EXTREME CONDITION

During the life of a person can be exposed to various exogenous and endogenous factors of extreme force, duration and / or unusual, unusual character. The action of extreme factors leading to the development of an emergency or to adapt to this factor, or extreme, critical, urgent status.

Urgent adaptation is characterized by limiting the body's stress adaptation mechanisms that prevents the shift of the most important parameters and constants of its life beyond the normal range. The content of the state of emergency is the first stage adaptation of the adaptation process. Upon termination of the emergency factor, eliminate or compensate for its effects to normal body condition.

Extreme, critical, urgent condition characterized by significant impairment of vital activity, in spite of the limit activation mechanisms of adaptation. Independent exit the body of such a state, as a rule, is not possible. In such cases it is necessary to provide timely and effective medical care.

EXTREME AND TERMINAL STATES

Extreme conditions - severe general condition of the body, developing under the influence of extreme factors of the external or internal environment characterized by significant disturbances of vital activity, fraught with death.

Extreme conditions occur, as a rule, limiting the activation and subsequent depletion of coping mechanisms, rude disorders of functions of organs and physiological systems.

Extreme conditions require emergency medical care.

The most common and clinically important urgent conditions include collapse, shock, coma and poisoning.

From extreme is necessary to distinguish the terminal state, which represent the final stages of life of the organism, the borderline state between life and death.

terminal states

By the terminal states include all the stages of dying - preagonia, agonia, clinical death, as well as the initial stage of the state after a successful resuscitation.

Terminal states are usually the result of unfavorable course of extreme conditions. If the terminal is not able to carry out intensive medical measures or they are ineffective, it comes, and then the clinical - biological death.

The pathogenic factor → Extreme condition → terminal condition → Clinical Death → biological death

Extreme and terminal condition have both similar and radically distinguish their features.

The similarity of extreme and terminal states

Extreme and terminal states have a number of common features:

- Common causes.
- Similar key pathogenesis.
- Borderline situation between life and death.
- Lead to the death of the organism.
- Require urgent medical care.

Although there were similarities, the two states are qualitatively different groups from each other.

Differences extreme and terminal states

criteria	Extreme state	Terminal state
Intensity of the specificity of the causal factor	Increase	Low or absent
The specificity of pathogenesis links	Increase	Low or absent
The effectiveness of adaptation	Increase	Low

reversibility	High: spontaneous, under the influence of treatment	Spontaneously usually impossible; in the treatment of relatively low
Effectiveness of treatment	Increase	relatively low

At the heart of the terminal states are much more severe and therefore prognostically unfavorable processes. If not carried out emergency remedial measures, the terminal state of becoming progressive, irreversible flow, leading to death. In contrast, under certain extreme conditions (collapse, and sometimes - in the early stages of shock) can activate the processes of adaptation, reducing the degree of deviation of homeostasis parameters, improving the body's ability to live in and out of these states.

When development terminal state gradually lost its importance of nature caused the causal factor. Pathogenic effects of his actions are so great that the specific reasons for the state of the terminal loses its meaning.

Specificity is low and terminal states of development mechanisms. When different types of these states key links of their pathogenesis are hypoxia, abnormalities of acid-base balance, blood gases, toxemia, and others. In contrast, under extreme conditions identified as the specifics of the calling their agent, and especially their development mechanisms. In this regard, for specific causal and pathogenetic therapy of extreme conditions can block their development and normalize the functioning of the organism.

General etiology of extreme conditions

Extreme factors different from other pathogenic agents that provide the data and the specific conditions under the action of the body is very high, very intense, often devastating effect.

Types of extreme factors.

Extreme factors are divided into exogenous and endogenous.

1. extreme exogenous factors may be physical, chemical or biological nature.
- Factors of the physical nature: mechanical, electrical, thermal, barometric, radiation, gravity.
- Chemical factors: limiting the deficit / surplus of oxygen, metabolic substrates, liquid; expressed intoxication of drugs, industrial poisons, acids, alkalis.
- Biological factors: significant deficit / surplus of exogenous bioactive substances; bacteria, parasites and fungi (toxins, metabolic products thereof, and / or decay).

2. Endogenous (adverse, severe course of disease and disease states).
- Severe impairment of functions of organs and physiological systems.
- A significant blood loss.
- Massive bleeding in the organs.
- Excess food or allergic immune responses.
- A significant deficiency / excess of BAS and / or their effects.
- Mental surge injury.

Conditions that contribute to extreme conditions.

1. Factors potentiating effects extreme agents.

For example, the state of sensitizing the body can lead to a more severe course of anaphylactic shock by the action of the allergen, the effects of blood loss are compounded at elevated temperature, heart failure when performing excessive exercise can lead to cardiogenic shock, etc.

2. The reactivity of the organism.

Often it is a decisive condition for the action of extreme factors. Unlike normergic response hypo - or hyperergic condition of the body facilitates the occurrence, course and outcomes exacerbating extreme condition.

Pathogenesis of extreme conditions

Extreme conditions are characterized, as a rule, dynamic stage development. In most cases (except for acute and quickly developing situations caused by extreme superstrong factors leading to rapid death of the body) in the dynamics of increasing severity of extreme conditions can be divided into three stages: the activation of adaptive mechanisms, exhaustion and lack of them, extreme regulation of the body's vital functions.

Stages of extreme conditions.

Stage 1: Activation of adaptive mechanisms
The reason - the alarm action:
1) damaging factor,
2) deviations of the parameters of homeostasis
Stage 2: Failure of adaptive mechanisms
Causes:
1) increase the degree and extent of damage of the organism,
2) stress and exhaustion of adaptive processes
Stage 3: Extreme regulation of vital activity
Causes:
1) a further increase in the extent and scale of the body damage,
2) progressive failure of adaptive mechanisms.

Stage activation of adaptive mechanisms of the organism

This stage is characterized by a generalized activation of the legitimate functions of tissues, organs and systems. This is the basis of development of adaptive reactions of varying degrees of severity and specificity. Essentially all of these reactions can be divided into two categories:
- Providing specific adaptation to the particular extreme factors;
- Implement the non-specific, standard processes, developing under the influence of any extreme effects, ie stress reaction.

Stage failure of adaptive mechanisms

The main feature of this stage: the lack of efficiency of adaptive responses and growth of the damaging effect of extreme agent.
1. Pathogenesis
- Increasing the efficiency of reduction reaction devices, compensation, protection and reparation.
- Progressive disorder of the physiological functions and the collapse of the functional systems of the body.
- Metabolic disorders and physical-chemical processes.
- Damage to the structural elements of organs, tissues and cells
- Braking plastic processes them.

As a result of these changes most homeostasis parameters deviates beyond the normal range, and often significantly. Vital functions of the whole organism is disturbed significantly.
2. Vicious circles

With all the extreme states, albeit with a different frequency, can form a vicious circle. This phenomenon lies in the fact that an initial pathogenetic factor causes a closed complex by increasing the degree of violation. Consequences of them, in turn, potentiate the action of an initial pathogenic factor based on positive feedback.
- Education pathogenetic vicious circle leads to growth disorders in the body, "weighting" extreme conditions up to move it to the terminal, even in the termination of extreme factors.
examples:
- During the collapse, shock and coma observed blood flow redistribution. A large amount of blood is collected in the extended venous and arterial vessels of the abdomen, lungs, subcutaneous tissue. This significantly reduces the bcc and hence the blood flow to the heart. The consequent decrease in

cardiac ejection of blood leads to an even greater decrease in VCB and exacerbate the patient's condition.

- Activation phenomenon lipoperoxide processes in tissues. Hypoxia, growing in all types of extreme conditions, results in the suppression of the activity of non-enzymatic and enzymatic antioxidant defense system tissue. This leads to intensification of education in which reactive oxygen species and lipoperoxide processes that damage the enzymes of tissue respiration, glycolysis, the pentose phosphate shunt. The latter is even more aggravated hypoxia - a vicious circle and potentiating its development.

In general, at the stage of failure of adaptation mechanisms in various extreme states is developing a set of regular stereotypical interrelated changes in the body. For chief among them are the characteristic triad of disorders.

- Frustration and lack of functions of organs and physiological systems: the neuro-humoral, respiratory, cardiovascular, blood, hemostasis, and others.
- Significant abnormalities of homeostasis parameters, including essential and critical.
- Damage to the subcellular structures, cells and disruption of intercellular interaction.

Manifestations.

1. Disorders of the nervous system. They are characterized by different degrees of sensitivity and character disorders, motion control, integration of activity of bodies, tissues and systems, VNS.

- In the collapse (due to significant violations of perfusion of organs, including the brain) develops a condition characterized by indifference to events, the oppressed and the deep anguish.
- When shock conditions (in the braking phase) showed a significant inhibition, detachment, "leaving a" loss of contact with others.
- In a coma consciousness violated. First, there is confusion and drowsiness, and soon - loss of consciousness, combined with hyporeflexia. At the same time revealed significant disorders of blood circulation, breathing and other vital functions. The latter is a result of integrating function disorder of the nervous system.

2. Violation of the CVS activities. Manifested arrhythmias and signs of coronary heart disease, various disorders of the central, organ, and microcirculation. This leads to the development of capillarotrophic failure in various organs. This in turn potentiates the failure of their functions - heart, kidneys, liver, lungs, and others.

3. Variations in the blood system and hemostasis. Causes a violation of the volume, viscosity and flow of blood; forming units of its formed elements, the phenomenon of sludge, blood clots. A frequent consequence of hemostasis disorders are trombohemorragic state, often leading to death of the organism.

4. Disorders of external respiration. Exacerbate extreme conditions due to the potentiation of hypoxemia, hypercapnia and hypoxia. At the stage of failure of adaptation mechanisms, tend to develop so-called periodic breathing forms (Biota, Cheyne-Stokes, Kussmaul), while during heavy - its complete cessation - apnea.

5. The lack of kidney function. It appears oligo - or anuria, filtration disorder, excretion, secretion and other processes in them. This often leads to varying degrees of severity of uremia.

6. Violation of the liver. Causes the disorder of protein, carbohydrate and lipid metabolism, metabolism of bile pigments, the inactivation process of toxic metabolic products, potentiates hemostatic disorders. At the duration of the current extreme conditions (eg, coma), severe liver failure can significantly speed up the transition to the terminal.

7. Disorders of the functions of the digestive tract. Develop, as a rule, in case of severe during these extreme conditions such as shock and coma. They manifest violations of the secretory and motor functions of the stomach and intestines (until his paresis), cavity and membrane digestion. Therefore patients often develop autoinfection intestinal syndromes, auto-intoxication and malabsorption.

