Ministry of Health of Belarus
Gomel State Medical University

Department of Orthopedic, Trauma and military field surgery
with the course of Anesthesiology and Critical Care Medicine

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Topic on the intensive care of diseases accompanied by impaired circulation

Educational and methodical development for practical training teachers for 4th year students of medical faculty

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Methodical development is designed for self-study. It provides:

1. Background.

2. The purpose of training (skills and knowledge).

3. Basic sections.

4. Suggested Reading.

5. Questions for self-preparation.

6. Topics of educational and research work of students.

7. Teaching tools for organization of independent work of students

8. Training Material

9. Self-study.

10. Clinical problems and test control.

Relevance of the topic

Acute circulatory disorders - shock - is found in both the surgical and in therapeutic practice. The cause may be a variety of factors: extensive injuries lead to traumatic shock, in total injuries and damage in various pathologies of the internal organs, one of the foremost is the heavy loss of blood, accompanied by hemorrhagic shock, and in the pathogenesis of the acute period of burn disease leading place belongs to the burn shock; to the number of severe allergic reactions include anaphylactic shock, cardiovascular disease may be associated with cardiogenic shock, and so on. Despite the variety of reasons, shocks of various etiologies have common traits and characteristics of the pathogenesis, which determine the nature of intensive care. Knowledge and understanding of the basic pathophysiological mechanisms of disturbances in such a complex system as the circulatory system, and methods of diagnosis helps the doctor in an emergency, is a serious health hazard, and sometimes life of patients, to quickly determine the nature and amount of pathogenetic events aimed at restoring adequate circulation.

Purpose of the lesson

To teach students the principles of pathogenetically substantiated intensive therapy of acute circulatory disorders.

The student should know:

• definition and classification, general principles of intensive therapy of diseases associated with acute cardiovascular disorders

• clinical and physiological effects of hypovolemia

• classification of stages of hemorrhagic and traumatic shock, the principles of intensive care, diagnosis, prevention and treatment of DIC.

• intensive treatment of hemorrhagic and traumatic shock.

• intensive care of anaphylactic, septic, cardiogenic shock, sequence of actions

• clinical pharmacology of anticoagulants, agonists, analgesics.

The student should be able to:

• Identify indications for the study parameters of central hemodynamics.

• Indicators of central hemodynamics to determine the type of hemodynamics and pathogenetically substantiated treatment of blood circulation disorders.

• be able to determine central venous pressure and interpret his performance in intensive care.

• first aid and determines treatment for different types of shocks

Sections studied before and needed for the session

• basic pharmacology of drugs

• physiology

• physiopathology

• internal medicine

• surgical disease

• obstetrics and gynecology

Recommended Reading

Textbooks on pharmacology, normal and abnormal physiology, surgery, obstetrics and gynecology, internal medicine for medical students.

Suggested Reading on lessons

Main Reading

1. Valley of OA Anaesthesia and Intensive Care / O.A.Dolina / / M., 1998. - 410-419 p.

Further Reading

1. Morgan Jr.., Edward J. Clinical Anesthesiology / G. Edward Morgan, Jr., Michael S. Magid / / M., 2004. - Vol.1-3. – 375 p.

2. Marini, John J. critical care medicine / John J. Marini, Arthur P. Wheeler. - M., 2002. – 992p.

3. Sumin, SA Emergency conditions. / SA Sumin. - M., 2006. – 799p.

4. Malyshev, V. Intensive care. Resuscitation. First aid. / VD Malyshev. - M.: Medicine, 2000. – 464 p.

5. Online Resources

Questions for self-study

Questions on basic knowledge

1. Stroke volume, heart rate, cardiac output, and total peripheral vascular resistance, blood volume, blood viscosity.

2. Pathogenesis of preload and water-salt metabolism as causes and effects of congestive heart failure

3. Communication pathogenesis of congestive heart failure and symptoms of left-and right heart failure

4. Heart failure with a high cardiac output

5. Pathogenesis of acute left ventricular failure as a cause of cardiogenic pulmonary edema

Questions about the topic studied

1. Central hemodynamic parameters (stroke volume, heart rate, cardiac output, and total peripheral vascular resistance, blood volume, blood viscosity).

2. Shock. Definition. Classification (cardiogenic, vasogenic, hypovolemic).

3. Principles of diagnosis and intensive therapy hypovolemic shocks: bleeding, burns, traumatic.

4. Principles of diagnosis and intensive therapy vasogenic shock. Anaphylactic shock.

5. Principles of diagnosis and intensive therapy of cardiogenic shock.

6. Principles of diagnosis and intensive therapy of septic shock.

Topics of educational and research work of students

1. Pathogenesis and intensive therapy of hemorrhagic shock in obstetrics

2. Corticosteroids in the intensive therapy of critical states.

Teaching tools for organization of independent work of students

1. Computer database.

2. Objectives, test control.

3. Thematic sick.

4. Patient records.

5. Bank jobs for self-study.

Training Material

OPTIONS OF CENTRAL HEMODYNAMICS

The clinic is necessary to assess the state of the circulatory system and the usefulness of the blood flow, not only by measuring heart rate (HR), blood pressure (BP) (systolic blood pressure (SBP), diastolic pressure (DBP)), but also on variables such as stroke volume (SV), cardiac output (CO), and total peripheral vascular resistance (SVR), and other indicators.

Typically, the measurement of blood pressure does not provide a complete picture of the nature of blood circulation disorders, although carries information about the dynamics of the pathological process and to some extent the effect of therapeutic interventions.

The value of blood pressure depends on the SV, blood volume, SVR, elasticity of blood vessels and blood viscosity. The most dynamic of these indicators is SVR. As a rule, a decrease of CO and blood volume it rises and increases in blood pressure may occur, but in such a way by increasing SVR deteriorating tissue oxygenation, increased blood viscosity, blood flow slows down, leading to the effects described in the section on the rheological properties of blood. On the contrary, with a slight reduction in blood pressure due to a decrease SVR, blood circulation, and, consequently, their oxygenation improved.

Most informative indicators of cardiovascular values SV and CO, which are judged on the work of the heart (in particular, its contractility) and the blood supply to the organs.

To determine these quantities using a number of methods.

Starr's method: SV = 100 + 0.5 PBP - 0.6 DBP - 0.6 A (A - age in years, PBP pulse pressure, DBP - diastolic pressure).

Fick method: CO = VO2 : (A-B), where VO2 - oxygen consumption per minute (calculated on spirogram), AB - arteriovenous oxygen difference (determined by laboratory).

There are other formulas for the CO. CO can be expected as well, knowing the SV: CO = SV × HR 1000 (in l / min.)

The normal value of the CO - 5-7l/min

SVR reflects the total resistance of the vascular system and the blood flow is calculated as Poiseuille:

SVR = FBC × 1333 × 60: CO

SVR increases with compensated hemorrhage, myocardial infarction, hypertension, and decreased during intoxication, collapse, decompensated blood loss. Normally 900-1500 din/s/cm^5.

Increase the CO - the reaction of the cardiovascular system to the increased release of catecholamines in the blood (if any stressful situation). Even with varying degrees of hypovolemia due to a compensatory increase in heart rate can be maintained CO to some extent at normal levels, but will suffer from the peripheral circulation. CO reduction observed in patients with decompensated hypovolemia, severe heart failure, caused by, for example, acute myocardial infarction, or cardiac arrhythmias, in shock.

For the purpose of leveling the anthropometric factors in the evaluation of data CO and SVR their lead to one surface of the body (1 m^2) and calculate a cardiac index (CI), which reflects the state of the most peripheral regions of the arterial bed. CI is normally 2.5-4 l/min/m2.

CI = CO (L / min): S (sq. m.), C - the surface of the body

The surface of the body can be calculated by the formula Breitman:

S = 0,0087 × (H + W) - 0.26, where S is the area of the body in square meters, H - height in cm, W - body weight in kg.

The main indicator of the consistency of the integral parts of the cardiovascular system and its functional integrity is the mean arterial pressure (MAP), which is expressed as the product of the CO and total peripheral vascular resistance (SVR): MAP = CO × SVR.

Clinically MAP is calculated as follows:

MAP = DBP + (SBP - DBP) / 3. Normally SBP = 60-100 mmHg

Since hypovolemia can be represented as a decrease in blood volume (BV). Under normal circumstances, the BV - the value relatively stable at 7% in men, and for women 6.5% of

body weight. Most often expressed in ml/kg of patient weight. In healthy adult men BV - on average equal to 70 mL/kg in healthy women BV, on average, equal to 65 ml/kg.

