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AND THEORETICAL CONFERENCE**

THEORETICAL AND PRACTICAL
SCIENTIFIC ACHIEVEMENTS:
RESEARCH AND RESULTS OF
THEIR IMPLEMENTATION

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scientific achievements:
research and results of
their implementation**

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SECTION 16.

MEDICAL SCIENCES AND PUBLIC HEALTH

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ACUTE RESPIRATORY DISTRESS SYNDROME IN TRAUMA PATIENTS: IS THE RISK OF ITS DEVELOPMENT DETERMINED BY THE NUMBER OF BLOOD TRANSFUSIONS OR THE SEVERITY OF INJURY, BLOOD LOSS AND SHOCK?

Background. Thoracic trauma is one of the serious reasons for the development of acute respiratory distress syndrome (ARDS) [1]. The development of ARDS is associated with a significant aggravation of the patient's condition, the need for long-term respiratory support (often with the provision of a tracheostomy), an increase in the number of complications, the duration of the period of hospitalization of patients in the intensive care unit, in the hospital in general, and an increase in mortality [2]. When determining the risks of the formation of ARDS, many researchers state that one of them is the transfusion of red blood cells and fresh frozen plasma [3, 4]. But the primary indication for the use of red blood cells and fresh frozen plasma in intensive care in trauma patients is grade 2 or more severe blood loss, as classified by the American College of Surgeons. The volume of blood transfusion is usually greater, the greater the volume of blood loss and the severity of the shock. Performing fluid resuscitation in severely injured patients with crystalloids or synthetic colloids along with blood products is common practice. Trauma-induced consumption and dilution of coagulation factors, acidosis, and hypothermia often cause coagulopathy. Early administration of blood components and early control of bleeding reduce the risk of trauma-induced coagulopathy. Hypothermia and dilution coagulopathy are associated with the infusion of large volumes of crystalloid plasma expanders. Therefore, in the early stages of care, critical care specialists pay great attention to the quality of fluid resuscitation for damage control, which aims to maintain acceptable arterial hypotension to control bleeding and prevent severe hemodilution coagulopathy. Massive blood transfusion protocols improve survival of patients with severe injuries. Early recognition that the patient will require a massive blood transfusion limits the use of crystalloids to the extent that had used 20 years ago [5]. The use of colloidal solutions, which is fresh frozen plasma, makes it possible to reduce the volume of

infusion therapy to compensate for hemodynamics in conditions of blood loss and shock. Fresh frozen plasma, which contains a sufficient amount of coagulation factors, can reduce the intensity of bleeding and prevent the development of dilution coagulopathy. Thus, the question naturally arises: how correct is it to say that the risk of developing ARDS has created by the transfusion of blood components, and not by the severity of the damage itself and the volume of blood loss. The purpose of our work was to assess the risks of developing ARDS in patients with thoracic trauma and hemopneumothorax during fluid resuscitation with the help of plasma substitutes and with the active use of blood components in the first 12 hours after the patient received injury.

Materials and methods. We took part in the examination and implementation of intensive care measures in 200 patients with thoracic trauma in the polytrauma department of a multidisciplinary city emergency hospital. Thoracic injury in these patients was the main component of the associated injury. Disorders of vital functions in these patients, namely, acute respiratory failure syndrome, hemodynamic disorders, and functions of consciousness against the background of combined systemic hypoxia primarily determined the severity of their condition. All these patients had clinical signs of chest contusion with the formation of hemopneumothorax. Specialists from the polytrauma department performed drainage of the pleural cavity from a lower approach in the operating room in all these patients. Other injuries, the presence of which determined the diagnosis of polytrauma, were traumatic brain injury, fractures of the limbs, and pelvic bones. We did not include patients with bleeding from the abdominal organs in the comparison groups. Our studies included: general radiography of the chest (in all patients), spiral computed tomography of the body (only 14% of patients), non-invasive monitoring of heart rate and pulse rate, blood pressure, oxygen saturation of peripheral arterial blood, monitoring and registration of electrocardiogram leads, invasive monitoring of central venous pressure. We provided the following laboratory studies: determination of the blood hemoglobin concentration, hematocrit, the number of blood cells, the concentration of total protein in the blood plasma, glycemia level, the concentration of fibrinogen in the blood plasma, clotting time & determination of the concentration of cardiac troponin I. In the intensive care unit (ICU) we monitored in our patients, the value of central hemodynamics, ECG leads, peripheral, arterial blood oxygen saturation, pressure level in the respiratory tract and the value of static pulmonary compliance during mechanical pulmonary ventilation. In all our patients' we performed invasive mechanical pulmonary ventilation controlled by airway pressure (PCV). In the first 12 hours from the patients' admission to the ICU, we provided pulmonary ventilation under conditions of sedation with ketamine and benzodiazepines, and general muscle relaxation (atracurium). We assessed the severity of the injury using the AIS (Abbreviated Injury Scale) and ISS (Injury Severity Score) scales. We assessed the severity of thoracic trauma using the TTSS (Thoracic Trauma Severity Score) scale, and the severity of lung damage and respiratory failure using the Murray scale, which has also used to determine the severity of the condition of patients with ARDS [6]. The presence of ARDS in patients was determined according to the criteria approved at the consensus conference on the problems of polytrauma in Berlin in 2012 [7].