8. Significant deviations from the normal range of homeostasis parameters. It is a natural manifestation of the failure of functions of organs and systems. The most significant variations:

a) Reduction of the partial voltage and the oxygen content in the blood. This indicates the development of hypoxia, which is on the rise as well as severity of the condition progresses.
- Hypoxia in almost all varieties of extreme conditions plays a very important, and in many cases - a major pathogenic role, being one of the key and obligatory links of their pathogenesis.
- The origin of hypoxia, is usually mixed. It is caused by disorders of the central, organ, tissue and microcirculation, external and tissue breathing process of oxygenation of hemoglobin in the lungs and its dissociation in the tissues, as well as deficiency of substrate oxidation.
- During the development of the state of extreme hypoxia causes or potentiating factor cascade of disorders defined by it: acidosis, the intensification of the processes of generation of reactive oxygen species and lipoxide, imbalance and other ions.
b) Reduction of the pH of blood plasma and other biological fluids (acidosis development). Acidosis may be a gas (respiratory) and non-gas (metabolic and excretory).
c) Abnormal content of different ions. The breakdown of biological oxidation in the tissues and as a result - their energy and acidosis cause a violation of physico-chemical state of cell membranes and enzymes. This leads to the intracellular accumulation. Na +, Ca2 + and other ions exit the cell K +, Mg2 +, H +, trace elements. Therefore the optimal ratio of the ions is disturbed between cells and body fluids. Excess hydrophilic Na + in cells causes their overhydration, swelling and, frequently, the membranes rupture.
d) Changes in the value of the MP and PD characteristics of the surface charge of cells, their colloidal state plasmolemma and cytosol, the osmotic pressure of blood plasma.
9. Changes in the body at the stage of failure of adaptation and reaction mechanisms of extreme conditions may differ significantly for different kinds of (collapse, shock, coma) and even with the same form, but in different patients. The reason is that the original disorder functions, metabolism, plastic processes and structures cause a cascade of secondary violations.

Stage extreme regulation of vital activity

The main characteristics of the stage of extreme regulation of the body's vital functions.

Hypo - and deafferentation:
- Central nervous structures,
- Organs and tissues.

↓

Lack of functions and the collapse of the physiological and functional systems

↓

Minimization:
- The functions of organs and tissues,
- Energy expenses,
- Plastic processes.

↓

Metabolic regulation of the functions of organs and tissues

↓

The terminal condition

The reasons: the growth of the degree and extent of damage to the body, progressive failure of adaptation mechanisms.

Pathogenesis

Key pathogenesis:
- Increasing (with a significant energy shortage, acidosis, disorders of the ion balance and other changes in the body), hypo - and deafferentation of the central and peripheral nervous structures, as well as the executive organs and tissues.
- The collapse of functional systems that maintain the body's vital parameters.
- The transition to elementary - level metabolic regulation of vital functions of organs and tissues.

With an increase of these changes is developing a terminal condition and death occurs. However, holding on extreme control stage effective treatment can block the progression of the disorder, restore and even normalize the condition of the victim.

PRINCIPLES OF TREATMENT OF EXTREME CONDITIONS

Immediate therapeutic measures when extreme conditions are based on the implementation of the four basic principles: etiotrop, pathogenetic, and symptomatic sanogenetic.

Etiological treatment

Causal treatment is designed to stop or decrease the strength and scale of pathogenic action adventure agent. This is achieved by different methods depending on the type of emergency conditions.

For example:

- Stop the bleeding;
- Termination of the low or high temperature;
- Normalization of the oxygen content in the inspired air;
- Elimination of failure, organ function, hormone deficiency or their effects;
- The use of antitoxic means.

Pathogenetic treatment

Pathogenetic therapy aims to block the development of mechanisms for extreme conditions. This objective is usually achieved by acting on the key or key binding pathogenesis. These include circulatory disorders, respiratory depression, hypoxia development, shifts AAR, ion imbalance, activation processes lipoxigenic and others.

Sanogenetic therapy

Sanogenetic therapy aims to activate and / or potentiation of defense mechanisms, compensation, adaptation and compensation for lost or damaged structures and functions of the body. Ensured by stimulation of the heart function, respiratory, kidney, liver and other organs and tissues; activation of repair processes of detoxification systems, the elimination of excess oxygen radicals and lipid; potentiation of plastic and other reactions.

Symptomatic treatment

Symptomatic therapy involves the removal of unpleasant, painful, aggravating the condition of the patients symptoms and sensations: headaches, fear of death, causalgia, hypo- or hypertensive reactions and others.

In addition to the implementation of the general principles of the above-described treatment of extreme conditions, in each patient carried a set of individual measures, taking into account the specific characteristics of extreme exposure, especially to respond to him of the victim, the dynamics of life and severity of disorders, moreover, a particular version of an extreme condition.

Collapse

Collapse - general, developing acute condition that occurs as a result of significant discrepancies bcc capacity of the vascular bed. It is characterized by circulatory failure, the primary circulatory hypoxia, a disorder of functions of tissues, organs and systems.

Species origin collapse		
Cardiogenic	Hypovolemic	vasodilatory
- Myocardial	- posthemorrhagic	- hyperthermal
- Arrhythmic	- dehydration	- toxic

- cardiomiopathic	- toxic and infectious - orthostatic	- orthostatic

ETIOLOGY

The immediate cause of the collapse is a rapidly developing significant excess capacity of the vascular bed in comparison with VCB.

The reasons for the collapse of

1. By reducing the value of blood ejected from the heart ventricles in the vascular bed develops cardiogenic collapse. This occurs when:

- Acute heart failure (caused by ischemia and myocardial infarction, significant Brady - or tachycardia);
- Conditions that impede the flow of blood to the heart (with stenosis of the valve hole, embolism or vascular stenosis of the pulmonary artery system);
- There are obstacles to eject blood from the left ventricle (the most common in stenosis of the valve opening of the aorta).

2. When reducing the BCC develops hypovolemic collapse. By this result:

- Acute massive bleeding;
- Rapid and significant dehydration (with profuse diarrhea, poisoning, sweating, uncontrollable vomiting);
- The loss of a large volume of blood plasma (for example, extensive burns);
- Redistribution of blood from the deposit large amounts of it in the venous blood vessels, blood capillaries and sinuses (eg, in shock, gravitational overloads, some intoxication).

3. By reducing the total peripheral vascular resistance develops vasodilatory collapse. This can occur in severe infections, intoxication, hyperthermia, endocrinopathy (with hypothyroid conditions, acute and chronic adrenal insufficiency), incorrect use of drugs (eg, sympatholitic, ganglioblockage, drugs, calcium channel blockers), hypocapnia, blood excess adenosine, histamine, kinins, profound hypoxia and a number of others.

Risk factors

Collapse development is largely dependent on a number of specific conditions (risk factors):

- Physical characteristics of the environment (low or high temperature, level barometric pressure, humidity);
- The state of the body (the presence or absence of a disease, pathological process, psycho-emotional status, etc.).

These and other conditions can both promote and impede the emergence collaptoid nature, as well as a significant effect on the severity of its course and outcome.

Types of collapse

In addition to the most common - cardiogenic, hypovolemic and vasodilatory collapse in the practice of medicine frequently isolated species collapse, taking into account its specific cause or group of related reasons: posthemorrhagic, infectious, toxic, radiation, pancreatic, orthostatic, hypocapnic and others.

GENERAL PATHOGENESIS AND MANIFESTATIONS COLLAPSE

1. Violation of the functions of the CAS is an initial and main pathogenetic link of collapse and is characterized by inadequate blood supply to organs and tissues. Typical of these circulatory disorders:

- Reduced stroke volume and cardiac output;
- Acute hypotension;
- Venous congestion;

- The redistribution of blood flow (blood deposition in the capacitance vessels of the abdomen, lungs, spleen and brain hypoperfusion, heart and other organs);
- Violation of the microcirculation of blood and lymph;
- The development of capillary-trophic failure.

2. The disorder of the nervous system. It has an important pathogenetic significance. The collapse is usually accompanied by:
- Lethargy;
- Apathy, indifference to what is happening;
- Tremor of the fingers;
- Sometimes convulsions;
- Hyporeflexia;
- Syncope with significant hypoperfusion and cerebral hypoxia.

3. Violations of the gas exchange function of the lungs. This is evidenced by:
- Frequent and shallow breathing;
- Hypoxemia;
- Hypercapnia in the blood flowing from the lungs.

4. The breakdown of the excretory renal function. Given that the collapse of the system is characterized by acute arterial hypotension and therefore - renal hypoperfusion, patients often detected:
- Oliguria;
- Baruria;
- Hyperasotemia.

5. Disorders of blood and hemostasis systems:
- Hypovolemia;
- Increase in blood viscosity (in connection with the release of its liquid part through the walls of blood vessels, the permeability of which is increased under conditions of hypoxia);
- Hyperaggregation platelets and red blood cells;
- Blood clots;
- The development of the phenomenon of sludge.

Many of the above symptoms of collapse caused by the development of hypoxia, circulatory initially, and subsequently mixed (including respiratory, hematic, fabric, substrate). With increasing severity of hypoxia may develop significant disturbances of vital activity, fraught with death.

FEATURES OF CERTAIN COLLAPSE

Despite the similarities of the pathogenesis and manifestations of various kinds of collapse, some of which have significant differences.

Hemorrhagic collapse

An initial pathogenetic factor hemorrhagic collapse - a rapid and significant decrease in the bcc (hypovolemia). The increase in this context of vascular tone does not eliminate the disparity of their capacity to significantly reduce the BCC. As a result, developing hypoperfusion of organs and tissues. This leads to increasing initially circulatory and then mixed (hemic, tissue) hypoxia.

The consequence of hypoxia is a progressive disorder of the nervous system, lungs, kidneys, etc.

To replenish the lost blood (mainly its formed elements by activating hematopoiesis) takes a few days (in severe cases, not less than 7-12 days); therefore (bicarbonate, etc.) to eliminate hemorrhagic collapse and its consequences require urgent administering to the calculated amount of whole blood, red cells, plasma expanders, solutions containing the necessary quantity of ions and buffer system components.

Orthostatic collapse

An initial element of the pathogenesis of orthostatic collapse (fainting) - systemic vasodilatation resulting in a sharp decrease in arteriolar tone walls and capacitive vessels. There is a sharp transition of the body in a vertical position from lying or sitting position, especially after prolonged inactivity. This results in the activation of cholinergic effects on vascular walls due to the stimulation of the neurons of the vestibular centers with a sudden change in body position.

