

**MINISTRY OF HEALTHCARE OF UKRAINE
HSEEU "Ukrainian Medical Stomatological Academy"**

"Approved"
at the meeting of internal
medicine №1 department
Head of Department
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**GUIDELINES
FOR STUDENTS
INDEPENDENT WORK
IN THE PRACTICAL CLASSES PREPARING**

<i>Academic discipline</i>	Internal medicine
<i>Module</i>	Emergency conditions in clinic of Internal Medicine
<i>Content module</i>	Emergency conditions in clinic of Internal Medicine
<i>Study subject</i>	Curation of the patient with shock
<i>Course</i>	VI
<i>Faculty</i>	of foreign students training

Poltava 2016.

1. Relevance of the topic:

Currently, there are over 100 definitions of shock. This is due primarily to the continuous evolution of concepts of the pathogenesis of shock and its mechanisms of development.

It is important to emphasize the most essential characteristics of the shock. Firstly, the shock - a typical pathological process, as in the pathogenesis of shock of various etiologies (traumatic, burns, bleeding, etc. ...) are more similarities than differences, which allows us to consider it as a stereotypical component of the body's reaction to the impact of extreme factors of the external and internal environment. Second shock - is evolutionarily formed pathological process. Thirdly, the shock has a certain phase of development. The main element in the development of shock is the disparity between the intensity of metabolic processes in organs and tissues іh circulatory zabezpechennyah, the equivalent of which is specific to the restructuring mikrogemotsirkulyatsii system, accompanied by the emergence of oxygen debt. Traumatic shock - it decompensation of vital functions in trauma, the range of which, on the one hand, exceed the protective capacity of the organism, on the other - is limited to injuries incompatible with life. You can not equate the typical pathological processes - shock and dying. As you move from one process to another shock specificity decreases. For the different phases and periods of shock are characterized by some non-specific phenomena: the centralization of circulation, autogemodilyutsiya, ekstravaskulyarny translocation, hyperviscosity syndrome, disseminated intravascular coagulation.

2. Specific objectives.

The student should know:

- Determination of shock
- Etiology of hypovolemic shock.
- The pathogenesis of hemorrhagic shock.
- Classification of shock on its severity.
- multiple organ failure with hypovolemic shock.
- Clinical shock criteria.
- Laboratory shock performance.
- A diagnostic program for shock.
- Differential diagnosis of hypovolemic shock.
- General provisions for the treatment of hypovolemic shock.
- Indications for transfusion.

3. Basic knowledge, abilities, skills, necessary for a study themes.

(Interdisciplinary integration)

№	Names of previous disciplines	The skills
1	Anatomy	Anatomical features of the structure of cardiovascular system
2	Physiology	Physiological characteristics of cardiovascular system
3	Pathophysiology	The etiology and pathogenesis of the various com
4	Biochemistry	clinical-laboratory and instrumental investigations
5	Propaedeutics of Internal Diseases Symptomatology	Clinical variants of the course. Own methods of inspection of patient
6	Pharmacology	Principles of treatment, recipe

4. Tasks for independent work during preparation for classes

4.1 A list of key terms, parameters, characteristics which the student has to learn in preparation for the occupation.

№	Термин	Определение
1	Shock	is a severe disease process, accompanied by the depletion of the vital functions of the body and leading it to the brink of life and death because of the critical reduction of capillary blood flow in the affected organs.
2	Depending on the causes of the following types of shock:	traumatic <ul style="list-style-type: none"> • hemorrhagic • burn • turnstile (develops after removing the harness after four hours or more after application) • dehydration • cardiogenic • pancreatic • septic • infectious-toxic • anaphylactic
4	Depending on the primary mechanisms underlying the pathogenesis of shock release:	1) hypovolemic shock (hemorrhagic, angidremichny); 2) shock associated with impaired heart pumping function (cardiogenic); 3) vascular forms of shock (anafilaktichny, pancreatic); 4) complaints of pain, which interferes with the central regulation of blood circulation (traumatic, burn).

4.2 Theoretical questions to the lesson

- How is the shock index Algovera?
- How to define the HPC?
- Normal performance CVP.
- Identify the most important criteria for shock.
- Blood loss more than 1500 ml. - To what extent the shock of gravity applies?
- What are the indicators of hemoglobin and hematocrit in the acute bleeding are indications for transfusion?