A necessary criterion for judging the degree of hypovolemia is *central venous pressure* (CVP), which is practically the pressure in the right atrium. The zero point of the phlebotonometer, called the Waldman apparatus, should be at the level of the right atrium, which corresponds to the point of intersection of the lower edge of the pectoralis major muscle with the V rib. The readings of the device are recorded after stabilization of the liquid level in the glass tube. When conducting mechanical ventilation during the measurement of CVP, the respirator is turned off.

Normal figure of CVP may be in the range of 20 - 120 mm Hg. However, in clinical practice, it is often important to not only measure the absolute value of CVP as this quantity in the dynamics of intensive care. Often low CVP do not conform with the BV of the vascular bed. Severe cases of low CVP are sequestered blood, and therefore the CVP can be a criterion hypovolemia. SV during low CVP decreased, high CVP may be due to hypervolemia (e.g., wrong infusion therapy or heart failure). Both options are threatening patient development of pulmonary edema. CVP does not always determine the "venous return to the heart," but in many cases, the changes are the same. Since the driving force of venous return is a pressure gradient between the right atrium and venules. If this value increases from zero, its growth will be accompanied by an increase in venous return. But from the point where the pressure in the right atrium will be quite high as compared to the peripheral venous pressure, venous return will decrease.

Based on this important indicator of the usefulness of the CO is determined in clinical hemodynamic types (in % respect to the proper CO), which is an important predictor for intensive care:

hyperdynamic type - with the CO over 110%

normodynamic type - at the CO within 100 ± 10%

hypodynamic type - if the CO is below 90%.

Of course, the most favorable types of hemodynamics are normal and hyperdynamic types.

Rheological properties of blood. Blood - a suspension of cells and particles suspended in the plasma colloids. This is typical of non-Newtonian fluid whose viscosity, unlike Newton, in different parts of the circulatory system differs hundreds of times, depending on changes in blood flow.

For the viscosity of the blood counts protein composition of the plasma. Thus, albumin and the ability to reduce the viscosity of cell aggregation, whereas globulins are opposed.

Particularly active in the high viscosity and aggregation propensity of cells to fibrinogen, the level of which varies in any stress. Hyperlipidemia and hypercholesterolemia also contributes to a violation of the rheological properties of blood.

Hematocrit - one of the important parameters associated with the viscosity of blood. The higher the hematocrit, the greater the viscosity of the blood and worse its rheological properties. Hemorrhage, hemodilution, and conversely, loss of plasma and dehydration significantly affect the rheological properties of blood. So, for example, controlled hemodilution is an important means of preventing the rheological disorders in surgery. When hypothermia blood viscosity increase by 1.5 times compared to that at 37 ° C, but if you lower the hematocrit from 40% to 20%, with this change in temperature viscosity does not change. Hypercapnia increases the viscosity of the blood, so it is in the venous blood is less than the arterial. With a decrease in blood pH of 0.5 (with a high hematocrit), blood viscosity is tripled.

Disorders of the rheological properties of blood. The basic phenomenon of rheological blood disorders - the aggregation of red blood cells, which coincides with an increase in viscosity. The slower the flow of blood, the more likely the development of this phenomenon. So-called false aggregates ("coin posts ") are physiological in nature and fall into the healthy cells as conditions change. True aggregates arising from disease, do not break down, causing the phenomenon of sludge. The cells in the protein aggregates are covered film, bonding them in irregular clumps.

The main factor causing aggregation and sludge, is hemodynamic instability - slow blood flow that occurs in all critical conditions. It is often combined hemodynamic disturbances and hyperglobulinemia under such severe conditions as peritonitis, acute intestinal obstruction, acute pancreatitis, prolonged compression syndrome, burns. Enhance the aggregation state of the fat, amniotic and air embolism, damage to red blood cells in an artificial blood circulation, hemolysis, septic shock, and so on, that is all the critical state.

We can say that the main reason for the violation of blood flow in capillarone is a change in the rheological properties of blood, which in turn depend mainly on the speed of blood flow. Therefore, impaired blood flow in all critical conditions has 4 stages: breach of the rheological properties of blood, blood sequestration, hypovolemia, generalized loss of microcirculation and metabolism.

And in thanatogenesis terminal state is not important, what came first: reduction of BV due to the blood loss or decrease in cardiac output due to right ventricular failure (acute myocardial infarction). When the result of a vicious cycle of hemodynamic is basically the same.

SHOCK

Shock - *a severe emerging circulatory failure of the critical tissue perfusion disorder, which leads to a deficiency of oxygen in tissues, cell damage and the development of multiple organ failure.*

Despite the fact that the shock triggers may be different, common to all forms of shock is a critical decrease in perfusion in the tissue, resulting in impaired function of the cells and, in advanced cases, to death. The most important pathophysiological link shock - disorder of capillary circulation, leading to tissue hypoxia, acidosis, and ultimately - to permanent status.

It should be emphasized that the CO can not be an indicator of tissue perfusion, this is confirmed by its high numbers in septic shock. Shock can be with high or low CO. The latter form is called hyperkinetic shock.

Clinical criteria of shock:

• *symptoms of critical disturbance of capillary circulation* of affected organs (pale, cyanotic, marbled, cold, wet skin, symptom of a "pale spot", impaired function of lungs and central nervous system, decreased urine output to 0.5 ml / min or less, the difference between cutaneous and rectal temperatures above 4 ° C, the presence of metabolic acidosis and a decrease in arteriovenous oxygen differences are a sign that the latter is not absorbed by the tissues)

• *symptoms of the impaired central circulation* (small and rapid pulse, sometimes bradycardia, decreased systolic and pulse pressure)

An important mechanism of shock:

1. a sharp decline in the BV

2. decrease in cardiac performance

3. violation of the regulation of vascular

These reasons can cause profound hypotension. Depending on the main trigger of the pathogenesis and characteristics distinguish the following ***clinical forms of shock****:*

• hypovolemic shocks (hemorrhagic, traumatic burns, dehydration)

• cardiogenic shocks (associated with reduced contractility, arrhythmia, occlusion of large vessels)

• vasodilation shocks (anaphylactic, neurogenic (spinal injury))

• septic shock

Hypovolemic shock

Hypovolemic shock due to acute loss of blood, plasma or body fluids. Hypovolemia (decreased blood volume) leads to a decrease in venous return and cardiac filling pressure decrease. This in turn leads to a decrease in stroke volume and a fall in blood pressure. Due to stimulation of the sympathoadrenal system increases heart rate (HR) and there is vasoconstriction (increased peripheral resistance), which allows to maintain central hemodynamics and blood circulation is centralized. In this case, a significant difference in blood flow centralization (best blood supply to the heart, brain and lungs) is the prevalence of α-adrenergic receptors in blood vessels innervated by n. splanchnicus, and vessels of the kidneys, muscles and skin. Such a reaction of the body to be warranted, but if not corrected hypovolemia due to insufficient tissue perfusion the picture of shock.

For hypovolemic shock, therefore, characterized by the decrease of BV, reduce cardiac filling pressure and cardiac output, lower blood pressure and increased peripheral resistance.

Cardiogenic shock

The most common cause of cardiogenic shock is acute myocardial infarction, myocarditis, and less toxic damage of the myocardium. In violation of the pumping function of the heart, arrhythmias, and other acute causes of a drop in the effectiveness of heart contractions, a decrease in stroke volume occurs. As a consequence, there is decrease in blood pressure at the same time increases the CSN because of the inefficiency of his work.

The result is again stimulated sympathoadrenal system, increases heart rate and peripheral resistance.

Changes in principle similar to those of hypovolemic shock, and with them are forms of hypodynamic shock. Pathogenetic difference lies only in the value of the cardiac filling pressure: with hypovolemic shock is reduced and in cardiogenic - increased.

Anaphylactic shock

An anaphylactic reaction is a particular expression of hypersensitivity to foreign substances. At the heart of anaphylactic shock is a dramatic reduction of vascular tone under the influence of histamine and other neurotransmitter substances.

Capacitance due to the expansion of the vascular system (veins) develops a relative decline of BV: there is a discrepancy between the volume of the vascular bed and BV. Hypovolemia leads to a reduction of blood flow back to the heart and reduce the BV. This leads to a decrease in the SV and BP. A direct violation of the myocardial contractility also contributes to a decrease in cardiac performance. Characteristic for anaphylactic shock is the absence of a pronounced reaction of the sympathoadrenal system, this largely explains the progressive clinical development of anaphylactic shock.

Septic shock

In septic shock, primary disorders concern the periphery of the blood circulation. Under the influence of bacterial toxins, short arteriovenous shunts open, through which blood rushes, bypassing the capillary network, from the arterial to the venous.

In this case, a situation arises when, with a decrease in blood flow into the capillary bed, the blood flow at the periphery is high and the SVR is reduced. Correspondingly, blood pressure decreases, and SV and heart rate increase compensatory. This is the so-called hyperdynamic circulation reaction in septic shock. A decrease in blood pressure and SVR occurs with normal or increased SV. With further development, the hyperdynamic form becomes hypodynamic, which worsens the prognosis.

