, it was found that the tissues of a revived body during the first 30 minutes absorb 35–80% more oxygen than before clinical death, which is achieved by increasing the minute volume of blood circulation by 32–80%. [Zhou B., Tian R., 2018]. It is characteristic that the heart is damaged not only during blood loss and clinical death, but also in the first hour after revival. Recirculation and reoxygenation increase the depression of the contractile function of the myocardium, increase the leakage of enzymes from cardiomyocytes and increase the consumption of glycogen per unit of function performed, especially during an unfavorable course of the recovery period. A comprehensive assessment of heart damage in the recovery period after clinical death caused by acute blood loss suggests that functional, metabolic and structural changes in the heart are characterized by a certain phase [Beloborodova N.V., 2019]. The heart is damaged not so much during blood loss and clinical death, but in the first hour after revival due to the influence of a number of pathogenic factors on it: a significant increase in adrenergic effects on the heart, excessive activation of lipid peroxidation processes with the development of signs of membrane destruction, prolonged inhibition of membrane ion pumps, delayed waves of structural and functional changes in mitochondria, immune disorders and the development of delayed-type hypersensitivity to cardiac antigens, absent or weakly expressed during dying and appearing or increasing only with the beginning of recycling and oxygenation of the body [Dolgikh V.T., 2012, Banerjee P., 2018 , Oberhuber D., Frank M., Flammer A. J., 2017].

The problem of damage to internal organs during acute blood loss remains relevant due to the growing risk of injury, vascular damage during diseases and surgical interventions (Moroz V.V., 2011, Kotelnikov. G.P., Mironov S.P. 2008., Thom O. et al., 2010). Of particular importance is the problem of preventing multiple organ failure after the “golden hour” from the moment of blood loss. During this period, due to the centralization of blood circulation in ischemic tissues, disturbances in microcirculation and blood rheology, hypoxic damage to cells progress, which are aggravated in the process of reperfusion from the moment blood flow is restored due to the correction of hypovolemia with infusion solutions (Moroz V.V., 2001, 2015, Dutton R.P., 2007).

Conclusions. Features of violations of the main life-supporting systems of the body during acute massive blood loss of severe and extremely severe degrees in victims are the less effective

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implementation of a compensatory increase in one-time and minute cardiac output, the greater significance of microcirculation disorders in the lungs in the pathogenesis of acute respiratory failure, the greater severity of hypocoagulation syndrome and metabolic acidosis in compared with patients with similar severity of injury with less significant blood loss.

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