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4.3 Practical tasks that are performed in class

- Collection of complaints, anamnesis of patients with hemorrhagic shock.
- Collection of complaints, anamnesis of patients with gipovolemichnim shock.
- In what sequence occurs when blood flow reduction gipovolemichnomu shock?
- To be able to provide emergency aid for the main types of shock;

Content of the topic:

The concept of "shock" changes all the time, along with the notion of the process of the body's vital functions. In the past, content with the definition of shock as a kind of reaction to the effect of extreme stimuli, accompanied by hypotension. Now the shock is understood as a symptom of disorders of vital body functions, arising from inconsistencies between the tissue blood flow and metabolic needs of the tissues.

Underlining this characteristic circulatory disorders, shock is called a "crisis of microcirculation." But such a definition is not final. Despite the fact that hypoperfusion leads to tissue hypoxia, P. L. Marino (1998) is considered inadequate oxygenation of tissues of the central problem of shock. If we further develop these ideas, it is legitimate to consider the final destination in the definition of shock following the violation of central hemodynamics and tissue hypoperfusion violation of tissue metabolism.

Etiology of shock can be varied, depending on which there are several types of it: the traumatic, hemorrhagic, burns, gipohloremichesky, ekzotoksichny, cardiogenic, septic, anaphylactic, and the like.

Pathogenesis of shock at first largely varies depending on the cause, but over time the pathophysiological changes of various kinds come together, and in the final stages of a shock becomes quite similar features. Thus, the starting torque shock from dehydration is a shortage of BCC (primary hypovolemic shock). In embolism of the heart and pulmonary arteries impaired pumping function of the heart (cardiogenic shock). Septic and anaphylactic arises from the reduction of tone and peripheral venous vessels (vascular or vasogenic shock).

But such a shock pathogenetic systematization quite sketchy. In practice, different etiology forms have mixed pathogenesis. So, traumatic shock can be both primary gipovolemichnim (blood loss, plazmovtrata), cardiogenic (contusion of the heart, the effect of "myocardial depression factor" endogenous intoxication during prolonged compression syndrome), vascular (pain reflex influence kinins).

Then all kinds of shock combined in its pathogenesis, as a result of reduction in blood flow to the heart or violations of the pumping function decreases cardiac output. Its decrease in body responds protective reaction in the form of so-called adrenergic circulatory centralization. It is most pronounced in primary gipovolemichnomu less - in cardiogenic, septic, virtually non-existent - in anaphylactic shock.

The purpose of this reaction - to maintain cardiac output, blood pressure and blood supply to organs priority, which currently play an important role in the preservation of life. Included is this special adaptive response of the signal, which serves as a baro irritation - and volyumoretseptoriv laid down in the aortic arch.

The receptors respond to a decrease in cardiac output through the hypothalamus and neurohypophysis that produces kortikotropny hormone, increases the activity of the sympathetic division of the autonomic nervous system with the release of catecholamines in the blood.

Acting on .beta.-adrenergic receptors of the heart and a-adrenergic receptors in the vascular wall, adrenaline and noradrenaline increase the heart rate and peripheral resistance, thereby maintaining for some time a normal cardiac output and normal blood pressure. However, vasoconstriction is not uniformly captures all peripheral vessels, but only those which have a receptor. As a result, there is centralization of circulation, that is, the redistribution of blood that was left, with the predominant flow to the heart and brain by decreasing the blood supply to the organs, which are for the survival of the organism in the acute period of less importance (liver, pancreas, intestines, kidneys, skin, muscle). This simpatoadrenergichna reaction initially is suitable, as it provides a normal level of blood flow in the coronary vessels of the heart and vessels of the brain. But if the bcc quickly returns to normal, then there is a risk of ischemic lesions of the bodies by which the centralization of circulation.

Classification of shock

Hypovolemic: bleeding, dehydration.

The redistributive: septic, anaphylactic, neurogenic, gipoadrenalovny.

Cardiogenic: myopathic, mechanical, arrhythmic.

Extracardiac obstructive: cardiac tamponade, massive pulmonary embolism (PE).

Stage

Preshock.

Early (reversible).

Intermediate (progressive).

Refractory (irreversible).

Classification:

Varieties and causes shock:

1. Traumatic shock:

a) as a result of mechanical trauma (wounds, fractures, compression of the tissue, etc.).

b) as a result of a burn injury (thermal and chemical burns)

c) as a result of the low temperature - cold shock

g) by electric shock - an electric shock

2. Hemorrhagic or hypovolemic shock:

a) bleeding, acute blood loss;

b) acute impairment of water balance - dehydration.