An important risk factor, and then the pathogenetic link orthostatic collapse - reduction of reactive properties of the resistive vessel walls to vasopressor agents: catecholamines, angiotensin, etc. One of the major reasons for this may be adrenal insufficiency (warrant the deficiency of catecholamines and corticosteroids), upset kardiovazomotornogo center functions and hypothalamus (causing violation of the tone of the vessel walls) and some others.

Toxic-infectious collapse

The cause of toxic and infectious collapse: the pathogenic effects on the body toxins exogenous and endogenous origin of infectious or non-infectious nature (microbial toxins and parasites in their massive destruction, for example, as a result of antibiotic treatment, the products of disturbed metabolism, phosphorus compounds, carbon monoxide and many other).

Toxins are responsible for:

- Direct neuro, cardio and myotropic damage;
- Metabolic disorders and the implementation and effects of vasopressor agents vasodepressor;
- Disorders of mechanisms of regulation of vascular tone and cardiac activity.

As a result of growing expressed in varying degrees of decreased tone of resistive vessels, cardiac output, VCB.

With a significant and progressive decrease of these indicators are observed rapidly growing life of the organism disorder and poses a threat to his life.

METHODS OF TREATMENT OF COLLAPSE

The therapy is based on the realization collapses etiotropic, pathogenetic, and symptomatic sanogenetic principles. The result of the treatment of the collapse is largely determined by how much time has passed from the appearance of signs of collapse before the start of therapy: the shorter the period, the lower the degree of disorders of vital functions and the effectiveness of therapy.

Etiotropic treatment

Measures are taken to stop the action of extreme factors and / or reduce the extent of its damaging influence: stop bleeding, injected antitoxin, antidotes, anti-microbial drugs, and others.

Pathogenetic therapy

This is achieved by eliminating or reducing the degree of the effects of non-compliance capacity of the vascular bed, and bcc. For this purpose, patients infused blood products, shelter - or plasma expanders, buffers; administered drugs that increase the tone of the walls of the resistive and capacitive vessels, activating function of the heart and respiratory center; oxygen therapy is performed by inhalation of gas mixtures with high oxygen content or partial hyperbarotherapy; if there are signs of adrenal insufficiency using corticosteroids.

Sanogenetic therapy

Sanogenetic principle of collapse therapy involves the stimulation of adaptation mechanisms: the activation of the hematopoietic-specific and non-specific factors and processes IBN system, detoxifying the liver and other functions, excretory capacity of the kidneys, and others.

Symptomatic therapy

Symptomatic treatment includes measures to eliminate the painful, unpleasant and aggravating the patient's condition manifestations of collapse:. Pain, fear of death, depression, anxiety, etc. To this end, (depending on the specific situation) used antidepressants, antipsychotics, sedatives and painkillers, stimulants, tranquilizers.

Shock

Shock - overall, an extremely heavy condition of the body that occurs under the influence of super-strong extreme factors. Characterized by-step progressive disorder of the body's vital functions as a result of the increasing dysfunction of the nervous, endocrine, cardiovascular and other vital systems.

Output patient of the shock state can usually only during medical emergency and effective measures. Without this shock usually results in a terminal state.

ETIOLOGY SHOCK

An important feature of shock is that it causes extreme high power factor, usually leading to extensive destruction of various structural elements of the tissues and organs.

Causes

- Different versions of injury (mechanical damage - the destruction, breaks, break, crush tissues, extensive burns, electric shocks, etc.).
- Massive blood loss (as a rule, combined with the injury).
- Large volume transfusion of incompatible blood.
- Contact with the internal environment of the body sensitized to allergens.
- Significant extensive necrosis or ischemia organs (heart, kidney, liver, intestines and others.).

Risk factors

For conditions, potentiating the action of extreme factors and promote the development of shock include:

- Hypothermia and hyperthermia,
- Prolonged fasting,
- Nervous and mental excitement,
- Significant physical fatigue,
- General hypo - or hyperergic state
- Chronically proceeding severe disease.
- Individual reaction to this extreme exposure. Practice is rich in cases of shock in a relatively small injury and at the same time the lack of shock at the massive damage to the body.

TYPES OF SHOCK

A common classification of shock there.

The criterion for differentiation states of shock mainly serves the cause. With this in mind, the most common and clinically important types of shock include traumatic shock (wound), burns, post-transfusion, allergic (anaphylactic), electric, cardiogenic, toxic, psychogenic (psychological) and others.

In practical medicine shock states are divided depending on the severity of their course: the shock of I degree (light), shock II degree (moderate severity), III of the degree of shock (heavy).

GENERAL PATHOGENESIS AND MANIFESTATIONS STEPWISE SHOCK

Regardless of the severity of clinical manifestations distinguish two successive stages of developing shock.

- First, there is the activation of specific and nonspecific adaptive reactions. This step was previously called the stage of generalized excitation, or erectile. In recent years, it is called adaptive, compensatory, non-progressive, early.
- If the adaptation processes are insufficient, developing the second stage of shock. Previously, it was called the total braking stage, or torpid (from the Latin torpidus -. Sluggish). At present time it is called stage deadaptation or decompensation. At this stage, identify two substage: progressive (consisting in the depletion of compensatory reactions and tissue hypoperfusion) and irreversible (in the course of which develop changes that are not compatible with life).

Erectile stage

Stage erectile (compensation, non-progressive, adaptation) is the result of significant severity and scale of the damage to the organs and tissues of extreme factors, as well as emerging under his influence, secondary changes in the body.

The basic pathogenesis of shock at the stage of compensation:

Neuroendocrine link

Excessive generalized activation of the nervous and endocrine systems, developing in connection with hyperafferentation different modalities, is characterized by a significant increase in effector effects on organs and tissues from the sympathetic-adrenal and hypothalamic-pituitary-adrenal system, the release into the blood hormones of the thyroid, pancreas and other endocrine glands.

The result is a hyperactive CVS and respiratory system, kidneys, liver and other organs and tissues. Implication:
- Hypertensive reactions
- Tachycardia,
- Increased and deepening of breathing,
- Redistribution of blood flow in different regions of the vascular bed,
- The release of the blood from the depot.

These reactions are adaptive directionality. They provide in conditions of extreme factor delivery to the tissues and organs of oxygen and substrate metabolism, maintaining perfusion pressure.

With increasing degree of damage, these reactions take excessive, inadequate and uncoordinated nature, which greatly reduces their effectiveness. It determines to a large extent or even irreversible heavy aggravate during states of shock.

Consciousness is not lost in shock.

Usually it celebrated nervous, mental and motor agitation, manifested excessive fussiness, agitated speech, increased responses to various stimuli (hyperreflexia).

The degree of functional disorders of organs and tissues, metabolic disorders, their specific biological significance are different depending on the variety of shock and its severity. Specificity of states of shock is detected, as a rule, only at the initial stage of its development. Later, with the growth of the degree and scale of life disorders, leading pathogenetic importance triad of interrelated and co-occurring pathogens: hemodynamic instability, hypoxia, and Toxemia.

Hemodynamic link

Violation of hemodynamics at shock is the result of disorders of the heart, change the tone of resistive and capacitive vessels, reducing BCC, changes in blood viscosity, as well as the activity of the hemostatic system factors.

1. Disorders of cardiac activity.

Causes:
- Direct action on the heart of extreme factors (mechanical trauma, toxins, severe hypoxia, an electric current, etc.).

- Hypercatecholaminemia cardiotoxic effects, high blood hormones of the adrenal cortex and thyroid.

Implication:

- A significant tachycardia.
- Various cardiac arrhythmias.
- Reduction of shock and cardiac output.
- Violations of the central organ, tissue and microcirculation.
- Systemic venous congestion.
- Slowing of blood flow in the microvasculature, most pronounced in the venules.

2. Change the tone of resistive and capacitive vessels.

Under the influence of shock factors of vascular tone at the beginning, tends to increase. The main reason - increased activity of the sympathetic-adrenal system and a significant increase in connection with the level of catecholamines in the blood. For some time, increased tone of the walls of resistance vessels (arterioles) helps to maintain systemic blood pressure.

The consequences of increasing the tone of the capacitance vessels walls (venules)

- Increased blood flow to the heart.
- Increased blood supply, stroke volume, and as a consequence - perfusion pressure.
- Later (due to metabolic changes in tissues and organs) accumulate BAS lowering the tone of the vessel walls (both resistive and capacitive). These substances include adenosine, biogenic amines histamine, N0, PGE, I2.
- Under the influence of extreme factors (especially calling plasma - or blood loss) there is a redistribution of blood flow.

Manifestations of blood flow redistribution:

- Increase in blood flow (or at least no loss) in the arteries of the heart and brain.
- Simultaneous decrease in blood flow in the vessels of the skin, muscles, abdominal organs, kidneys (due to vasoconstriction). This phenomenon is called blood centralization.

Causes of blood flow redistribution:

- Uneven adrenoceptor content in different vascular regions. The highest number found in the walls of the vessel muscles, skin, abdominal, kidney and considerably smaller - in the heart and brain blood vessels.
- Formation of a large amount of myocardial tissue and cerebral vasodilating agents - adenosine, prostacyclin, NO, etc.

The value of blood flow redistribution:

- Adaptive. Blood supply to the heart and brain in the conditions of the phenomenon of centralization of blood circulation helps to maintain the life of the organism as a whole. On the contrary, these organs ischemia significantly exacerbates the shock.
- Pathogenic. Hypoperfusion vessels in the skin, muscles, abdominal organs and kidneys leads to the development in them of ischemia, the current slowdown in the microvasculature (especially postcapillaries and venules), disruption of fluid reabsorption in the venules. The latter causes a decrease in VCB and blood clots.

3. Reduction of the VCB, the change in blood viscosity and activity of the hemostatic system factors are identified at an early stage of shock states.

Implication:

- Increased blood viscosity.
- Reducing its strength.
- Formation of blood clots in the vessels of the microcirculation.
- Improving the content and / or activity of blood coagulation factors. The latter is mainly due to the damaging effect of shock factors agent hypercatecholaminemia, the release of Ca2 + from the damaged cells, and other factors.

Together, disorders of cardiac activity, tone of vascular walls, increasing their permeability and impaired reabsorption of fluid in the bloodstream is initiated by the growing violation of central and regional circulation and microcirculation for all varieties of shock.

The hypoxic link

The hypoxic link of pathogenesis is a major component of the pathogenesis of regular and shock.

Causes:

- Initially, hypoxia is usually the result of hemodynamic disorders and circulatory character wears.
- As the worsening state of hypoxia becomes mixed. This is the result of progressive respiratory disorders, changes in blood and tissue metabolic system.