Table. Comparative characteristics of hemodynamic disorders in various types of shock.

Type of shock	CO	CVP	BP	SVR
Hypovolemic	↓	↓	↓	↑
Cardiogenic	↓	↑	↓	↑
Septic	↑	↑	↓	↓
Anaphylactic	↓	↓	↓	↓

Despite the differences in the pathogenesis of submitted forms of shock, ending their development is the *reduction of capillary blood flow*. Consequently, the delivery of oxygen and energy substrates, as well as removal of end products of metabolism are insufficient. Hypoxia, the nature of metabolism changes from aerobic toward anaerobic. Less pyruvate is included in the Krebs cycle and goes into lactate that, along with hypoxia leads to tissue *metabolic acidosis*.

Under the influence of acidosis, there are two phenomena that lead to further deterioration of the microcirculation in shock:

1. *shock specific vasomotion*: precapillaries expand, while postcapillaries are still narrowed. Blood rushes into the capillaries, and the outflow is impaired. The intracapillary pressure rises, the plasma goes into interstitium, which leads to a further decrease in bcc, and to a violation of the rheological properties of blood

2. *violation of the rheological properties of blood*: there is an aggregation of cells in the capillaries. Red blood cells stick together in columns of coins, clumps of platelets form. As a result of increasing the viscosity of the blood, an almost insurmountable resistance to blood flow

is created, capillary microthrombi are formed, and disseminating intravascular coagulation develops.

So the center of gravity of changes in progressive shock is increasingly moving from macrocirculation to microcirculation.

Dysfunction of cells, their death due to impaired microcirculation during shock can affect all cells of the body, but certain organs are especially sensitive to circulatory shock. Such organs are called shock.

By *shocking human organs* include primarily the lungs and kidneys, and in the second place - the liver. We should distinguish between changes of these organs in shock (in shock lung, kidney during shock, liver in shock), which terminates at the output of the patient from shock and organ failure associated with the destruction of the tissue structure, when after leaving the shock is a complete failure or loss of organ function (shock lung, kidney shock, shock liver).

Lung during shock is characterized by impaired absorption of oxygen, and is recognized on an arterial hypoxia. If developing shock lung (respiratory distress syndrome), after elimination of shock rapidly progressive severe respiratory distress, decreased partial pressure of oxygen in arterial blood, decreased elasticity of the lung, and it becomes more and more intractable. Begins to increase the partial pressure of carbon dioxide so much that it becomes necessary to an increasing amount of breath. In this advanced stage of shock syndrome, shock lung, apparently, not regress: the patient dies of arterial hypoxia.

Kidney in shock characterized by a sharp restriction of blood circulation and reducing the number of glomerular filtrate, concentrating defect, and reduction in the number of urine. If these disorders resolve after shock did not undergo immediate regression, we further reduced urine output and increases the amount of slag substances - develops shock kidney, the main manifestation of which is the clinical picture of acute renal failure.

The liver is the central organ of metabolism and plays an important role during the shock. Development of shock liver may be suspected when liver enzymes increase and after knocking shock.

Hypovolemic shocks

Feature of hypovolemia in hemorrhagic shock is to reduce the oxygen capacity of the blood by increasing blood loss. At the start of the pathogenesis of traumatic shock plays an important role pain factor, the decay products of tissue toxicity. Traumatic shock does not always correlate with the volume of blood loss.

Clinic and diagnostics

Diagnosis is based on an evaluation of clinical and laboratory findings. In acute blood loss is essential to determine its value. To do this, use one of the existing methods, which are divided into 3 groups: clinical, empirical and laboratory studies.

Clinical methods allow us to estimate the amount of blood loss on the basis of clinical symptoms and hemodynamic variables.

The severity of blood loss is determined by its type, speed of development, the volume of blood lost, the degree of hypovolemia and the possibility of shock. The clinical picture is determined by the volume of blood loss and the stage of shock. Due to the fact that the clinical signs of blood loss depend on the degree of discrepancy between the delivery and consumption of O_2 by body tissues, factors contributing to the development of shock

• premorbid background, violating basic metabolism;

• hypotrophic syndrome;

• children's age;

• elderly age.

Clinically isolated three stages of shock:

Stage 1 - is characterized by pale mucous membranes and skin, psychomotor agitation, cold extremities, normal or mildly elevated blood pressure, increased heart rate and breathing, increased central venous pressure, normal urine output.

Stage 2 - is characterized by lethargy, pale gray skin, covered with a cold clammy sweat, thirst, shortness of breath, decreased blood pressure and central venous pressure, tachycardia, hypothermia, oliguria.

Stage 3 - characterized by weakness, passing into coma, pale and sallow hue and marble pattern skin, progressive respiratory failure, hypotension, tachycardia, anuria.

Assessment of the levels of blood pressure and pulse rate also makes it possible to estimate the deficit BV. The ratio of the pulse rate to the level of systolic blood pressure allow calculate shock index Algovera.

Table. The dependence of blood volume deficiency on the shock index of Algover

Deficiency of blood volume in% of the proper blood volume	Shock index
0	0,54
10	0,8
20	0,9-1,2
30	1,3-1,4

40-50	≥1,5

Test of *filling the capillaries*, or a symptom of the "white spots" - to evaluate the capillary perfusion. Performed by pressing the thumb nail, skin, forehead, earlobe. Normally, the color returns after 2 seconds, with a positive sample, after 3 seconds or more.

The *central venous pressure* - a measure of right ventricular filling pressures, reflects its pumping function. A catheter for measuring central venous pressure is introduced through the subclavian or jugular vein so that its tip was in the right atrium. Normally CVP ranges from 6 to 12 cm of water column. CVP reduction below these limits indicates hypovolemia. With a deficit of BV in 1 liter CVP decreased by 7 cm of water column.

Hourly *urine output* - an indicator to maintain the level of filtration and tubular reabsorption as a characteristic lack of (liquidation) circulation. Normally hourly urine output is 0.5-1 ml / kg of patient weight.

When empirical assessment methods for bleeding blood loss take its average statistical value characteristic of a particular type of damage. They are more useful in traumatology. However, surgical interventions in different areas of the body are also accompanied by some loss of blood. Laboratory methods include determination of Ht, Hb, density or viscosity of the blood.

Laboratory methods are divided into:

• design (use of mathematical formulas);

• hardware;

• indicator.

From the calculation of the most common methods is the formula Moore: blood loss = BV_p x (Ht_p – Ht_a)/Ht_p, where: BV_p – proper blood volume, Ht_p - proper hematocrit of 45% for males and 42% for women, Ht_a - actual hematocrit of the patient. This formula can be used instead of hematocrit hemoglobin, taking as its required level of 150 g / L.

General principles of treatment

1. Stop bleeding, control of pain.

2. Ensuring adequate gas exchange.

3. Replacement of the BV deficit

4. Treatment and prevention of organ failure of multiple organ failure:

• treatment of heart failure;

• prevention of renal failure;

• correction of metabolic shock;

• stabilization of the metabolic processes in the cell;

• treatment and prevention of DIC.

5. Early prevention of infection

To constant bleeding with external bleeding - pressing of the vessel, compressive bandage, tourniquet, ligature or clamp on the bleeding vessel. With internal bleeding - emergency surgery, the concurrent combined treatment for the removal of the patient from shock.

The fight against pain and protection from mental stress performed by intravenous administration of narcotic and non-narcotic analgesics, tranquilizers. The initial dose of narcotic analgesics in patients with the most severe reduction should be lowered by 50% because of possible respiratory depression, nausea and vomiting that occur during intravenous administration of these drugs. In case of injury - immobilization of a limb, novocaine blockades.

Ensuring adequate gas exchange is aimed at acquiring O_2 and CO_2 removal. Events are both preventive and therapeutic value. All patients are shown to ensure airway, preventing aspiration of gastric contents, prophylactic administration through nasal catheter at least 4 liters / min O_2. If necessary, tracheal intubation and transfer to mechanical ventilation with positive pressure at the end of exhalation (PEEP = 5 cm of water column) are indicated (ror preventing alveolar wears off at the end of exhalation), if necessary the rate increase, but it should be remembered that a PEEP of 10-15 cm of water column leads to a decrease in cardiac output.

Replacement of the BV deficit is a priority treatment of hypovolemic shock.

To fill the BV deficit is necessary:

1. Improved Trendelenburg position to increase venous return.

2. Peripheral infusion through 2-3 peripheral or 1-2 central veins.

The rate of replacement is determined by the value of blood pressure and the cause of blood loss. As a rule, the initial rate of replacement is a jet or a quick drop. After stabilization of blood pressure at a safe level, infusion is carried out by drip.