3. Septic (bacterial-toxic) shock (widespread suppurative processes caused by gram-negative or gram-positive microflora).

4. Anaphylactic shock.

5. Cardiogenic shock (myocardial infarction, congestive heart failure).

Septic shock was previously associated with the direct action of bacterial toxins and decomposition products that are formed under their influence. Now it is generally accepted that only bacterial exotoxins - protein toxins (diphtheria, botulinum, and the like) - are able to directly cause irreversible necrotic changes in the patient's cells. Endotoxins - bacterial cell wall lipopolysaccharides - by themselves do not cause cell necrosis. They only stimulate the patient's cells, mainly macrophages, substances - cytokines, inflammatory mediators that lead to the defeat of his same body cells (a kind of "cannibalism"). Cytokines induce the so-called systemic inflammatory response syndrome (SIRS; Systemic Inflammatory Response Syndrome - SIRS), manifestations of which is vasodilatation and blood pressure drop. Vasodilatation caused by nitric oxide (NO) - one of the strongest sudinorozshiryuyuchih agents.

Generalized reaction of the organism in the form of SIRS and the clinical picture, similar to septic shock, may also develop under the influence of a large array of dead, damaged tissues, as in burns, pankreonekrozi, serious injury, blood loss. It turned out that it is impossible to differentiate clinically pathological conditions due to infection by the changes taking place

under the influence of tissue breakdown products. Obviously, described the mechanisms of septic shock characterized by shock genicity and other factors, such as anaphylactic.

- If anaphylactic shock as a result of the release of histamine into the bloodstream and gistaminopodibnih substances lose their tone as a peripheral, and bulk of the veins. Sharply reduced peripheral resistance and decreased blood pressure. There is a mismatch between the available volume of the circulating blood volume and vascular and venous blood return is sharply reduced. It reduced contractility of the heart muscle, so the blood pressure continues to decrease. Simpatoadrenergichna reaction does not occur as a reaction to irritation pretty broken. Only in the future, if the patient survives, vasodilation changes to vasoconstriction.

Regardless of etiopatogenetichnih features shock inevitably decreases capillary blood flow. As a result of this change of oxygen and energy to the tissues of substances, the metabolism in the cells with accumulation of lactic acid acidosis and development. Under the influence of the last precapillary vessels dilate, decentralized circulation and increases blood clotting. Bloodstream further slows, blood accumulates in the capillary vessels, which increases in hydrostatic pressure and plasma passes into the interstitium.

In slowly flowing blood and zguscheniy happening aggregation formed elements increases viscosity. All this leads to an almost insurmountable resistance to blood flow, then the formation of microthrombi. In extreme cases blood flow in tissue stops completely. This process of intravascular coagulation. Such a violation of microcirculation is characteristic of all types of shock, and regardless of the reasons leading to the disruption of cell function. In the cells of energy production decreases, deteriorating the function of cell membranes, resulting in specific organ functions are reduced or terminated.

Particularly sensitive to shock lungs, kidneys, liver. Functional disorders of these organs (designated as "shock organs" - "easy to shock," "kidney in shock," "shock liver") are recovering from the shock elimination. If the shock effect of these bodies is a long-term, then they come necrotic changes that occur after the removal of the shock failure or lack of function of these organs. In this case we speak of "shock lung", "shock kidney" and "shock liver", united by the term "multiple organ failure" (MODS).

Clinical signs of shock is appropriate to consider depending on the etiological varieties.

- Hemorrhagic shock develops as a result of primary hypovolemia. Primary-gipovolemichnim there is also a shock for burns and dehydration.

Pathogenesis concluded that there is an adaptation of the organism to hypovolemia venous system by changing capacitance, which in a healthy person contains about 75% of BCC. However, the possibility of raising the blood from the venous depot limited. If you lose 10% of BCC and more begins to fall in CVP, decreases venous return to the heart, there is a "small release syndrome", leading to reduced tissue perfusion. In response occurs centralization of blood flow, are excluded from it less important organs (skin, muscle, n zone. Splanchnicus intestine, kidney, liver) and stored blood supply to vital organs (brain, heart, lungs). Vasoconstriction leads to tissue hypoxia and acidosis development. Under these conditions, the blood comes pancreatic proteolytic enzymes and stimulate the formation of kinins that increase vascular permeability and water and electrolytes transferred into the interstitium. As a result, in the capillaries occurs aggregation of blood cells, the conditions for thrombus formation. This process immediately precedes irreversible shock.