Effects:

- Reduced the effectiveness of biological oxidation potentiates the disruption of tissues and organs, as well as the metabolism in them.
- Hypoxia is one of the reasons for the suppression of the activity of the antioxidant defense system of factors tissues. In this regard, they generated an excess of reactive oxygen species, lipid peroxidation reactions are activated and accumulate products of these reactions - peroxide, hydroperoxide, etc. and aldehydes.

Toxemic link

Toxemic link activated at an early stage of the shock.

Causes:

- The action of extreme factors (in toxic, toxic and infectious diseases, burns, and other types of anaphylactic shock).
- Damage to the extreme factors of cells and release of these large amounts of unclaimed BAS (kinins, biogenic amines, adenine and adenosine nucleotide, enzymes), normal and disturbed metabolism products, ions, denatured compounds.
- Violation of the inactivation and / or excretion of toxic compounds by the liver, kidneys and other organs and tissues.

Effects:

Increase intoxication potentiates hypoxia, hemodynamic instability, the functions of organs and tissues.

Metabolic link

Causes:

- Excessive activation of neural and humoral effects on tissues and organs,
- Upset hemodynamics in tissues and organs,
- Hypoxia,
- Toxemia.

Effects:

In general, changes in metabolism are characterized by a predominance of catabolic processes.

1. Protein content of the tissue, usually reduced. Simultaneously increasing their level of degradation products.

Reason - significant activation of proteolytic enzymes, under the influence of excess catecholamines, Ca2 +, H +, and other enzymes.

2. The total protein content in the blood is also reduced.

One of the leading causes of: output of low molecular weight protein fractions of blood vessels due to the significant increase in the permeability of their walls.

3. The level of glycogen in tissues is reduced.

Causes:

- Glicogenmobilisation effects of excess catecholamines, glucocorticoid and other hormones;

- Inhibition of glycogen synthesis in the cells due to insulin deficiency;
- A consequence of these processes is hyperglycemia,
4. The residual (non-protein) typically nitrogen in the blood increases.

Causes:
- Activation of gluconeogenesis process;
- Disruption of the liver;
- A decrease of urea excretion by the kidneys,
5. Lipid metabolism is characterized by:
- Intensification of lipolysis in tissues;
- Increase in the content of free fatty acids, which are intensively oxidized, esterified, and subjected to lipid peroxidation;
- A decrease in the level of phospholipids in tissues, indicating the activation of phospholipase;
- Decreasing blood free fatty acids, because they are included in tissue metabolism rapidly (assuming sufficient for this level of ATP in the cells) and transformed into a free radical reactions.
6. Contents of adenine nucleotides (ATP, ADP, AMP) and inorganic phosphate in tissue varies in different ways.
- ATP level therein is reduced as a result of intensive ATP hydrolysis.
- Phosphate concentration drops because of their access to the blood.
- ADP and AMP content in the tissues increases as the ATP hydrolysis increased, and its resynthesis insufficient in oxygen deficit conditions.
7. The level of ions and fluid in tissues as a whole increases.

The reason - cytolemma damage, as well as the membranes of mitochondria and sarcoplasmic reticulum.

Manifestation - increase in the tissues [K +], [Ca2 +], [Na +], osmotic and oncotic pressure and fluid volume.

Aggravation of the above-described complex disorders of the functions of organs and tissues, metabolism and plastic processes in them, as well as the growing depletion of adaptive responses create the conditions for the transition stage adaptation (compensation) in decompensated shock.

Torpid stage

Stage of decompensation (deadaptation, progressive, irreversible, torpid) - as a result of the action of extreme factors and progressive failure of tissue functions, organs, systems, as well as the depletion of the body's adaptive capabilities.

In contrast to the compensation stage, the degree and extent of disorders much more pronounced. Various lesions develop simultaneously and potentiate each other. The most frequently observed decompensation functions of the kidneys, lungs, liver (syndromes "shock kidney", "shock lung", etc.). Under these conditions, organ failure function is extreme. This can cause the patient's death.

Neuroendocrine link

Consciousness at decompensation stage is not lost. However, in severe shock during signs of lethargy and confusion. This is manifested by the fact that the patient is delayed and often inappropriately answers questions, hardly versed in the environment.

The intensity of the effects of nerve and hormone levels:
- A reduced or retained at a higher level;
- The effects of neural and hormonal influences progressively reduced until absence.

Causes:
- Significant damage to the nervous and other tissues.
- Significant physical and chemical changes in the tissues (acidosis, ion imbalance, hyperhydration, and others.).
- Reduced sensitivity (hyposensitization) cells to hormones and neurotransmitters.

- The fall of the severity of conditioned and unconditioned reflexes (hyporeflexia).
- Reduction in blood hormone levels of most endocrine glands.

The totality of these changes leads to an imbalance of neurotransmitters and as the content of hormones in the blood plasma and interstitial fluid and their effects. This is the reason for the disintegration and physical functional systems of the organism that causes discoordination and tends to minimize the functions of organs and tissues.

Hemodynamic link

At the stage of decompensation, hemodynamic shock pathogenesis link becomes crucial.

Causes

- The progressive cardiac dysfunction, the development of his lack of contractile activity and arrhythmias.
- The total decrease in the tone of resistive and capacitive vessels. This eliminates the phenomenon of adaptive circulatory centralization. Systolic blood pressure in severe shock during reduced to 60-40 mm Hg, which is fraught with the termination of the filtration process in the kidney glomeruli and development of uremia.
- Further reduction bcc and increase in blood viscosity due to a yield of the liquid portion of blood into the extracellular space.

Manifestations

- Total hypoperfusion of organs and tissues,
- A substantial disorder of microcirculation,
- Capillarotrophic failure.

Changes in the vessels microvasculature:

- Slowing of blood circulation;
- The appearance in some regions vascular large number of so-called plasma capillaries (in which no blood cells), while others - the capillaries filled with blood cells or aggregates with signs phenomenon sludge and stasis.

Changes hemostatic imbalances are in the development of concentration and / or activity of coagulation factors, anticoagulative and fibrinolytic systems.

Effects

The development at the stage of decompensation of shock:

- DIC,
- Tissue ischemia,
- Degenerative changes in organs and tissues,
- Tissue necrosis,
- Hemorrhage therein.

The hypoxic link

Causes:

- Systemic disorders of hemodynamics,
- Hypoventilation lungs.
- Reduction of the VCB.
- Impaired renal function
- Disorders of metabolism.

Effects:

- The development of severe hypoxia mixed type.
- Uncompensated acidosis.

Under such conditions, exacerbated metabolism disorders, impaired metabolic products accumulate more and more suppressed function of organs and tissues.

Toxemic link

Toxemia is characterized by:

1. The increase of blood and other biological fluids impaired metabolic products and physiologically active substances (e.g. lactic and pyruvic acids, fatty acids, polypeptides, biogenic amines);

2. accumulation of blood in the compounds:

- Released from damaged and disrupted cells (enzyme, denatured proteins, ions, and various other impurities.);
- Formed in the body due to failure of the liver and kidneys indoles, phenols, skatole, urea, uric acid and others.

These substances are contributing significantly to organ damage.

Metabolic link

Metabolic unit shock on decompensation stage appears:

- Dominated processes catabolism of proteins, lipids, carbohydrates, complex compounds (PL, glycoproteins, phospholipids, etc.);
- Minimization of intensity and metabolic processes in cells of the plastic;
- Overhydration cells;
- Accumulation of biological fluids unoxidized substances (lactic acid, pyruvic acid, CT et al.);
- An increase in tissue levels of lipid peroxidation products.

Cell link

Cellular link of the pathogenesis of shock on decompensation stage is characterized by:

- Increasing suppression of the activity of enzymes and cell activity;
- Damage and destruction of cell membranes;
- Violations of cell-cell interactions.

In general, lack of functions of organs and tissue hypoxia, toxemia, gross metabolic disorders cause significant deviations from normal vital parameters of homeostasis, manifested:

- Hypotension and collapse;
- A significant reduction in rates of pO2 and pH;
- An increase in the osmolarity of blood plasma;
- Increasing oppression of life of the organism as a whole.

PATHOGENESIS CERTAIN SHOCK

Features of different types of shock are mainly determined by their cause and nature of the response to her body. Given these two factors, below is a description of the individual and clinically relevant variants of shock.

Anaphylactic shock

It is a shock that occurs as the pronounced manifestation of anaphylaxis or atopy. Drug anaphylaxis occurs in 1 out of every 2700 hospitalized patients; 0.4-2 deaths per 1,000,000 per year from anaphylactic shock in response to a sting of Hymenoptera.

Etiology.

1. Medicines: antibiotics (especially penicillin) protein preparations (enzymes - trypsin, chymotrypsin; hormones - insulin, ACTH), and vitamin B1, NSAIDs, local anesthetics, drugs used for immunotherapy (allergen antiserum immunoglobulins vaccines).
2. The venom of stinging insects (bees, wasps, hornets).
3. Food products (fish, shellfish, cow's milk, eggs, beans, peanuts).
4. Contact with latex products (gloves, catheters).
5. Patients with cold urticaria at the general supercooling (eg, bathing in cold water) may develop anaphylactic shock Clinic.

6. Sometimes, anaphylaxis can occur for no apparent reason. Episodes are repeated, accompanied by an increase in histamine concentration in blood plasma. In such cases we say about idiopathic anaphylaxis.
7. Genetic predisposition (hypersensitivity to specific antigens).

Risk factors.

The presence of atopic diseases and anaphylactic reactions in the anamnesis.

Pathogenesis

1. The release of histamine with IgE - mediated degranulation of mast cells leads to the expansion of peripheral blood vessels (especially the arterioles), lower peripheral resistance, blood Escrow on the periphery due to increased peripheral vascular and blood pressure fall.
2. Unlike anaphylactic, anaphylactoid reactions occur under the influence of non-immune activators mast cells, such as iodinated X-ray contrast agents, the solutions of dextrans, and polymyxin, tubocurarine, opiates, thiopental, pentamidine, hydralazine, doxorubicin stilbamidina et al.

Manifestations

1. Arterial hypotension, syncope, shock. The interval between the appearance of symptoms of shock and exposure to allergens varies from a few seconds when the allergen injections or insect bite up to 15-30 minutes with ingestion of the allergen.
2. Nausea, vomiting, involuntary urination and / or defecation.
3. Itching, redness, possible urticaria, angioneurotic edema (skin, subcutaneous tissue, mucous membranes) allergic origin.
4. bronchial obstruction.
5. Spastic syndrome.
6. shortness of nasal breathing.
7. Difficulty swallowing (the first sign of laryngeal edema).
8. Expansion of the pupils.
9. Tachycardia.