Treatment of organ failure

Treatment of heart failure. If the victim before the accident was healthy, to normalize heart function quickly and effectively fill the gap BV. If the patient has a history of chronic heart or blood vessels, the hypovolemia and hypoxia exacerbate the underlying disease. So sick of this

nature requires greater myocardial contractility - vasoactive and inotropic agents. They are assigned to in case of persistent hypotension, can not be corrected adequate fluid therapy.

Of inotropic agents drug "first line" to keep the heart and kidneys is dopamine (400 mg in 250 ml of isotonic solution) or dobutamine. Dopamine infusion rate chosen depending on the desired effect: 2-5 mcg/kg/min ("renal" dose): extends mesenteric and renal vessels without increasing heart rate and blood pressure, 5-10 mcg/kg/min: pronounced inotropic effect, soft vasodilation due to stimulation of β2-adrenergic receptors or moderate tachycardia, 10-20 mg/kg/min to further strengthen inotropic effect, marked tachycardia, more than 20 mg/kg/min with the threat of sudden tachycardia tachyarrhythmias narrowing of veins and arteries due to α1-adrenoceptor stimulation and deterioration of tissue perfusion.

The need for vasopressors against the adequate fluid resuscitation indicates decompensated severe irreversible shock.

Treatment of kidney failure. In order to prevent the development of oliguric ARF form, there should be:

• monitoring hourly urine output (normal adult it is 0,5-1 mL/kg/h in children - more than 1 mL/kg/h);

• measuring the levels of sodium and creatinine in urine and plasma environments (in renal failure serum creatinine level greater than 150 mg/dL, glomerular filtration rate - below 30 mL/min);

• stimulation of diuresis on the background of restored BV (CVP of 10-12 cm of water column), satisfactory cardiac output, infusion of dopamine in the "renal" dose. The advantage given to loop diuretics: furosemide, which is the initial dose of 40 mg and, if necessary, increased to 160-240 mg;

• abolition of nephrotoxic drugs;

• avoid the use of vasoconstrictors.

Prevention and treatment of DIC are:

1. Categorical rejection of heparin to interrupt intravascular coagulation (due to lack of methods for accurate diagnosis of the first phase of DIC and its transition into the second phase).

2. Early and rapid administration of fresh frozen donor plasma. The main purpose of the application is to update the hemostatic potential of blood by balancing the state of proteases and antiproteases, clotting, and anticoagulants, components kinin and fibrinolytic systems and their inhibitors.

3. The use of protease inhibitors (contrycal, gordox).

4. Stimulation of the vascular-platelet link of hemostasis (dicynone, etamzilat).

Treatment and prevention of infection is carried out according to the standard schemes.

Burn shock - regarded as a variant of traumatic, but there are significant differences, which are defined by massive shifts of water areas with the development of long-lasting edema, mainly in the area of thermal injury. In the first hours after the burn victims, even in the absence of massive shifts in the water areas of the body, the weight of the patient is associated with pain and psycho-emotional stress, which serve as a trigger for neuro-endocrine response, manifested by the release into the bloodstream of hormones and other biologically active substances pituitary and adrenal cortex. Increase in vascular permeability in burns important role belongs to vasoactive amines (histamine), kinin system (bradykinin), a derivative of the cascade of fatty acids, C3 fraction of complement, oxygen radicals that appear in response to a direct thermal damage to the skin and underlying deeper tissues. Among these inflammatory mediators is particularly important derivatives of arachidonic acid.

When burn shock been less pronounced erectile phase duration and a large common shock.

Hypovolemia - the leading factor in the development of pathological changes in burn patients. Almost immediately after the impact of the thermal factor in burned skin areas undergoing significant change: the content of the water is increased by 75%, sodium – by 100%, protein in the interstitial fluid - by 350%.

With burn injury violated the protective function of the skin and increase perspiration losses through a burn wound. Fluid loss is celebrated immediately after the burn, but clinically significant values, it reaches only 6-8 hours. Extrarenal loss in burn victims with an area up to 20-30% of body reach 50-100 ml/kg body weight. With a decrease in extracellular fluid volume by 15-20% or more, due to the intense evaporation from the surface of the burn, and 16-20 times higher than normal, there haemoconcentration and hypovolemia.

Thus hypovolemia form different mechanisms:

1. Increased vascular permeability causes a decrease in the BV transition from the liquid part of blood (plasma) of the channel in the interstitial space of a burned and undamaged tissue.

2. The increase in protein content and increase oncotic pressure in the interstitial space encourage active flowing into the fluid from the blood vessels, which reduces the BV.

3. Increased in baked tissue osmotic pressure causes increased inflow into the affected area and increase its swelling, which reduces the BV.

4. Dysfunction of cell membranes of unfired tissues leads to the movement of intercellular fluid into cells, reducing the BV.

Clinic of burn shock

Practical experience has shown that the symptomatic manifestations of the general reaction to thermal injury with the possibility of developing an adverse outcome with burns over 15-20% of the body surface. Why in the world for immediate holding such patients infusion or oral fluid therapy, which reduces the severity of upcoming disorders and their consequences, called burn shock.

Mild shock (area burned less than 20%) of patients experiencing severe pain and burning at the site of burns. In the first minutes and hours can be exciting. Tachycardia to 90. Blood pressure is normal or slightly elevated. Breathlessness not. Decreased urine output. Ate treatment is delayed for 6-8 hours, or is not carried out, there may be oliguria and moderate haemoconcentration.

In **severe shock** (20-50% burned area) rapidly growing lethargy, weakness when saving consciousness. Tachycardia was more pronounced (up to 110), blood pressure is stable only in infusion therapy and the introduction of cardiotonics. Patients suffer thirst, marked dyspepsia (nausea, vomiting, hiccups, flatulence). Often there is a paresis of the gastrointestinal tract, acute gastric dilatation. Decreased urination. Urine output is only guaranteed using drugs. Haemoconcentration expressed - hematocrit is 65. From the first hours of the injury is determined by a moderate metabolic acidosis with respiratory compensation. Patients are cold, the body temperature is below normal. Shock can last 36-48 hours or more.

At the third stage (**most severe**) of shock (burn more than 50% burned area) condition was extremely grave. After 1-3 h after injury mind becomes confused, lethargy and stupor occur. Weak pulse, blood pressure decreased to 80 mm Hg and below (in the background infusion therapy, administration of cardiotonic, hormonal, and other means). Shortness of breath, breathing shallow. Often there is vomiting, which can be repeated, the color "coffee grounds." Severe gastrointestinal paresis. Urine in the first portion with evidence of micro- and macrohematuria, then dark brown with sediment. Quickly comes anuria. Haemoconcentration detected within 2-3 h, hematocrit rises to 70 or more. Increases hyperkalemia and decompensated mixed acidosis. Body temperature falls below 36 °. Shock can last up to 3 days or more.

Treatment fired in a state of shock is based on the pathogenesis of the premises and is found by the rules of intensive or critical care therapy.

1. Eliminating the impact of the damaging factor.

2. Anesthesia.

3. Ensuring adequate gas exchange.

4. Infusion therapy.

5. Correction of blood aggregation.

6. Treatment and prevention of organ dysfunction, organ failure.

7. Prevention and treatment of wound infections.

8. Correction of disorders of energy metabolism.

9. Early detoxification.

10. Prevention and treatment of gastrointestinal bleeding

Features of infusion therapy. Infusion is administered intravenously in a balanced salt solution through catheters 1.2-1.4 mm in diameter, set in one or two peripheral veins. Hold the central vein puncture. The volume and rate of infusion are determined by the weight of the patient, depending on the daily volume of fluid required to redress the deficit BV.

During infusion therapy guided by the following rules:

1. The rate of infusion and the componentity of the solutions are determined by the time from the moment of injury.

2. First 24 hours of burn shock:

a. daily infusion volume is calculated according to the formula Parkland:

V=4 x m x (A x 100), where mass (m) is in kilograms (kg), area (A) as a percentage of total body surface area, and volume (V) is in milliliters (mL).

b. infusion rate:

• 50% of the estimated volume administered in the first 8 hours;

• 25% - in the second 8 hours;

• 25% - in the third 8:00.

Formula should be regarded as indicative of installation. In the future, the amount and rate of administration of therapeutic agents adjusted on the basis of indicators urine output, hematocrit, hemoglobin, pulse and blood pressure over time.

As with burns from the vascular bed, along with a large number of plasma out of sodium (0.5-0.6 mEq /% burn / kg of patient weight), infusion therapy is primarily aimed at filling the vascular bed and the restoration in it of sodium. For this purpose, normal saline or Ringer's lactate. The latter is preferable, since its composition is closer to the extracellular fluid.