Clinical signs of shock depends on the critical reduction of capillary blood flow in various organs and tissues. Since the time of release of Pirogov and erectile torpidnu phase, the main criterion considered manifestations of the CNS functional activity. We agree that there is a general trend of shock marked correctly. It is used in modern classification stages of the clinical course of shock. But this division is not specific enough from the point of view of the pathophysiological changes occurring in the body in shock. Phase CNS disorders is more

integral indicator of the depth of shock and not enough to justify specific tactics intensive care of the victims.

- I. Compensated reversible shock. BCC deficit does not exceed 25% (700-1300 ml of the adult). Mild tachycardia, blood pressure unchanged or slightly decreased. Pustshayut subcutaneous veins, decreases in CVP. There are signs of peripheral vasoconstriction - cold extremities, a symptom of "pale spot" urine output (at a rate of 1-1.2 ml / min) is reduced by half.

II. Decompensated reversible shock. The deficit bcc 25 45% (1300-1800 ml). Tachycardia reaches 120/140 beats / min, ATS <100 mm Hg. Art., reduced the value of pulse pressure. Severe shortness of breath, partly offset by metabolic acidosis respiratory alkalosis, but it can also be a sign of "shock lung." There is a growing cold extremities, akrotsianoz. It appears cold sweat. Diuresis 0.3 ml / min.

III. Irreversible shock. The deficit BCC <50% (2000-2500 ml), the duration of circulatory decompensation over 12 hours. Pulse more than 140 beats / min, blood pressure below 60 mm Hg. Art. or not is determined. Consciousness is absent. Develops oliguria. Shock Treatment of dehydration is the consistent implementation of the following procedures:

- • 1. vein catheterization, if there are conditions - the backbone (usually the subclavian or jugular for Seldingerom).

- 2. Restoration of the BCC by inkjet or by intravenous drip of blood products and blood components. Recently offer at the scene and in the early treatment of shock of hospital treatment spend extra small volume infusion intravenously 4 ml / kg of 7.5% strength NaCl solution (50 ml dose). At the same time quickly formed a transmembrane osmotic gradient. Immediately there is a redistribution of water from the interstitial and cellular space in the vessels, which increases the BCC and reduces the threat of swelling of cells and development of MODS. The effect occurs within 1 minute and lasts about 30 minutes (D Kraymeyer, 1997). Even more effective combination of 7.5% solution of NaCl with colloidal plasma substitutes (e.g. to 6% or poliglyukinom gidroksimetilkrohmalem to 6%), contributing to retention of water in the vessels prepared for this lately-giperonkotichni gipertonichno special solutions administration 4-6 ml / kg which is 2-5 minutes in a peripheral vein quickly raises blood pressure and cardiac output while reducing peripheral resistance by reducing the pressure on the blood vessels from the outside through the endothelial swelling. It reduces the risk of the OPA, in particular increased diuresis. Also decreases bacterial translocation from the gut and the frequency of anaphylactic reactions of colloidal plasma substitutes.

- 3. The fight against metabolic acidosis. We introduce a 4% solution of soda at a dose of 150-300 ml.

- 4. The introduction of glucocorticoids (0.7-1.5 g of hydrocortisone or other adequate doses of corticosteroids). Not only do they improve the contractile function of the myocardium, but also to relieve peripheral vasospasm and increase the density of the membrane, protecting the cell structure and function of keeping them in shock. Contraindications to their introduction - a suspicion of acute bleeding from a stomach ulcer.

- 5. Removing peripheral vasospasm. Antipsychotics (eg, droperidol 2-4 ml by slow intravenous injection), or even ganglioplegic provided steady establishment of infusion therapy under the control of blood pressure to prevent collapse.

- 6. The use of inhibitors of pancreatic enzymes - 30 000-60 000 IU Trasylol or kontrikalu on saline to reduce the intensity of the pathological formation of kinins.

7. Ingalyatsiya humidified oxygen.

- 8. Application of hyperthermia as a physical cooling (ice bubbles of taxation) and medical (50% analgin solution of 2 ml or 5 ml reopirin deep intramuscular injection).