Treatment. Careful monitoring of vital signs nA throughout the treatment period, and a few hours after relief of anaphylactic shock clinical symptoms may recur within 24 hours

- Principles: increased peripheral vascular resistance, recovery bcc, normalization of AAR and maintenance functions of vital organs. Emergency treatment: epinephrine, diphenhydramine, cimetidine (H2-receptor blocker), glucocorticoids.

Complications: recurrence of anaphylactic shock, shock kidney, liver shock, shock lung.

The course and prognosis: a favorable prognosis with timely emergency care; outlook deteriorates significantly when administered epinephrine later than 30 minutes after the first signs of anaphylaxis.

Toxico-infection- shock

Infectious-toxic shock syndrome - toxic shock in infectious diseases caused by exposure to high doses of the body of toxins and pathogens (or) the decay products of damaged tissues. Develop hypotension, oliguria, tachycardia, tachypnea, fever, microcirculation disorders due to diffuse cell and tissue damage by toxins. The main lesions are caused by endotoxin (thermostable lipopolisahiridny component of the cell walls of microorganisms). Less toxins are the cause of Gram-positive bacteria, viruses and yeasts.

Treatment:

1. Causal treatment

- Antibiotics (preferably appointment bacteriostatic drugs as microbicides, drugs can cause deterioration of the patient due to the rise of endotoxemia).
- Introduction antistaphylococcal plasma and γ-globulin (with a staph infection) or fresh frozen plasma (when unidentified exciter)

2. Pathogenetic therapy.

- Elimination of hypovolemia introduction volume dependent solutions.
- Improve the microcirculation introduction reopolyglucin.
- To maintain vascular tone - dopamine.
- For removal of intoxication - hemodes.
- If necessary - diuretics.
- Cardiac glycosides - indicated.

Cardiogenic shock

Cardiogenic shock - a shock with a sharp decrease in cardiac output and a decrease in oxygen supply of tissues as a result of violations of myocardial (heart attack, hemodynamically significant arrhythmias, dilated cardiomyopathy) or morphological disorders (acute valvular insufficiency, ventricular septal rupture, the critical aortic stenosis, hypertrophic cardiomyopathy). Cardiogenic shock hemodynamically characterized by an increase in end-diastolic left ventricular pressure (pulmonary artery wedge pressure> 18 mm Hg) reduction in cardiac output (cardiac index <2 L / min / m2), an increase in systemic vascular resistance decrease in mean arterial pressure (<60 mm Hg).

Compensatory mechanisms: ADH secretion, release of aldosterone and renin secretion of catecholamines.

Physiological reactions: decreased urine output, leading to fluid overload; vasoconstriction. causing an increase in afterload;

Pathogenesis. In addition to violations of myocardial contractile function, developed in the cardiogenic shock matters pain factor (myocardial infarction and pulmonary embolism). Depending on the pathogenesis and clinical features distinguish the following forms of cardiogenic shock:
- Reflex cardiogenic shock. A crucial role is played by disorders of vascular tone caused by reflex reactions.
- True cardiogenic shock due mainly to impairment of myocardial contractility.
- Arrhythmic shock associated with the occurrence of arrhythmia heart rate.
- Unresponsiveness shock. The term is used in relation to cardiogenic shock, not amenable to drug therapy.

Manifestations. The sharp drop in blood pressure on the background of signs of a myocardial infarction, acute myocarditis, etc. Adynamic Patients complain of severe weakness. Appearance: pointy facial features, pale, cyanotic skin, sticky cold sweat. Breathing palpitations, superficial. Pulse frequent, sometimes arrhythmic, weak filling. An important symptom - oliguria or anuria. In severe cardiogenic shock - loss of consciousness, joining pulmonary edema.

Treatment. Carry out the treatment of the underlying disease causing the cardiogenic shock, including surgery (drainage of pericardial tamponade, pulmonary embolectomy of the arteries, coronary bypass surgery, etc.).

Forecast. Mortality rate - more than 70%.

Burn shock

The cause of burn shock: extensive skin burns (usually more than 25% of its total surface) II and / or III degree. In children and the elderly develop shock occurs at a burn for about 10% of the surface of the skin.

The pathogenesis and manifestations

The main links of the mechanism of burns and traumatic shock similar.

Burn shock leads to:
- Frequent infections and sepsis burn surface development
- Severe toxemia
- Short-term erectile stage for heavy torpid
- Severe pain afferent impulses from the affected zone
- Part of the development of "shock kidney"

- Significant dehydration

Much pain afferent impulses from the burnt skin and soft tissues, which cause a generalized activation of the nervous and endocrine systems.

This leads to the development of a strong emotional, motor and speech excitement, activate the functions of organs and tissues, as well as materials in their metabolism.

The relatively short stage of compensation. Often it goes into decompensation stage before rendering the first medical aid.

The short first stage due to overexertion and the rapid depletion of adaptive reactions of the organism, as well as significant hemodynamic disorders and intoxication.

Severe dehydration resulting from massive loss of blood plasma. The latter is defined by a significant increase in the permeability of the vessel walls, especially in the area of the burn.

Increased blood viscosity, the development of the phenomenon of sludge, thrombosis and microcirculation disorders that develop as a result of a large loss of blood plasma.

Severe intoxication:

- Products of thermal denaturation of the protein, and proteolysis;
- BAS excess produced during tissue injury (kinins, biogenic amines, oligo and polypeptides, and others.);

Frequent damage to the kidneys caused by the violation of their blood supply and hemolysis of red blood cells.

Allergic reactions and diseases of the immune autoaggression. Observed in patients withdrawn from the state of shock and in the development of their burn disease. This is due to the denaturation of blood proteins and tissues as well as infection of the organism. Immunological reactions are contributing significantly to the severity of a burn shock and burn patients.

Septic shock

Septic shock is possible in case of peritonitis, infections of the urinary and biliary tract, pneumonia, necrotizing pancreatitis, septic abortion and childbirth. Most often, septic shock results from gram-negative bacterial action (E. coli, Klebsiella, Proteus), but may be pathogens, and other agents (gram-positive bacteria, viruses, fungi, protozoa).

Synonyms

- Toxic-infectious shock
- Endotoxin shock
- bacteremic shock
- Infectious-toxic shock

Traumatic shock

The reason traumatic shock: massive damage to organs, soft tissue and bone under the influence of mechanical factors (e.g., breakage or crushing of tissues and organs, the gap limb bone fracture and others.). Typically, mechanical injury combined with greater or lesser degrees of wound infection and blood loss.

The pathogenesis and manifestations

Traumatic shock is characterized by significant pain afferentation.

1. Stage of compensation.

Phase compensation for intensity and duration, typically correlates with the scale and the degree of injury: the greater, the shorter this stage, and vice versa. This is explained by the breakdown process of compensatory, protective and reduction reactions.

The patient is excited. Many spoke about the incident with it, tossing in bed, sensitive to touch or attempt to move it, and a lot of random gestures (hyperreflexia).

There have been signs of activation of the sympathetic-adrenal system.

- Pale skin and visible mucous.
- Expansion of the pupils.

- Increased blood pressure, increased heart rate, blood flow velocity.
- The increase in respiratory rate.

These reactions have a definite adaptive value: in terms of damage the body, especially in the presence of blood loss, they are an important component of the general adaptation syndrome and process emergency hypoxia compensation.

Record the increased release of steroid hormones by the adrenal glands. It promotes the use of glucose nervous tissue, myocardium and other organs, as well as the stabilization of blood vessels and cell membranes. The latter prevents excessive increase their permeability and correspondingly reduces the swelling of tissue, degree of thickening of the blood, out of the lysosomes and further from the cells into the extracellular fluid enzymes and other macromolecular compounds, ie, It reduces the level of toxemia.

2. Stage of decompensation.

Stage of decompensation is characterized by:
- Depletion and the breakdown of adaptive reactions of the organism;
- Progressive reduction in the efficiency of neuroendocrine regulation;
- increase failure of organs and their systems.

These changes occur:
- Fall in blood pressure and the development of a collapse (systolic blood pressure can be reduced with mild shock and 90 mm Hg, with an average - up to 70 mm Hg, with a heavy - up to 40-50 mm Hg).
- Increased heart rate (up to 180-210 per minute), its weak content, loss of its individual waves (a sign of cardiac arrhythmias).
- Increased massive exit of fluid from the vascular tissue.
- Reduction of the bcc (below the norm by 25-40% or more) and an increase in Ht.
- Blood hypercoagulability and thrombosis, especially in the microvasculature. Later may develop disseminated intravascular coagulation, thrombosis, fibrinolysis, and hemorrhage.
- The deposit of a large amount of blood in the vessels of the abdominal cavity, lungs, spleen, liver. This leads to a progressive decrease in cardiac output, blood pressure.
- Impaired microcirculation in lung edema them, obstruction of the bronchioles and focal atelectasis, which causes acute respiratory failure. These changes are known as the syndrome of "shock lung."
- A significant reduction in the blood supply to the kidneys, thrombosis and sludge, edema and ischemia parenchyma, form cylinders in the kidney tubules. It causes acute renal failure and uremia ("shock kidney").
- Significant disorders of hemodynamics in the liver. This leads to the development of its total insufficiency (a syndrome of "shock liver").
- Disorder hemodynamics in blood vessels of the mesentery and the walls of the intestine, causing a violation of the digestive tract function with the development of auto-infection and intestinal auto-intoxication.

These and other disorders (without effective medical care) potentiate each other can lead to inhibition of the body's vital functions and death.

Bacteremic shock

Bacteremic shock - toxic shock with bacteremia caused by hitting into the blood large dose of bacterial toxins. Painful shock - shock caused by strong pain stimulation (eg, trauma). Hemolytic shock - a shock that occurs when intense hemolysis (for example, during a transfusion of incompatible blood).

Hemorrhagic shock

Hemorrhagic shock - a kind of hypovolemic shock. There are mild hypovolemic shock (loss of 20% VCB), moderate (20-40% loss VCB), severe (loss of more than 40% of VCB).

Compensatory mechanisms: the secretion of antidiuretic hormone, aldosterone, renin, catecholamines. Physiological reactions: decreased urine output, vasoconstriction, and tachycardia.

Pathogenesis. The adaptation of the patient to the blood loss is largely determined by changes in the capacity of the venous system (containing a healthy person up to 75% of blood volume). However, the ability to mobilize blood from the depot limited to: the loss of more than 10% of VCB begins to decrease central venous pressure, and decreased venous return to the heart. There is a small release syndrome, leading to a decrease in the perfusion of tissues and organs. In response, there are non-specific compensatory endocrine changes. The release of ACTH, ADH and aldosterone leads to delay kidney sodium, chloride and water while increasing potassium loss, and a decrease in urine output. The result of the release of epinephrine and norepinephrine - peripheral vasoconstriction. From the blood off less important organs (skin, muscle, gut), and stored blood supply to vital organs (brain, heart, lungs), ie there is centralization of circulation. Vasoconstriction leads to profound tissue hypoxia and acidosis development. Under these conditions, the proteolytic enzymes of pancreas enter the blood and stimulate the formation of kinin. Recent increased vascular permeability that facilitates transfer of electrolytes and water in the interstitial space. As a result, in the capillaries occurs aggregation of red blood cells, creating a base for the formation of blood clots. This process immediately precedes irreversible shock.