Depending on the severity of the shock should be considered in the calculation of the ratio of different colloids and crystalloids. In severe shock calculated volume should include two thirds of crystalloids and third colloids, and the extremely severe shock and burns over 50% of body crystalloids and colloids are used in a ratio of 1:1.

If the infusion begins in hypotension several hours after the injury, along with the introduction of crystalloid possible transfusion of plasma substitutes (polyglukin, HES).

Introduction of protein colloids should be started after 12-24 hours after the start of infusion therapy, when it is a balancing of intra- and extravascular sectors. FFP provides the greatest effect, which has all the protein fractions and affects osmotic and oncotic properties of blood. Albumin should be used when violations decrease the permeability of the vascular wall and stop the growth of swelling in the area of the burn. Air infusion of protein drugs - 1-2 ml/kg/hour.

Glucose solutions in the first day of severe burn injury in adults do not use, because due to dysfunction of the capillary membrane, they penetrate into the extracellular space and contribute to the formation of edema, malnutrition tissues and exacerbate already existing damages.

On the second day, the volume of infusion is 50-70% of the initial volume (at the achievement of the urine output <50 mL / h). Burn shock can last up to 3 days. Fluid therapy should be done all the time without a break. In order to improve the rheological properties of blood are appointed by protein-free medium and low molecular weight colloid solutions in a volume of 400-800 ml at a rate of 2 ml/kg/hour. At present, the burn shock transfusion do not spend, but the latter is indicated for large blood loss during necrotomy or massive hemolysis after removing the patient from shock.

In severe and extremely severe shock, when therapy is started late, it may be impossible to maintain blood pressure above 90 mm Hg by the introduction of crystalloids and colloids in calculated amounts. In such cases it is advisable not to increase the amount of fluid injected, as this may lead to increased interstitial and intracellular fluid, and use drugs inotropic action (dopamine at a dose of 5-10 mcg/kg/min). This dose dopamine improves myocardial contractility and increases cardiac output. At a dosage of 1-3 mg/kg/min, it helps to improve renal perfusion.

Difficulties arise in the treatment of burned skin burns when combined with thermal-inhalation airways disease. In these patients for shock sharply compounded toxic effects on the respiratory tract and the body in general, toxic combustion products. Feature of fluid therapy in these patients is the need to "balance" in the volume of infusion, as there is always the threat of pulmonary edema, and decrease the rate and amount of intravenous fluids causes decreased perfusion of the kidneys, helps to preserve and exacerbate hypovolemia. In such cases, you can resort to infusion therapy hypertonic solution of sodium (240 mEq / L). It is necessary to ensure that the level of plasma sodium less than 160 mEq / l. The introduction of a hypertonic solution

is advisable to limit the first 8-10 hours after the burn, that is the time most pronounced disorders vascular permeability. In cases where the growing phenomenon of respiratory failure, patients should be carried out mechanical ventilation with positive pressure on exhalation.

Normalization of rheological properties of blood is carried out by the above infusion therapy, i.e. due to the correction of hypovolemia, and through the use of low-dose heparin (20,000 units / day or more).

Normalization of diuresis, stabilize blood pressure, reduced haemoconcentration, increased body temperature, cessation of dyspeptic disorders and learning drunk fluid are indicators of the adequacy of treatment and patient exit from the state of burn shock.

Cardiogenic shock (CS)

Classification of cardiogenic shock (EI Chazov).

Reflex CS: decreased cardiac output due mainly reflex effect of the affected area on the pumping function of the heart and peripheral vascular tone. Therefore, to prevent the transition of reflex form CS "true" cardiogenic shock requires immediate and complete relief of angina attack.

Arrhythmic form CS due to acute hemodynamically significant arrhythmias and requires immediate restoration of sinus rhythm and normalization of heart rate, especially in paroxysmal tachycardia.

"True" CS is characterized by profound hypotension, severe left ventricular failure and peripheral circulation. The leading factor in the pathogenesis of the "true" CS is the sharp decline of cardiac pump function and tissue perfusion.

"Areactivity" CS differ from the "true" maximum severity of hemodynamic and clinical manifestations, lack of response to the introduction of inotropic agents and nearly 100% mortality.

"Hypovolemic" CS can be detected in a timely manner with the introduction into clinical practice of rapid methods for the determination of basic parameters of central hemodynamics.

Criteria for the diagnosis of cardiogenic shock:

1) systolic blood pressure less than 90 mmHg for 1 hour or more, the pulse pressure is reduced to 15-20 mmHg;

2) signs of hypoperfusion - cyanosis, cold moist skin, severe oliguria (urine output less than 20 ml per hour), congestive heart failure, mental disorders;

3) the heart rate above 60 bpm;

4) hemodynamic signs - cardiac index less than 2.2 L / min / m., And left ventricular filling pressures, which hypovolemia does not exceed 10-12 mm Hg, and at a "true" CS is usually higher than 25 - 30 mm Hg.

Diagnosis:

1. Similar studies in patients with uncomplicated MI.

2. Hourly monitoring of diuresis with urinary catheter.

3. Echocardiography (when possible).

4. Determination of arterial oxygen saturation (when possible).

5. Bedside chest radiography.

Treatment of cardiogenic shock

Treatment of cardiogenic shock requires immediate intensive activities.

It is mandatory that a permanent venous access using a catheter (preferably two catheters) into a peripheral vein, and the urinary catheter. If invasive monitoring of intracardiac and central hemodynamics is not possible, intravenous infusion of drugs is carried out under close monitoring of blood pressure, pulse, respiratory rate, lung auscultation. It is sufficient to rise to the level of systolic blood pressure of 90-100 mm Hg.

The choice of treatment for CS depends on the characteristics of the pathogenesis of left ventricular failure and the initial clinical and hemodynamic situation in each case.

Reflex CS (as a result of a pronounced anginal attack) - adequate analgesia - 1-2 ml of 0.005% solution of fentanyl or 1 ml of 1% solution of morphine or 1 ml of 2% solution of promedol intravenously slowly.

Arrhythmic CS (as a consequence of tachyarrhythmias (a) and bradyarrhythmias (b)) - correction of rhythm disturbances.

a) 5-10 ml of 10% solution of novocainamide in combination with 0.2-0.3 ml of 0.1% solution of mesatone intravenously for 5 minutes; or amiodarone 5 mg / kg intravenously slowly, then 150-300 mg dropwise in 5% glucose solution (with supraventricular and ventricular arrhythmias), 6-10 ml of 2% solution of lidocaine (trimecaine) intravenously within 5 minutes (with ventricular arrhythmias).

Electropulse therapy is used after preliminary anesthesia (sodium thiopental, sodium butyrate) in the absence of the effect of antiarrhythmic drugs.

b) temporary pacemaker, in its absence - 1-2 ml of 0.1% atropine solution intravenously slowly or (and also in the absence of effect) 1 ml of 0.05% alupent solution in 200 ml of 5% solution of glucose intravenously, drip, under the control of blood pressure and heart rate.

"True" CS (as a result of a sharp decrease in the pump function of the left ventricle), if indicated, inject low molecular weight dextran intravenously at a rate of at least 20 ml per minute (250 ml / 10 min.) Until the signs of shock disappear or until the CVP rises to 120-140 mm Hg. If systolic pressure remains below 90 mmHg, cardiotinics are used.

The use of positive inotropic drugs is the main method of standard therapy for cardiogenic shock. In severe cases, they are combined with vasopressors.

- dopamine: 5-20 mcg / kg / min, increase in dose by 1-4 mcg / kg / min, every 10-30 min - until the optimal answer
- dobutamine: 5-20 mcg / kg / min as a continuous infusion (a combination of low doses of dopamine (to stimulate dopamine receptors) and dobutamine is possible)
- norepinephrine: 1-2 mcg / min with blood pressure less than 70 mm Hg against the background of dopamine

Perhaps the use of phosphodiesterase inhibitors:

- milrinone (bipyridine), loading dose of 50 mcg / kg in 10 minutes, supporting - 0.375-0.75 mcg / kg / min as a continuous infusion

Phosphodiesterase III inhibitors have an inotropic and vasodilating effect, cause an increase in CO, SV and a decrease in the resistance of the pulmonary and peripheral vessels.

Treatment of the cause (myocardial infarction), possibly **earlier revascularization**.

Adequate thrombolytic therapy is an essential way to prevent and treat cardiogenic shock. Features of its implementation:

1. Low blood pressure is not an obstacle to the appointment of thrombolytics, since anti-shock measures are carried out simultaneously.

2. Thrombolytics can be prescribed in terms exceeding formally established, i.e. later than 12 hours from the debut of acute MI (within 24 hours).

3. When prescribing streptokinase to prevent a possible allergic reaction, preliminary iv bolus administration of prednisolone at a dose of up to 240 mg is indicated (preliminary administration of prednisolone does not prevent a characteristic hypotensive reaction to rapid infusion of streptokinase, since the latter is not associated with an allergic reaction).