All patients in the operating room and then in the ICU received tranexamic acid (1000 mg twice) and etamsylate (1000 mg twice). Early antibacterial therapy in all cases included the administration of 4000 mg of ceftriaxone per day and 1500 mg of metronidazole. For pain relief we used morphine hydrochloride, metamizole sodium and dexketoprofen. We formed two study groups. Patients of the first group, numbering 100, received fluid resuscitation mainly due to crystalloid and colloid plasma substitutes. These patients received no more than 2 units of red blood cells and 2 units of fresh frozen plasma. The main plasma substitutes were normal saline, Ringer's solution, and colloidal solutions based on 4% modified gelatin. Patients of the second group, also numbering 100, received at least 3 units of red blood cells and 3 units of fresh frozen plasma during surgery and in the first 12 hours from admission to the hospital. There were cases of massive blood transfusion, the need for which had determined by the volume of blood loss. In

our study, we compared the incidence of GRDS in patients of 2 groups, taking into account the severity of injury, blood loss and the need for blood transfusions. We also conducted a targeted search for current reports on the problem on specialized websites for medical professionals on the Internet.

The Results & Discussion. During bleeding, the first of all, the body of a trauma patient loses intravascular fluid. In urban conditions, the time it takes to transport a patient from the scene of the incident to the clinic rarely exceeds 40 minutes. Specialists from the hospital's emergency department manage to provide the required minimum of tests and provide assistance in the next 20 minutes. At this time, the rate of infusion therapy, as a rule, is not high. Within one hour, fluid from the interstitial compartment does not have time to reduce fully the deficiency of intravascular fluid. Therefore, despite serious injury and blood loss, hemoconcentration indicators, namely hemoglobin and hematocrit, remain high. These indicators cannot immediately be used to determine the severity of blood loss and the severity of shock. Patients almost constantly admitted to our operating room with hemoglobin concentrations in the blood within the range of 12-15 g/dL. Yet, all of them suffer severe trauma, blood loss and shock. Therefore, we assess the severity of shock in trauma with blood loss not by the value of hemoglobin and hematocrit, which were obtained in the emergency department. We assess the severity of shock based on blood pressure readings, peripheral arterial blood oxygen saturation and, if possible, full monitoring of this indicator. Modern equipment allows recording the perfusion index value from 0 to 20%. We consider a perfusion index value close to 1.5% or less to be a strong sign of shock. The more severe the shock, and the less peripheral tissue perfusion, the lower the perfusion index, and the less stable this indicator is. The rate of diuresis is a very important indicator of hemodynamic efficiency, but it has no immediate diagnostic value. Blood transfusion in conditions of urgent surgery is not the first method of hemodynamic correction. Improvement in cardiac output and blood pressure must be achieved through high rates of intravenous administration of plasma expanders under the control of arterial and central venous pressure. Oxygen therapy using 100% oxygen also improves hemodynamics. Visually on the monitor, the improvement in hemodynamic changes has clearly been shown by the perfusion index and the shape of the photoplethysmogram. Of course, we monitor the heart rate and the correspondence of the frequency of the QRS complexes of the electrocardiogram to the number of plethysmographic waves. In conditions of severe shock, restoration of the normal rate of diuresis always lags behind the improvement in blood pressure and peripheral tissue perfusion. After hemodynamic parameters improve under the influence of plasma expander infusion, we must repeat the value of hemoconcentration parameters. It is in order to better understand the volume of blood loss. Having in dynamics two indicators of hemoglobin and hematocrit, we can more accurately estimate the volume of blood loss.

If we consider the mechanisms of formation of ARDS in polytrauma, then we believe that the main ones are the following two scenarios. In any case, these two mechanisms were present in the overwhelming majority of cases of our observations.

The first mechanism primarily triggers the development of ARDS through significant blood loss, shock and prolonged arterial hypotension. As a rule, patients in such cases, due to massive blood loss, receive intravenously, in addition to blood components, large volumes of plasma replacement solutions. Lung damage occurs against a background of prolonged arterial hypotension, hypoperfusion and significant capillary leakage with the formation of pulmonary interstitial edema. The process includes a massive release of inflammatory mediators.