Manifestations. With the development of hemorrhagic shock distinguish 3 stages.

1. Compensated reversible hemorrhagic shock. The volume of blood loss does not exceed 25% (700-1300 ml). Tachycardia moderate AP or not modified or slightly lowered. Desolation subcutaneous veins, decreased central venous pressure. There is a sign of peripheral vasoconstriction: cold extremities. The amount of urine is reduced by half (at a rate of 1-1.2 ml / min).

2. Decompensated reversible hemorrhagic shock. The volume of blood loss of 25-45% (1300-1800 ml). The pulse rate reaches 120-140 per minute. Systolic blood pressure decreased below 100 mm Hg, pulse pressure decreases the value. There is a severe shortness of breath, partly offset by metabolic acidosis respiratory alkalosis, but also able to be a sign of shock lung. Boosts cold extremities, acrocyanosis. It appears cold sweat. The rate of urine - less than 20 ml / h.

3. The irreversible hemorrhagic shock. Its appearance is dependent on the duration of circulatory decompensation (typically hypotension over 12 hours). The volume of blood loss greater than 50% (2000-2500 ml). Pulse exceeds 140 minutes, systolic blood pressure drops below 60 mmHg or not is determined. Consciousness is absent. Develops oligoanuria.

Treatment. In hemorrhagic shock absolutely contraindicated vasopressor drugs (epinephrine, norepinephrine) because they exacerbate peripheral vasoconstriction). For the treatment of arterial hypotension, which developed as a result of blood loss, consistently perform the procedures listed below.

1. Catheterization of the main vein (usually in the subclavian or internal jugular).

2. The jet or drip intravenous blood substitutes (polyglucin, gelatinol, reopolyglucin etc.). Determine the blood group of the patient and its compatibility with the donor blood. Perform blood transfusion.

Fresh frozen plasma transfusions, and if possible - or albumin protein. The total volume of fluid for intensive therapy can be calculated as follows.

- If blood loss of 10-12% of the bcc (500-700 ml) the total amount of liquid should be 100-200% of the volume of blood loss at a ratio of salt and plasma expander solutions - 1: 1.
- With an average blood loss (up to 15-20% of VCB, 1000-1400 mL) compensation in the amount of produce 200-250% blood loss. Transfusion of blood medium consists of (in amount 40% blood loss) and salt and colloidal solutions in the ratio 1: 1.
- When a large blood loss (20-40% of VCB, 1500-2000 mL) total volume transfused liquid is not less than 300% of blood loss. Blood poured into a volume of 70% of the lost. The ratio of salt and colloids - 1: 2.

- If massive blood loss, constituting 50-60% of VCB (2500-3000 mL), the total volume of infusion should be at 300% higher than the loss of blood, with transfused blood volume should be at least 100% of blood loss. Saline solutions and colloids are used in a ratio of 1: 3.
3. Combating metabolic acidosis: infusion of 150-300 ml of 4% sodium bicarbonate solution.
4. Glucocorticoids simultaneously with the start of blood substitution.
5. Removing spasm of peripheral vessels.
6. Inhalation moistened oxygen.
7. Hyperthermia - natural cooling (ice encasing it bubbles), analgin (2 ml of 50% solution) or reopirin (5 ml) deep V / m.
8. Broad-spectrum antibiotics.
9. Maintaining diuresis (50-60 ml / h).
10. Cardiac glycosides.

Transfusion shock

Transfusion shock - a shock that occurs when incompatible blood transfusion as an extreme expression of post-transfusion reactions. Hypovolemic shock - a shock that occurs when the loss of more than 20% of BCC because of the acute bleeding or dehydration.

Obstructive shock

Obstructive shock occurs when a massive pulmonary embolism, cardiac tamponade, atrial myxoma, acute valvular stenosis (eg, prosthetic valve thrombosis) or tense pneumothorax; cardiac output falls sharply due to the reduction of blood flow obstruction or ventricular filling with adequate bcc, myocardial contractility, and vascular tone.

Pleurapulmonary shock

Pleurapulmonary shock - traumatic shock arising in case of damage (including during surgery) in the chest and thoracic cavity due to over-stimulation of the receptors of the visceral and parietal pleura.

Spinal shock

Spinal shock - a temporary sharp drop in excitability of nerve centers located below the level of spinal cord injury, manifested by the weakening of the corresponding spinal reflexes. Toxic shock - a shock caused by the impact on the body of toxic products of tissue decay or bacterial toxins (eg, traumatic toxicosis, bacteremia).

SHOCK TREATMENT

The effectiveness of the treatment of states of shock is largely determined by the time intervals at which it started after exposure to the causative agent: the shorter the interval, the more successful the treatment and prognosis.

Etiotropic treatment

Etiotropic treatment is carried out by eliminating or mitigating the shock genicity factors:
- The impact of the termination of the damaging agent,
- The use of anesthesia and / or local anesthetics.

These measures are aimed at preventing and / or reducing the severity of excessive pathogenic afferentation from pain and other extero-, intero- and proprioceptors. In the treatment of shock, timely and effective causal treatment largely determines the success of the treatment of the victim.

Pathogenetic therapy

Pathogenetic treatment is aimed at breaking the key elements of the mechanism of shock, as well as the stimulation of adaptive reactions and processes (the latter known as sanogenetic therapy).

1. Elimination of disorders of the central, organ, tissue and microcirculation. It is achieved by a complex of measures.

With all the varieties of shock (but especially in traumatic and burn) decreased VCB, disturbed blood supply to organs and tissues, as well as the blood flow in the vessels of the microvasculature. In order to eliminate or reduce the degree of these variations:

- Patients poured blood, plasma and / or plasma substitutes (the latter including high-colloids, preventing fluid into the extravascular output channel);
- Simultaneously (or in the above liquids) use so-called buffer solutions (sodium hydrogencarbonate, etc.), Potassium chloride normalization acid-base balance and liquids containing various ions to remove them imbalance;
- Used vasoactive drugs and cardiotropic allowing normalize myocardial contractile function, vascular tone and eliminate heart failure;
- Use tools that reduce the permeability of the vessel walls: calcium drugs and corticosteroids.

2. The elimination or reduction of the degree of disorder of blood supply to organs and tissues. As a rule, it can reduce the severity of disease functions of most organs and tissues.

3. Eliminating (or reducing the degree) failure of external breathing. Enabled by means of the ventilator, breathing gas mixtures with increased oxygen content, the use of respiratory analeptic.

4. Improve the blood supply of the kidneys, and in severe cases - the use of the apparatus "artificial kidney" (if there is evidence of renal failure and uremia development).

5. Removal of hypoxia, acid-base balance deviations and ionic balance. It achieved typically by normalizing the circulatory, respiratory, function kidney and other organs. However, along with this conduct and special events: breathing gas mixtures with increased oxygen, hyperbaric oxygenation, the introduction of anti-oxidants.

6. Reduction of toxemia. For this purpose, conduct special medical action:

- Hemosorption and plasmapheresis;
- The introduction of antidotes and antivenoms;
- Infusion of colloidal solutions (absorbent toxins), blood, plasma, plasma expanders, diuretics.

The elimination of toxemia in substantially contributes to the normalization of the kidneys, liver, gastrointestinal tract.

Symptomatic therapy

Symptomatic treatment is aimed at reducing the painful and unpleasant sensations, feelings of fear, worry and anxiety, usually accompanied by shock. For this purpose, cardiotropic and vasoactive substances, a respiratory analeptic, various psychotropic drugs (antipsychotic, tranquilizers, antidepressants, sedatives, stimulants, etc.).

Coma

Coma occurring in various pathological processes can be divided into the following groups.

1. Conditional primary CNS (neurogenic). This group includes anyone that is developing at a stroke, traumatic brain injury, epilepsy, inflammation and tumors of the brain and its membranes.

2. Developing for violations of gas exchange.

- Hypoxic. Connected with insufficient intake of oxygen from the outside (suffocation) or impaired transport of oxygen in severe acute circulatory disorders and anemia.
- Respiratory. Due to hypoxia, hypercapnia, and acidosis due to significant violations of pulmonary gas exchange in respiratory failure.

3. The conditioned metabolic disorders with insufficient or excessive production of hormones (diabetic, hypothyroid, hypocorticoid, hypopituitary coma), an overdose of hormonal preparations (thyrotoxic, hypoglycemic coma).
4. Toxogenic coma associated with endogenous intoxication during poisoning, kidney failure and liver (hepatic, uremic coma), pancreatitis, or with exposure to exogenous poisons (coma for poisoning, including alcohol, organophosphorous compound, etc.).
5. Primarily due to loss of water, electrolytes and energy substances (hyponatremic coma at a syndrome of inappropriate ADH production, chlorhydropenic that develops in patients with persistent vomiting, alimentary-dystrophic, or hungry, coma).

Disturbances of consciousness

Comatose states are characterized, above all, a violation of consciousness.

The degree of disturbance of consciousness often plays a decisive role in the outcome of many diseases and pathological processes. Therefore, to determine the state of consciousness - one of the highlights when the patient survey, especially in emergency situations.

The main types of disorders of consciousness.

Disorders of consciousness are usually subdivided into a change of consciousness and the consciousness of oppression.

1. Changes in consciousness - productive forms of impaired consciousness, developing on the background of wakefulness. They are characterized by a disorder of mental functions, perverted perception of the environment and the self, is not usually accompanied by immobility. These include delirium, amentia and twilight disorders of consciousness. They are the leading symptoms of most mental disorders and treated in psychiatry.

2. Depression of consciousness - non-productive forms of disturbance of consciousness, characterized by deficiency of mental activity with a reduction in the level of consciousness, a distinct inhibition of intellectual functions and motor activity.

To determine the degree of depression of consciousness worked well so-called Glasgow scale (Scottish scale). Assessment of the degree of oppression of consciousness produced in points. For example, if 8 points above the patient has a good chance of improvement; less than 8 - life-threatening situation; 3-5 - very likely death (especially if detected fixed pupils).