In large clinical trials, thrombolytic therapy reduced hospital mortality from cardiogenic shock. Mortality of this group of patients was less than 50%.

Combined drug therapy of CS, in addition to normalizing the main hemodynamic providers, involves the correction of disorders such as hypoxemia and metabolic acidosis.

Oxygen is administered through nasal catheters or a mask at a speed of 4-6 l / min with its increase if necessary up to 8-10 l / min. Blood oxygen saturation is monitored. If, despite inhalation of 100% oxygen at a rate of 8-10 l / min, it is not possible to achieve a sufficient increase in blood oxygen saturation (PaO2 <60 mm Hg), artificial lung ventilation is indicated.

Decompensated metabolic acidosis worsens the functional state of the myocardium and significantly reduces the effectiveness of inotropic drugs. Correction of metabolic acidosis with prolonged cardiogenic shock is carried out by introducing a 4*% sodium bicarbonate* solution under the control of ABB indicators from the calculation of 0.5-1 mmol (1-2 ml) per kg. It must be remembered that excessive administration of sodium bicarbonate can lead to metabolic alkalosis with subsequent deterioration of oxygen transport and rhythm disturbance up to asystole.

The absence of a convincing clinical effect within 1-2 hours of intensive drug therapy is the basis for the use of *intra-aortic balloon counterpulsation.*

Anaphylactic shock

Pathogenesis of anaphylactic shock.

1. *Anaphylaxis* - the antigen - antibody.

2. *Anaphylactoid* (non-immune) - without the participation of the antigen - antibody, direct destruction of mast cells and release of inflammatory mediators.

The clinical picture. Manifestations of anaphylactic shock caused by a complex set of symptoms and syndromes. Shock is characterized by rapid development, rapid occurrence, severity and consequences.

There are roughly 5 different clinical manifestations of anaphylactic shock:

- Mainly affecting the cardiovascular system - the patient develops a sudden collapse, often with loss of consciousness. Especially dangerous is prognostic clinical variant of unconsciousness with involuntary emission of urine and stool. However, other allergic reaction (rash, bronchospasm) may be missing;

- ***Mainly affecting the respiratory system*** in the form of acute bronchospasm (asphyxia variant). This option is often combined with sneezing, coughing, a feeling of heat in the whole body, redness of the skin, hives, torrential sweat. Be sure to attach the vascular component (lowering blood pressure, tachycardia). In this regard, the face color changes from cyanotic to pale or pale gray;

- ***With a primary lesion of the skin and mucous membranes***. The patient experiences a sharp itching with subsequent development of allergic urticarial or angioneurotic edema type. Both can have symptoms of bronchospasm or circulatory collapse. Of particular concern are angioneurotic edema of the larynx, which is manifested initially stridor, and then the development of asphyxia.

With the above clinical variants of anaphylactic shock may occur symptoms indicating involvement of the gastrointestinal tract: nausea, vomiting, severe colicky abdominal pain, bloating, diarrhea (sometimes bloody);

- ***With a primary lesion of the central nervous system*** (cerebral version). To the forefront the neurological symptoms - psychomotor agitation, fear, severe headache, loss of consciousness and seizures, status epilepticus, or resembling the cerebral circulation. Marked respiratory arrhythmia;

- ***With a primary lesion of the abdomen*** (abdominal). In these cases the typical symptoms of "acute abdomen" (sharp epigastric pain, signs of peritoneal irritation), leading to misdiagnosis perforated ulcers or intestinal obstruction. Abdominal pain syndrome usually occurs in 20-30 minutes after the first signs of shock. In abdominal form of anaphylactic shock marked shallow disturbances of consciousness, a slight decrease in blood pressure, lack of pronounced bronchoconstriction and respiratory failure.

There is a definite pattern: the less time has elapsed from the time of allergen into the body, the more severe clinical picture of shock. The largest percentage of deaths observed in the development of shock after 3-10 min from the time of exposure to an allergen, as well as in the form of lightning.

Although in most cases the diagnosis of anaphylaxis is not difficult, it is sometimes necessary to differentiate it from acute cardiovascular failure, myocardial infarction, epilepsy, solar and heat stroke, pulmonary embolism, etc.

Thus, given the sharp and heavy for the condition of patients in anaphylactic shock, the need for emergency intensive care and the lack of specific, available for use in the general practice of laboratory data, it should be stated that the ***diagnosis*** based on the main shock of typical clinical manifestations and anamnesis data.

Clinical variants of the course of anaphylactic shock.

1. *Acute malignant* - no complaints, expressed collapse resistant to therapy, poor prognosis, diagnosis retrospectively.

2. *Acute benign* - stunning, moderate respiratory failure and circulatory therapy effective.

3. *Abortifacient* - fast disappearing symptoms, the most favorable course.

4. *Prolonged* - more than 6 hours, the allergen depot.

5. *Acute recurrent* - repeated shock 4-5 to 10 days, allergen depot.

Treatment of anaphylactic shock is to provide immediate relief to the patient as well as the minutes and even seconds delay and confusion, a doctor could lead to a patient's death by asphyxia, heavy collapse, cerebral edema, pulmonary edema, etc.

Remember that all drugs should be injected with syringes, does not use for administration of other drugs. The same requirement applies to drip infusion system and catheters to avoid re anaphylactic shock.

Combined treatment should be absolutely urgent, held in a clear sequence (about the possibility of a time) and have a certain pattern:

• first need to put the patient to turn his head to the side, pull the lower jaw to prevent the tongue, asphyxia and prevention of aspiration. If the patient has dentures, they should be removed. Ensure supply of fresh air to the patient or inhaled oxygen;

• immediately introduce a 0.1% solution of adrenaline. If there is no venous access and opportunities quickly catheterized vein, adrenaline should be administered intramuscularly in a dose of 0.3-0.5 ml. Intramuscular injection can be performed quickly. It was noted that in many cases, anaphylactic shock, even intramuscular antishock mandatory funds be sufficient to fully normalize the patient's condition. You can not type in one place more than 1 ml of epinephrine, because, having a great vasoconstrictor, and it inhibits the absorption of its own. The drug is administered by fractional 0.3-0.5 ml at different sites every 10-15 minutes before removing the patient from collaptoid state or vein catheterization. Binding benchmarks for epinephrine should be indicators of heart rate, respiration and blood pressure;

• If possible, stop the further flow of the allergen into the body - discontinue the drug, carefully remove the stinger with poison sac, if stung by a bee. In no case can not squeeze or massage the sting of the bite, as it increases the absorption of the poison. Above the site of injection (stinging), tourniquet, permitting localization. Site of injection drugs (stinging) drugged with 0.1% solution of adrenaline in the amount 0.3-1 ml and attach to it the ice to prevent further absorption of the allergen. With instillation of allergen medication nasal passages or conjunctival sac should be washed under running water. It must be remembered, if anaphylactic shock originated in the treatment room or dressing room, where the air is saturated with vapors of different drugs, the patient after injection of adrenaline, hormones urgent need to put in a separate room or another room, and then continue the intensive care. When taken orally, the allergen is washed with a sick stomach, permitting his condition;

• In parallel with the initial measures appropriate to make the vein puncture and put a catheter for infusion of fluids and drugs;

• If hypotension (once - in the presence of IO access to or following the initial intramuscular) adrenaline slowly injected in a dose of 0.25 to 0.5 ml pre-diluted in 10 ml of isotonic sodium chloride solution, or as an infusion of 1 - 4 mg / min adults (in children - 0.1 mcg / kg / min.). Perhaps intratracheal instillation - 1 ml solution of 1:1000 for 10 ml of 0.9% solution of sodium chloride. Necessary to monitor blood pressure, pulse and respiration. If hypotension remains stable amid severe tachycardia, must establish a drip of 1-2 ml of 0.2% solution of norepinephrine in 300 ml of 5% glucose solution;

• to restore blood volume and improve microcirculation to intravenously administered crystalloid and colloid solutions. The increase in BV - essential to the successful treatment of hypotension. Infusion therapy can be started with the introduction of isotonic sodium chloride solution, Ringer's solution up to 1000 ml. In the future, it is advisable to use colloidal solutions: 5% albumin, dextrans (reopolyglucin), hydroxyethyl starch. The amount of the liquid is determined by the plasma substitutes and blood pressure, central venous pressure and the state of the patient;

• corticosteroids are used from the beginning of anaphylactic shock, as to provide the degree of severity and duration of allergic reaction is impossible. The initial doses of hormones in the acute period: hydrocortisone - 100 mg iv or methylprednisolone 40-250 mg (1-2 mg / kg) iv every 6 hours. Drugs administered intravenously. The duration of treatment and the final dose depends on the patient and the effectiveness of treatment of an acute reaction;

• with bronchospasm, do not respond to inhaled adrenaline-β-adrenergic agonists. For relief of bronchospasm against occupied hypotension recommended as intravenous aminophylline 2.4% solution with 10 ml of isotonic sodium chloride solution or a 40% glucose solution. With persistent bronchospasm aminophylline dose is 5-6 mg / kg body weight;

• If you have stridor and no effect of the treatment you should immediately make intubation. In some cases, a conicotomy is made for health reasons;

• The need to ensure adequate pulmonary ventilation: be sure to suck the accumulated secretions from the trachea and mouth, as well as up to relieve the serious condition hold oxygen therapy, if necessary - MV or assisted mechanical ventilation;

• antihistamines better to enter after the restoration of hemodynamic parameters, as they do not have immediate effect and are not life-saving. Some of them may themselves have a hypotensive action, especially pipolfen (Promethazine).