The second mechanism is associated with severe contusion of the chest and lungs, when areas of pulmonary contusion quickly form. In this case, there are foci of hemorrhage in the lungs. Through a rupture of the lung tissue, we often detect the presence of hemopneumothorax in such patients. Compression of the lung by blood and gas in the pleural cavity leads to a sharp increase in venous-arterial shunting in the pulmonary circulation. This causes severe systemic arterial hypoxemia. Against the background of a decrease in oxygen tension in the blood, capillary leakage

of fluid from the vessels into the interstitium activated. At the same time, the motility of the myocardium also decreases; due to arterial hypoxemia, cardiac output and blood pressure decrease and shock develops. Lung damage facilitated by a large accumulation of blood in the pleural space, a delay in drainage of the pleural cavity, and ineffective drainage, which then becomes the cause of fibrothorax, pleural empyema, polysegmental pneumonia and pulmonary sepsis.

To stop prolonged pulmonary hemorrhage, numerous transfusions of whole blood and its components are necessary. In this cases for quickly stop the bleeding it is necessary to limit the administration of plasma replacement solutions. Whole blood and blood components should form the bulk of fluid therapy. The use of artificial plasma expanders should be limited, as this poses a high risk of developing hemodilution coagulopathy and continued bleeding. That is why in such cases, we prefer to use antihypertensive infusion therapy with a predominance of blood components. In all cases where we see a high risk of developing ARDS, we use long-term forced artificial ventilation against the background of ketamine anesthesia with the use of muscle relaxants.

Considering the results of the development of ARDS in our 2 study groups, we did not find that a greater number of blood transfusions determined a higher incidence of ARDS in the first group of patients. Hemotransfusion is a means of removing the patient from a state of shock due to blood loss due to trauma, and through improving hemodynamics, oxygen transport and increasing coagulation potential, it helps stop bleeding and reduces pulmonary damage. Blood transfusion reduces the patient's stay in a state of shock, as we had convinced of by monitoring the perfusion index. Active early use of blood components also reduced the need for epinephrine use. The severity of the injury contributed to the development of ARDS. We found that the risk of developing ARDS increased significantly when the severity of polytrauma was assessed on the ISS scale of 32 points or higher. The development of ARDS is associated with severe prolonged arterial hypotension, the need for massive use of epinephrine, and a prolonged decrease in arterial blood oxygen saturation against the background of hemopneumothorax.

Afshar's study, which included 27,385 trauma patients, showed that the ISS score most accurately assessed the risk of developing ARDS in trauma. The prognostic value of other scales also had assesse: Glasgow coma score, Severity Characterization of Trauma, Revised Trauma Score, thorax Abbreviated Injury Score. The receiver operating characteristics for ISS had the best discrimination and had an area under the curve of 0.88 (95% CI 0.87–0.89). Glasgow coma score (0.71, 95% CI 0.70–0.73), A Severity Characterization of Trauma (0.86, 95% CI 0.85–0.87), Revised Trauma Score (0.71, 95% CI 0.70–0.72) and thorax Abbreviated Injury Score (0.73, 95% CI 0.72–0.74) performed worse ($p < 0.001$) and Trauma and Injury Severity Score (0.88, 95% CI 0.87–0.88) performed equivocally ($p = 0.51$) in comparison to ISS. Using a cutoff point ISS greater than or equal to 16, sensitivity and specificity were 84.9% (95% CI 83.0%–86.6%) and 75.6% (95% CI 75.1%–76.2%), respectively [8]. In our work, we found a higher prognostic assessment of the high risk of developing ARDS according to the ISS scale for the reason that Afshar's study did not consider patients with a severity of chest injury according to the AIS scale of 3 points or higher. Thus, we found that the risk of developing ARDS was significantly justified by the severity of injury and shock. The severity of thoracic trauma significantly contributed to the development of ARDS. All patients with severe thoracic trauma were on mechanical ventilation for a long time. The strategy of targeted long-term invasive respiratory support immediately after urgent surgery significantly reduced the risk of developing ARDS or reduced its severity. Prolonged invasive respiratory support immediately after urgent surgery improved the recovery of patients from shock, contributed to more rapid expansion of damaged lungs, reduced blood loss through pleural drainage, and improved oxygenation. The administration of blood components to patients with chest trauma helped to reduce the intensity of loss of bloody fluid from the pleural cavity and improved central and peripheral hemodynamics in these trauma patients. The target indicator of blood hemoglobin concentration when correcting the oxygen capacity of the blood in our patients was 10 g/dL.

Conclusion. In our work, we did not find that the number of blood transfusions is the factor that determines the high risk of developing ARDS in patients with polytrauma. Blood transfusions are a mandatory component of intensive care for severe trauma with blood loss and hemorrhagic shock. Indeed, the volume of blood loss determines the need for blood transfusion. Yet it is incorrect to say that therapy with blood components increases the risk of ARDS. The severity of injury, blood loss, and shock determine the increased risk of ARDS. Moreover, the presence of severe thoracic trauma most seriously affects the risk of developing ARDS blood transfusions help speed up the recovery of patients from shock, reduce the rate of blood loss and compression of lung tissue in the presence of lung ruptures and an increase in the volume of hemopneumothorax. Early use of whole blood and its components can improve the condition of a patient with severe trauma and more quickly bring the patient out of shock.

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