Classification

To assess the level of consciousness use the following classification.
1. Clear consciousness.
2. Obnubilation - limited waking state; usually associated with sleepiness:
- Moderate (I),
- Profound (II).
3. Sopor - areactivity condition from which the patient can only be removed for a short time with intensive re-stimulation.
4. Coma - areactivity condition from which the patient can not be deduced by the stimulation, even primitive protective reflexes may be absent during a deep coma:
- Moderate (I),
- Deep (II),
- Prohibitive (III).

Disorders of consciousness can be short or long, with light or deep. Transient loss of consciousness observed in syncope, whereas epileptic seizures, it may take a little longer, and at the brain injury - sometimes several hours. Prolonged loss of consciousness usually occurs in severe intracranial lesions, or metabolic disorders.

Types of violations of consciousness

1. Stunning is the result of an increase (by the action of pathogenic factor) excitability threshold. In connection with this stunning characterized by decreased sensitivity to external stimuli.

Implication:

With stunning notes:

- Preservation of consciousness against the background of varying degrees of violations of sequence, logic and clarity of thinking (confusion).
- Physical inactivity.
- Disorientation in situation.
- Increased drowsiness (somnolence). Strong stimuli (sound, light, pain) only temporarily withdrawn from the patient's state of stunned.

Status stun often preceded by stupor.

2. Sopor condition characterized by general inhibition of psychic activity, significant inhibition of consciousness (but not its complete loss unlike coma), loss of voluntary movements, while maintaining reflexes (unlike coma) on strong sound, light and painful stimuli. The latter is usually expressed transient motor responses, moan, movement of facial muscles.

Often considered stupor stage of coma, loss of consciousness prior (ie, the actual development of the coma).

3. Delirium is characterized by:

- False affective perception of the environment and events, its own role in them (delusions);
- Spontaneous endogenous visual and / or auditory sensations (hallucinations);
- Verbal and motor excitation.

In a state of delirium the patient is actively involved in the events they perceived (he can attack, defend, escape, and clearly describe "visible" images of them, "leads a conversation" with an absent interlocutor).

4. Amentia characterized by:

- Incoherence (disrupted character) thinking;
- Violation of orientation, perception of surrounding objects, events, and self;
- Chaotic, disorderly excitation;
- Unfocused physical activity.
- In case of recovery the patient does not remember (amnesia) of what happened to him during amentia.

5. Twilight state of consciousness characterized by:

- Violation of orientation in the environment;
- Detachment from the place of real events;
- Behavior based on hallucinations (usually intimidating character);
- Sudden onset and termination;
- Often committing aggressive acts.
- Episode amnesic twilight state.

6. Stupor

From the different types of violations and loss of consciousness must be distinguished stupor. When stupor consciousness is not lost. Stupor - a condition characterized by complete immobility, the weakening or lack of reaction to external sound, light and pain stimuli on the background of the saved consciousness.

Stupor often develops in patients with mental health (eg, schizophrenia), as well as with severe somatic (eg, in patients with severe malabsorption syndrome) disease. Stupor is also observed in a number of depressive states (for example, after the loss of a loved one) and strong psychogenic traumas, developing under the influence of various extreme factors.

CAUSES OF COMA

This causes a variety of factors. They are usually divided into exogenous and endogenous. The latter can be infectious and noninfectious.

Exogenous factors

Exogenous factors - pathogenic environmental agents, usually of great strength, toxicity and / or destructive nature.

- Various traumatic (usually brain) factors (electricity, mechanical trauma to the head).
- Thermal treatment (hyperthermia, heatstroke, hypothermia).
- Significant changes in barometric pressure (hypo - and hyperbaric).
- Neurotrophic toxins (alcohol and its surrogates, ethylene glycol, toxic doses of drugs, sedatives, barbiturates, etc.).
- Infectious agents (neurotropic viruses, botulinum and tetanus toxins, pathogens of malaria, typhoid, cholera).
- Exogenous hypoxia and anoxia.
- Radiation energy (large doses of ionizing radiation).

Endogenous factors

Endogenous factors leading to the development of the coma, the result of severe disorders of vital activity. They observed an unfavorable course of various diseases and disease states. These conditions lead to significant deviations from normal vital parameters and constants, excess or deficiency of metabolic substrates and / or oxygen in the body.

- Pathological processes in the brain (ischemic stroke, tumor, abscess, swelling, etc.).
- Lack of blood circulation (cerebral hypoxia).
- Respiratory failure (the brain during hypoxia status asthmaticus, asphyxia, pulmonary edema).
- Pathology of the blood system (massive hemolysis, severe anemia).
- Endocrinopathies (hypoinsulinism, hypo - and hyperthyroid condition, adrenal insufficiency).
- Hepatic insufficiency, disorders of the digestive system (malabsorption syndrome, intestinal autointoxication, and / or self-infection).
- Kidney failure.
- Comatose states are developing in a number of cases in severe progressive course of collapse and shock.

GENERAL PATHOGENESIS AND MANIFESTATIONS

Pathogenesis comatose states, regardless of their causes, including some key links.

Hypoxia, a violation of processes of power supply cells, intoxication, acid-base balance disorder, an imbalance of ions and fluid disturbances electrogenesis imbalance content blockers and their effects are developed in all organs and tissues.

However, the greatest extent they are expressed in the brain. That is why the obligatory sign of the coma is a loss of consciousness. Damage to other tissues and organs, severe disorders of the neuroendocrine regulation of their functions, are responsible for the progression of multiple organ failure and increasing the oppression of life of the organism as a whole.

Common manifestations of comatose states

Organs and systems	altered function
Nervous and Endocrine	Disorders of consciousness, loss of consciousness, hypo - and arephlexia imbalance BAS and their effects
Cardio-vascular system	Heart failure, arrhythmias, hypotension and collapse, blood flow redistribution, capillarotrophic failure
Lungs	respiratory insufficiency

blood system and hemostasis	Deposition of blood, changes in blood viscosity syndrome trombohemorragic
Liver	Liver failure
kidneys	kidney failure
Digestion	Lack of cavity and membrane digestion, intestinal autointoxication, autoinfection

Hypoxia and violations of the processes of power supply

Upset oxygen supply to tissues and organs is the cause or coma, or its pathogenetic link. Violation observed with substrate causes cell failure ensure biooxidation therein.

1. ATP Resynthesis in brain neurons is provided mainly due to the oxidation of glucose energy in the reactions of tissue respiration. Neurons in the brain that are the norm in most oxygen-structures under conditions of hypoxia are the most vulnerable object in the body.

- In the brain mass is about 2% of body weight, accounting for about 20% of the cardiac output of blood. However, the brain (and heart) no stocks of ATP. In this connection, the termination (or decrease) in the oxygen delivery to the brain and / or metabolic substrates precludes its normal functioning.
- Termination of the cerebral circulation after 8-10 to lead to critical shortages of oxygen and energy supply disturbances neurons. The result is a loss of consciousness.
- Comes within the next 4-7 minutes depletion of glucose, as well as suppression (due to the increasing acidosis) anaerobic metabolism accompanied by irreparable expenditure of ATP energy. In this regard, the activity is inhibited by specific neurons lost consciousness and begin to develop rapidly progressive degenerative processes.
- Disintegration in neurons large molecular organic compounds, as well as excess accumulation in Na + and other ions leads to a significant increase in intracellular osmotic and oncotic pressure. This in turn leads to overhydration nerve cells, combined with the fluid outlet of the container in the interstitium (ie, brain edema), venous hyperemia and hemorrhage in the brain substance.
- Even while maintaining cerebral blood flow at the level of about 20% of normal develop delirium, stupor or coma.
- Brain Neurons are damaged to a greater extent in ischemia than hypoxemia. With normal perfusion pressure vessels in the brain, even at lower paO2 to 30 mmHg and below, no signs of neuronal necrosis.
- Damage to cells in a coma is aggravated due to the disorder of transport processes ATP energy from the places of their production in the mitochondria (in the process of tissue respiration) and cytosol (during glycolysis).
- Violation of the energy supply of cells, ultimately, causes their dysfunction, degeneration and the development of the disorder plastic processes in them. To the greatest extent this is expressed in the brain and heart. In this regard, in patients who are in a coma, lost consciousness, reduced severity or absent reflexes; develop arrhythmia and contractile function of the heart failure and hypotension; disturbed frequency and operation frequency of the neurons of the respiratory center, decreases the amount of alveolar ventilation, which leads to worsening of respiratory insufficiency and hypoxia.

Intoxication

Coma of any origin characterized by the accumulation in the body of toxic substances. They enter the body from outside (at coma exogenous origin) and formed in himself (with endogenous coma). A number of comatose states cause neurotropic toxins, alcohol and its surrogates, ethylene glycol, fungal toxins; Drugs when used incorrectly (for example, narcotics, barbiturates, tranquilizers).

- Toxic substances and their metabolic products have the most pathogenic effect on the neurons of the stem and the cerebral hemispheres of the brain, endocrine glands, heart, liver, kidneys, blood cells. Toxins damage the membrane structure of cells and enzymes. In connection with this function is inhibited neuronal cortical and subcortical structures. This in turn leads to disorders of the

cardiovascular, respiratory, endocrine and digestive system, kidneys, liver, blood systems, hemostasis, and others.

- Intoxication metabolic products aggravates the violation of the liver detoxification and excretory activity of the kidneys. Thus, when a diabetic coma significantly increased blood levels of CT, lactic and pyruvic acids. Thus, for example, an excess of acetoacetic acid substantially inhibits the activity of brain neurons and autonomic ganglia.
- When hepatic coma blood levels of putrescine, cadaverine, phenol derivatives significantly increased, indole, skatole (formed during the decay of the large intestine of protein) and ammonium compounds (carbon dioxide and ammonium carbamate, ammonium hydroxide). Normally these compounds are inactivated at the liver and excreted from the body by the kidneys. However, the liver and / or kidney failure called toxic compounds and their derivatives potentiate the damage to the brain and other organs, exacerbating the patient's condition.

Disorders acid-base balance

Deviation indicators acid-base balance - a natural phenomenon in a coma from any source.

1. In most cases, acidosis develops.

Causes:

- Hypoxia circulatory, respiratory, hematic and tissue type.
- Impaired renal function (inhibition acid- and ammoniogenesis, reduced their excretory functions).
- Disorder of the liver (the suppression of the inactivation process, the CT). This increases the degree of acidosis.

2. Much less frequently and tend to temporarily registers the development of alkalosis (eg, during hyperventilation lungs or liver coma, accompanied by a significant increase in blood ammonium ions).

Imbalance ions and water

Violation of the content and the relationship between the individual ions in the cytosol, cell-cell and other biological fluids is an important part of the pathogenesis of coma, particularly when it heavy flow.