Note that you can not enter suprastin in allergy to aminophylline. Application pipolfen contraindicated in anaphylactic shock caused by a group of drug phenothiazine derivatives.

Antihistamines can be administered intramuscularly or intravenously: 1% solution dimedrol to 5 ml or solution tavegil - 2-4 ml every 6 hours. Shown as the introduction of H_2 histamine receptor (famotidine, ranitidine)

• If seizures with strong excitation must enter 5-10 mg of diazepam intravenously.

• If, despite the efforts therapeutic measures, hypotension persists, we must assume the development of metabolic acidosis and begin infusion of sodium bicarbonate solution at the rate of 0.5-1 mg / kg body weight, control of ABG;

• the development of acute pulmonary edema, a rare complication of anaphylactic shock, there should be a specific drug therapy. The clinician should carefully differentiate hydrostatic pulmonary edema that develops during acute left ventricular failure, from edema, arising due to the increase in membrane permeability, which happens often in anaphylactic shock. The method of choice in patients with pulmonary edema that developed as a result of an allergic reaction is mechanical ventilation with positive pressure end-expiratory (PEEP) 5 cm H_2O, and while continuing fluid therapy to complete correction of hypovolemia;

• in case of cardiac arrest, no pulse and blood pressure shows emergency cardiopulmonary resuscitation.

Septic shock

Patients with septic shock are a special category of clinical and pathophysiological characteristics significantly different from the group of patients with cardiogenic and hemorrhagic shock. Hemodynamic status in septic shock is very different from the hemodynamic changes characteristic for the other categories of shock. In normal microvascular perfusion is controlled so that the tissues with higher metabolic rate is supported by more intensive blood flow. At rest only works 25-30% of the capillaries, which is 5-10% of the BV. In the early stages of septic shock is often reduced SVR and increased CO. The degree of peripheral vasodilatation is closely correlated with the severity of sepsis and is dependent on the intensity of emission of various mediators.

The distribution of blood flow is disturbed in spite of the increase in cardiac output, due to damage to the peripheral circulation autoregulation of tissue perfusion with a high level of exchange is not sufficient to meet the metabolic needs, whereas tissue with lower metabolic perfused redundant. A characteristic feature of septic shock is damage to tissue oxygen extraction mechanism. Development of systemic inflammatory response increases energy requirements of tissues and increasing oxygen debt. Violation ensure tissue oxygen than autoregulation disorders, is also associated with microaggregation, endothelial and perivascular edema, damage of intracellular transport mechanisms. Decompensated septic shock is characterized by joining hypovolemia, leakage of fluid from the bloodstream to tissues and heart failure. Myocardial depression, on the one hand, due to the decrease in coronary blood flow, and the other - the influence of circulating septic patients of various mediators, including tumor necrosis factor (TNF) and a factor depressing the myocardium.

By definition, a consensus conference on the ACCP / SCCM:

Septic shock (SS) *- is sepsis with signs of tissue and organ hypoperfusion and hypotension, infusion therapy does not eliminate the use and requiring catecholamines.*

Sepsis, systemic inflammatory response syndrome in the invasion of microorganisms.

Advanced diagnostic criteria of sepsis

General Criteria

• Fever temperature> 38 ° C

• Hypothermia temperature <36 ° C

• Heart rate > 90/min (> 2 standard deviations from the normal age range)

• Tachypnea

• Violation of consciousness

• Swelling or the need to achieve positive water balance (> 20 mL / kg per 24 hours)

• Hyperglycemia (> 7.7 mmol / L) in the absence of diabetes

Inflammatory changes

• The leukocytosis > 12×10^9 / L

• Leukopenia < 4×10^9 / L

• The shift to immature forms (> 10%) with normal leukocyte

• C-reactive protein > 2 standard deviations from the N

• Procalcitonin > 2 standard deviations from the N

Hemodynamic changes

• Hypotension: SBP <90 mm Hg, mean BP <70 mm Hg, or a decrease SBP more than 40 mm Hg (adults) or decrease SBP at least 2 standard deviations below the age norm.

• SrO2 saturation < 70%

• Cardiac index > 3.5 l/min/m^3

Manifestations of organ dysfunction

• Arterial hypoxemia PaO_2/FiO_2 <300

• Acute oliguria < 0.5 ml / kg / h

• Increased creatinine by more than 44 mmol / L (0.5 mg%)

• Thrombocytopenia <$100x10^9$ / L

• Violation of coagulation APTT > 60 seconds or INR> 1.5

• Hyperbilirubinemia > 70 mmol / L

• Intestinal obstruction (no bowel sounds)

Indicators of tissue hypoperfusion

• hyperlactatemia > 1 mmol / L

• Syndrome of delayed filling of capillaries, marbling limbs

Principles of treatment

1. Remediation of the site of infection and antimicrobial therapy

2. Restoration of perfusion and tissue oxygenation

3. Immunomodulation

4. Antitoxic and anticytokine therapy

5. Replacement, symptomatic, supportive therapy with multiple organ failure

1. Pathogenetic therapy of septic shock is reduced to *the rehabilitation centers of infection, antibiotic broad-spectrum*. Remediation of an infectious focus is the cornerstone of treatment of septic shock. Even the most powerful antibiotics and other methods of detoxification therapy is ineffective in the absence or lack of rehabilitation focus. Targeted antibiotic available after selecting the agent and determine its sensitivity to antibiotics, that is, at best, no sooner than 48 hours. However, early antibiotic treatment (within 30 min by admission) significantly reduces mortality in these patients. Therefore, it seems reasonable to use the so-called principle of de-escalation of antibiotic therapy with the initial administration of antibiotics as broad-spectrum (carbapenems, fluoroquinolones, cephalosporins 4th generation), followed by replacement, if possible, certain antibiotics (as a result of bacteriological research) spectrum.

2.1 *Hemodynamic Support.*

Infusion therapy belongs to the original measures to maintain hemodynamic and cardiac output in the first place. According to the American College and the American Association of Critical Medicine, approximately 50% of septic patients with major hemodynamic parameters can be normalized by adequate fluid resuscitation. The main objectives of infusion therapy in patients with sepsis are: restoration of adequate tissue perfusion, normalization of cellular

metabolism, correcting disorders of homeostasis, reducing the concentration of mediators of septic cascade and toxic metabolites.

Start infusion therapy with crystalloid administration - bolus of 20 ml / kg for 20-30 minutes and then after re-evaluation of hemodynamics, at a rate of 20-30 ml / kg / h under the control of central venous pressure and hemodynamics to a total dose of 4 liters (60 ml / kg)

For infusion therapy within the framework of targeted intensive care of sepsis and septic shock, crystalloid and colloidal infusion solutions are used with practically the same result.

All infusion media have both advantages and disadvantages. Taking into account the available experimental and clinical studies, to date, there is no reason to prefer any of infusion media. However, keep in mind that for adequate correction of venous return and preload level requires significantly higher volumes (2-4) infusion of crystalloids than colloids, due to the peculiarities of the distribution of solutions across sectors. In addition, the infusion of crystalloid more carries a risk of edema, and their hemodynamic effect is less prolonged than colloids. At the same time cheaper crystalloids do not affect the coagulation potential and not provoke anaphylactoid reactions. In this regard, the qualitative composition of the infusion of the program should be determined by the peculiarities of the patient: the degree of hypovolemia, DIC phase syndrome, the presence of edema and blood albumin levels, the severity of acute lung injury.