Manifestations

- Loss of K + cells.
- The development of hyperkalemia.
- Increase in the cells [H +].
-Increase Intracellular [Na +].
- Hyponatremia.

These changes are a consequence of decreased activity of Na +, K + - ATPase plasmolemma and damage cell membranes.

- Reduction of [Cl] and / or [HCO-3].
- Some versions of coma (eg, renal or hepatic) are characterized by different changes in the ionic balance. The above options are comatose states may be accompanied by an increase in blood levels of aldosterone (due to its increased synthesis in the adrenal glands or the reduction of inactivation in the liver), which determines the reabsorption of Na + and K + excretion in the kidney tubules to the development of hypernatremia and hypokalemia, respectively.
- Hyperosmia - and hyperoncia. It is the result of hydrolysis large molecular compounds (LP, proteoglycans, glycogen and others) to molecules of small and medium-sized (proteins, amino acids, glucose, MC).

Effects

- Hyperhydration marrow and other organs (for diabetic hyperosmolar coma, in contrast, developed hypohydration cells potentiates their damage).
- Increased fluid content in the intercellular space.
- An increase in the volume of fluid in the bloodstream (hypervolemia).
- Swelling of the brain and lungs.

- Diarrhea, vomiting, polyuria (eg when hypocloremic, diabetic, hyperosmolar coma). They can cause progressive extracellular first, and then a total hydropenias.
- Significant increase in blood viscosity.
- Violation of organ and tissue and microcirculation.
- Disseminated aggregation of blood cells, it hypercoagulability and thrombosis (DIC).

Violations electrogenesis

Implication:

The breakdown process of power supply cells, damage to their membranes and enzymes naturally cause disturbances:
- The formation of membrane potential and action potential.
- Excitability (decrease, increase);
- Conduction of excitation. To the greatest extent it is manifested in the structures of the brain and heart.

Effects:
- Disturbances of consciousness, until his loss.
- Seizures.
- Disorders of the nervous centers (especially respiratory and cardiovasomotor).
- The development of cardiac arrhythmias, including ventricular fibrillation.

Imbalance BAS and their effects

Implication:
- Violation of the synthesis and release of biologically active substances of different classes of cells: neurotransmitters, hormones, cytokines, and others.
- Activation of disorder, inactivation, delivery of biologically active substances to the target cells.
- Disruption of interaction of BAS with their cellular receptors.
- Upset response of the target cells. This is caused by damage to cell membranes and intracellular mediators implementation hormone effects of mediators and cytokines.
- The collapse of the physiological and functional systems.
- Minimization of functions of organs and tissues, energy expense and plastic processes.
- The transition to the so-called metabolic regulation of the functions of organs and tissues. Usually it is preceded by the development of a terminal condition.

FEATURES PATHOGENESIS OF SOME COMATOSE

The specificity of certain types of coma is usually detected at an early stage of its development. At these stages occur more especially the causes of coma and of initial units of its pathogenesis. With increasing severity of comatose states reduced their specificity and increasingly manifest their common traits.

Traumatic coma

Cause: trauma, accompanied by severe cerebral concussion and loss of consciousness. Unconsciousness in traumatic coma can last from several minutes to a day or more.

Manifestations
1. The motor responses and eye opening to painful stimulus is absent or greatly reduced.
2. It is absent or the patient emits inarticulate sounds.
3. Hypo - or areflexia.
4. Breathing and heart rate are violated.
5. BP and BCC reduced, even if there was no blood loss.
6. Frequent vomiting.
7. Involuntary urination.
8. In connection with the brain injury, focal hemorrhages and edema in his taped neuropathological symptoms: paralysis (usually hemiparesis), pathological reflexes, local sensitivity disorders, convulsions.

9. The liquor usually blood observed.
10. At the turn of the bones of the skull base there specific features:
- Symptoms of damage to the nuclei of neurons VII and VIII pairs of cranial nerves;
- In orbit bruising ("points" symptom);
- Bleeding and outflow of cerebrospinal fluid from the ear passages, nose, mouth.

Apoplectic coma

Causes

- Bleeding in the brain.
- Acute local brain ischemia with the end result in a heart attack (thrombosis or embolism, major brain artery). The last condition is called stroke (due to the rapidly growing changes in the brain and the body's vital activity disorder).

Risk factors

- Hypertension (especially periods of hypertensive crises).
- Atherosclerotic changes in cerebral vessel walls.

Pathogenesis

The leading pathogenetic factors of apoplectic coma are:

- Ischemia and hypoxia of the brain (as a result of local or widespread disorders of blood circulation in it);
- A significant increase in the permeability of microvessels wall;
- Rapidly increasing swelling of the brain substance.
- For stroke characteristic of the secondary disorders of blood circulation around the ischemic brain region with a rapidly increasing signs of loss of sensation and movement.

Manifestations

1. Apoplectic coma due to a brain hemorrhage:
- The patient suddenly loses consciousness;
- His face (in typical cases) purple;
- Visible blood vessels dilated and pulsate noticeably;
- Pupils do not react to light;
- Tendon reflexes reduced or absent (hyporeflexia), there are pathological reflexes (Babinski et al.);
- In connection with the damage and irritation of the brain substance intensively growing respiratory distress (it is noisy, raucous);
- Violated swallowing;
- Marked hypertensive response and bradycardia.

2. When apoplectic coma as a result of ischemic stroke is usually observed:
- Recurrent episodes of rapid passing of vertigo;
- Unsteady gait;
- Violations of speech;
- Sensitivity disorders;
- Sometimes - syncope (these disorders are the result of transient disorders of blood circulation in the vessels of different brain regions with the development of its transient ischemia);
- Disorders of consciousness, until his loss;
- Hypotension;
- Bradycardia;
- Cardiac arrhythmias;
- A rare shallow breathing;
- Pale and cold skin, and mucous membranes;
- After prolonged ischemia (depending on the affected area of the brain) are identified: hyporeflexia, movement disorders, sensory disturbances.

The consequences of a brain hemorrhage or ischemic stroke. They are different and depend on:

- Scale and topography of lesions,
- The degree of hypoxia and brain edema,
- The amount of damage to homes,
- The severity of hypertension,
- Atherosclerosis severity,
- Age of the patient.

Apoplectic coma refers to the most unfavorable flowing coma, fraught with the death or disability of the patient.

Hypochloremic coma

Reason hypochloremic (chlorhydropenic, chloroprivic) coma - a significant loss of body chlorinated substances. This occurs when:
- Long-term repeated vomiting (in patients with endogenous intoxication, food poisoning, toxemia of pregnancy, pyloric stenosis, intestinal obstruction);
- Incorrect treatment with diuretics;
- A long salt-free diet;
- Renal failure on its polyuric stage;
- Fistulas of the small intestine.

Considering that at the above conditions relatively slowly lost Cl, Na + and K +, as well as compensate for the effects of adaptive mechanisms in the coma typically develops gradually.

Implication:
- Disturbances of MT and AP due to a decrease in blood plasma and other extracellular body fluids content Na +, K +, Cl- and other ions.
- Disorders of cell excitability.
- Violations of the specific and non-specific cell functions. As a result, develop: muscle weakness, lack of exercise, lethargy, rapid physical and mental fatigue, dizziness, and sometimes convulsions, constant thirst, hypohydration.

Due to the loss of body fluids:
- Skin and mucous membranes dry,
- Reduced tissue turgor,
- The facial features are sharp,
- Dry tongue,
- Develops oliguria,
- Ht significantly increased,
- BP is usually reduced,
- BCC reduced
- Ion imbalance,
- Circulatory disorders of the brain.

Brain Ischemia leads to growing frustration of consciousness: stunned state becomes psychomotor retardation and loss of consciousness ends.

Therapies coma

I. Etiotropic treatment is essential. It largely determines the prognosis of the patient. In this regard shall take measures to stop or mitigate the pathogenic action of the causal factors.

1. Traumatic coma:
- Eliminates the damaging factor,
- Used painkillers, local anesthetics,
- If necessary – narcosis

2. When a coma caused by intoxication of the organism used:
- Specific antidotes,
- Antitoxins,

- Gastric lavage,
- Diuretics,
3. When a diabetic coma:
- The calculated insulin dose is administered,
- If necessary, together with glucose solution (for the prevention of hypoglycemic coma).
4. When used antibacterials coma infectious origin (antibiotics, sulfonamides, and antiseptics, acting on the intestinal flora and urinary tract).
II. Pathogenetic therapy is key to the treatment of any patient in a coma.
It includes measures aimed at the blockade, the removal and / or weakening of the damaging effects of the basic pathogenesis of coma: hypoxia, intoxication, disorders acid-base balance, an imbalance of ions and fluid, BAS and their effects.
1. The anti-hypoxic therapy:
- Mechanical ventilation.
- Breathing gas mixtures with increased oxygen content.
- Hyperbaric oxygen therapy.
- Introduction of antioxidants (e.g., glutathione agents, selenium, SOD, catalase, and ubiquinone al.).
- Normalization of the heart and vascular tone.
2. The elimination or reduction of the degree of intoxication by:
- Blood transfusion, plasma, and plasma substitutes, physiological sodium chloride solution.
- The introduction of solutions containing organic compounds large molecular - polyglucin, reo polyglucin etc. These drugs combined with diuretics to stimulate excretion of body fluids and being of toxic substances;
- In severe cases, as well as in renal insufficiency, uremic coma - hemodialysis and peritoneal dialysis.
3. Normalization of indicators AAR, the balance of ions and fluid by:
- Controlled administration of a buffer solution with the necessary (for each patient is chosen individually to the laboratory data into account) the content and ratio of various ions;
- Blood transfusion, plasma, plasma substitutes.
4. Normalization of BAS and their effects. Used for this purpose:
- Adrenal hormones (glucocorticoid and mineralocorticoid, androgenic steroids, catecholamines);
- The pancreatic hormones (insulin, glucagon);
- Neurotransmitters (acetylcholine, norepinephrine), and others.

These preparations normalize the function of the heart, kidney, brain and other organs homeostasis indicators, activate the adaptive specific and nonspecific reactions.
III. Symptomatic therapy is aimed at optimizing the functions of organs and systems, the elimination of seizures, pain, painful sensations in the pre- and post coma periods. Used for this purpose:
- Anticonvulsants;
- Analgesic agents (including drugs);
- Cardiotropic and vasoactive drugs;
- A respiratory analeptic.

Given that the coma is characterized by severe disorders of functions of organs, systems, regulatory mechanisms of the body, the effectiveness of therapeutic interventions should be monitored permanent registration status of the vital functions (cardiac, respiratory, renal excretory function, etc.). Consciousness, and homeostasis parameters.