Plasma expanders (dextran, gelatinol, hydroxyethyl starch) are shown in patients with severe deficiency of the BV. Hydroxyethyl starch (HES) with a molecular mass of 200/0, 5, and 130/0, 4 have a potential advantage over dextrans by a lower risk of leakage through the membrane and the absence of clinically significant effects on hemostasis. The use of albumin in critical conditions may increase mortality. Colloid osmotic pressure increase in albumin infusion is transient, and then in the syndrome of "capillary leak" is further extravasation of albumin (rebound syndrome). Albumin transfusions might be useful only when the level of albumin less than 20 g / l and no signs of it "leaks" into the interstitium. Application of cryoplasma shown in consumption coagulopathy and reducing blood coagulation potential. According to most experts, the minimum hemoglobin concentration in patients with severe sepsis should be between 90-100 g / l. Sepsis and secondary school should strive to achieve faster (the first 6 hours after admission) targets the following: CVP 8-12 mm Hg, SBP> 65 mm Hg, urine output of 0.5 ml / kg / h, hematocrit 30%, oxygen saturation of blood in the superior vena cava or right atrium of at least 70%.

Low perfusion pressure requires immediate inclusion of drugs that increase vascular tone and / or inotropic function of the heart. Dopamine and / or norepinephrine are the drugs of first choice correction of hypotension in patients with SS. Norepinephrine (with an initial rate of 1 mg / min (adults), selecting dose to achieve systolic blood pressure of 90 mm Hg. increases in SBP and increases glomerular filtration. Optimization of systemic hemodynamics by the action of norepinephrine leads to improved renal function without the use of low-dose dopamine. Recent

studies have shown that the use of norepinephrine in comparison with a combination of high doses of dopamine to norepinephrine ± leads to a statistically significant reduction in mortality.

Adrenalin - adrenergic drugs with the most pronounced side hemodynamic effects. Adrenaline has a dose-dependent effect on heart rate, mean BP, cardiac output, left ventricular work, delivery and consumption of oxygen. However, this action of adrenaline followed by tachyarrhythmias deterioration splanchnic blood flow, hyperlactatemia. Therefore, the use of epinephrine should be limited to cases of complete refractoriness to other catecholamines.

Dobutamine should be considered as the drug of choice to increase cardiac output and oxygen delivery and consumption at normal or elevated preload. Thanks to the pre-emptive effect on $ß_1$-receptors dobutamine more than dopamine contributes to the given parameters.

Catecholamines in addition to support circulation may interfere during systemic inflammation by interfering with the synthesis of key mediators that have distant effects. Under the action of adrenaline, dopamine, norepinephrine and dobutamine decreased synthesis and secretion of TNF activated by macrophages. Cardiocirculatory support drugs should be discontinued 24-36 hours after stabilization of central hemodynamics.

Refractory septic shock - a persistent hypotension despite adequate infusion, the use of inotropic and vasopressor support. In the case of refractory septic shock shows the introduction of corticosteroids - hydrocortisone 240-300 mg on the first day. After pressure stabilization dose can be reduced to 50 mg every 8 hours the next 48 hours. The duration of treatment - 5-7 days.

2.2. *Respiratory support.*

Light very early become one of the first to target organs involved in the disease process in sepsis. Acute respiratory failure (ARF) - one of the leading component of multiple organ dysfunction. Clinical and laboratory manifestations of ARF in sepsis syndrome match of acute lung injury and the progression of the disease process - acute respiratory distress syndrome (ARDS). Conduct oxygen inhalation, and on the testimony of endotracheal intubation and mechanical ventilation.

3. Appropriateness of including intravenous *immunoglobulin* (IgG and IgG + IgM) is related to their ability to limit excessive action of proinflammatory cytokines, increase the clearance of endotoxin and staphylococcal superantigen, increase the effect of ß-lactam antibiotics. The best results are obtained using antibodies in the early phase of shock ("warm shock") and in patients with severe sepsis. Use pentaglobin (IgG and IgM), intraglobin (IgG), ronleykin.

4. In order to prevent the formation and accumulation of kinin-like peptides and MDF shows the use of *protease inhibitors*: contrycal 80000-150000 Units or gordox at a dose of 200-400 KIU a day, pentoxifylline at a dose of 100-300 mg potentiates the anti-inflammatory action

of adenosine, prostacyclin and prostaglandin E class at the expense of synergies when exposed to cyclic AMP.

5. *Prevention and treatment of multiple organ failure*, including

• correction of microcirculatory disorders and systemic coagulation - reopoliglyukin, heparin (unfractionated heparin, low molecular weight heparin), combined with fresh frozen plasma, activated protein C

• glycemic control

• prevention of the formation of stress ulcer gastrointestinal tract

In conclusion, the clinical criteria for the adequacy of anti-shock therapy are:

1) stabilization of central hemodynamic parameters (SBP 60-100 mm Hg, CVP of 60-100 mm H_2O, HR 60-100 bpm);

2) normalization of hemic parameters (Hb 100 g / l, Ht 0.3);

3) restoration of urine output (0.5-1 ml / min).

Remember that the output state of shock involves not only the restoration of normal blood flow, but the lack of persistent multiple organ disorders.

Self-study

Task number one

Examine patients admitted to ICU diagnosed with gastrointestinal bleeding. Determine the amount of blood he/she had. To do this:

• Assess the level of consciousness;

• Gather history;

• Appreciate the color, moisture, skin temperature;

• Determine pressure, pulse, RR, urine output, central venous pressure, a symptom of the "white spots";

• Calculate the shock index (Algovera);

• Determine the value of BV deficit in % of predicted;

• appreciate the laboratory parameters (Hb, Ht, RBC);

• Calculate the amount of blood loss from the formula Moore.

Task number two

Analyze the history of a patient with severe nosocomial pneumonia, systemic inflammatory response syndrome, is in the intensive care unit and intensive care. To do this:

• analyze the degree of hemodynamic disorders and their correction;

• appreciate the severity of respiratory failure in a patient based on the diaries of follow-up, to evaluate the proposed method of treatment of respiratory failure, if necessary, make the corrections and justify them;

• evaluate antibiotic therapy

IX. Clinical problems

Objective number one

In patients admitted to hospital with a diagnosis of intra-abdominal bleeding, pulse 112 per minute, systolic blood pressure of 90 mm Hg. Determine the level of blood loss, and evaluate it on the classification of PG Bryusov?

Objective number two

The patient, 34 years old taken to hospital from fire. Thermal injury of the skin is missing, the nose and lips - traces of soot. Objectively - shortness of breath up to 28 per minute, noisy breathing, auscultation - tough, a lot of wheezing. Your presumptive diagnosis? Is it necessary to hospitalize the patient ICU?

Test control:

1) The criteria for admission to the ICU for adults:

a) III degree burn more than 5% BSA

b) III degree burn over 15% BSA

c) Isolated thermo-inhalation injury

d) II degree burns over 10% BSA

e) Body circumference burns

f) Burns of the face

2) What is the main pathogenetic link for burn patients?

a) Violation of the lungs

b) Impaired renal function

c) Hypovolemia

d) Violation of the respiratory system

3) Measures of intensive therapy in septic shock:

a) Rehabilitation of the source of inflammation

b) Infusion Therapy

c) Oxygen

d) The use of vasoactive drugs

e) Antibiotic

f) Epidural blockade

g) Immunomodulating therapy

4) Indications for use of corticosteroids in sepsis:

a) The initial stage of septic shock in one-stage entry into the blood infection

b) Always indicated in sepsis

c) Refractory septic shock

5) In type 1 allergy inflammatory mediators released after degranulation of mast cells and basophils act primarily on the following organs - the target, except for:

a) The smooth muscles of the bronchi

b) Vascular smooth muscle

c) The skeletal muscles

d) Endothelial post-capillary venules

e) Peripheral nerve

6) The clinical picture of immediate hypersensitivity in the smallest degree by the following inflammatory mediator, released by degranulation of mast cells and basophils:

a) Histamine

b) Prostaglandins

c) Catecholamines

d) Heparin

e) Kinin

7) During an anaphylactic reaction following substances are released, except for:

a) Histamine

b) Slow-reacting substance of anaphylaxis

c) Heparin

d) Epinephrine

8) The symptom of "white spots" in the rate of:

a) 2 seconds

b) No more than 3 seconds.

c) 1 second

d) No more than 4 seconds

9) In the normal hourly urine output is:

a) 0,5-1 ml / kg

b) 1-2 ml / kg

c) 0,1-0,3 ml / kg

d) 2-3 ml / kg

10) Young men blood volume is:

a) 60 ml / kg.

b) 50 ml / kg.

c) 70 ml / kg

d) 80 ml / kg

Answers:

Objective number one

The data are sufficient to determine the index Algovera shock. 112/90 = 1.2, which corresponds to 40% of blood loss, which is abnormal in appearance, large in size and the degree of severe hypovolemia.

Objective number two

The patient thermo-inhalation injury which is an indication for hospitalization in intensive care.

Answers to test questions:
1) a, c, e, f 2) c 3) a, b, c, d, e, g 4) a, c 5) c 6) c 7) d 8) a 9) a 10) c