- 9. The introduction of broad-spectrum antibiotics as a shock leads to inhibition of the immune system.
- 10. Maintaining adequate diuresis (50-60 ml / h). Against the background of adequate fluid resuscitation, when CVP reaches 120-150 mm of water. . Article if urine output remains low, administered osmotic diuretics (mannitol in the form of a 10-15% solution of 5% glucose - 300 ml intravenously). If there is no effect from mannitol administered Lasix (furosemide) at 40-160 mg intramuscularly or intravenously to a total dose of 2000 mg the first day of treatment in the initial period of the arrester.
- 11. Maintaining cardiac kardiotonikami. Contraindications to the cardiac glycosides is a complete or partial atrioventricular block and the occurrence of ectopic foci of excitation. If bradycardia develops, prescribe stimulants P-adrenoceptor (izadrin tablets for sublingual use of 0,005 g). When ventricular arrhythmias administered Cordarone 150-300 mg or 0.1-0.2 g lidocaine intravenously.

The following table provides an indicative plan of infusion therapy on the replenishment of the BCC based on the amount of blood loss of data after its evaluation of shock Algovera index (the ratio of the pulse rate to the systolic blood pressure).

- Traumatic shock. The peculiarity of its pathogenesis - multi-faktornist pain, toxemia, bleeding and consistent cooling. The syndrome of prolonged compression (crush syndrome), and extensive soft tissue damage to the main pathogenic factor is the early toxicosis. As a result, toxic lesions of the renal epithelium and blockage convoluted tubules mioglobinovimi cylinder there arrester. In some cases, oliguria and anuria even if satisfactory BP provides insights into the degree of severity of the shock. When a burn injury, in addition to the pain and toxemia important pathogenetic factor is plazmovtrata with the affected surface, which is accompanied by the development of protein deficiency.

The clinical picture is clearly seen the phase course of shock seen in the days of Pirogov, who gave a classical description of erectile (excitation normotenziya or hypertension, pallor without cyanosis) and torpid phase (lethargy, lack of exercise, oliguria, shortness of breath, pale with an earthy tint and cyanosis cold clammy sweat). Modern systematization of traumatic shock phase involves the same three stages as in hemorrhagic shock.

For the treatment of traumatic shock applied the same methods as for the treatment of hemorrhagic shock. At the scene, and ambulance transport are important as soon as possible measures taken by ambulance: providing airway and adequate ventilation, hemodynamic restoration and its support for the introduction of the jet of blood substitutes (eg, 7.5% strength NaCl solution and polyglucin, stabizolu or refortanu), aseptic bandage on the wounds, immobilization of fractures and correct laying on a stretcher (half-sitting - position in wounds chest position Fowler - with TBI, horizontal - with abdominal wounds, "the position of the frog" - in case of damage of the pelvis), pain relief (analgesic with antihistamines), cardiac glycosides.

Septic shock (toxic-infective, endotoxin, bakteriemichny) developed peritonitis, infections of the urinary and biliary tract, pneumonia, pankreonekrozi, septic abortion and childbirth, and the like. In most cases it is caused by the Gram-negative bacteria, but may also occur under the influence of other agents (gram-positive bacteria, anaerobic bacteria, viruses, fungi, protozoa).

Features of pathogenesis is largely dependent on the sepsis pathogens. For Gram-negative flora in the decay it releases an endotoxin, which stimulates the adrenal medulla releases catecholamines, which are under the influence of vessels constrict. We distinguish gram-positive exotoxins which cause proteolysis with consequent formation plazmokininiv, which under the action of vascular paralysis occurs. Violated the contractile function of the myocardium under the influence of toxins and hypoxia pogirshannyu hemodynamics

promotes blood clots. Develops ODN for "shock lung", a violation of the respiratory muscle function, tachypnea (as compensation for metabolic acidosis), the effect of hyperthermia and direct action of bacterial toxins on the respiratory center. Through tissue ischemia and toksemiya often liver failure, which causes a disorder of blood clotting by type of DIC, acute renal failure based on nekronefrozu, damage to the pancreas, followed by hyperglycemia, excessive activation of kinins which cause an increased permeability of the vascular walls and reduces blood pressure, hemorrhagic gastroenterocolitis. There is a secondary immunodeficiency.

The clinical picture combines symptoms of infection (pyrexia, chills, bacteremia, change the white and red blood cells), neuropsychic, hemodynamic and respiratory disorders, lesions of parenchymal organs (kidney, liver, pancreas), homeostasis (coagulation TOC type of disorder CBS, VEO, dysproteinemia). septic shock treatment includes elimination, mainly surgical, focus of infection, correction of hemodynamic disorders (glycosides, such as digoxin 0.025% solution at a dilution of 1 ml to 10 ml of 5% glucose intravenously slowly dopamine, which is the dose of 0.5-2.0 mg / (kg • min) causes expansion of the renal vessels and improves renal function, a dose of 2.0-10.0 mg / (kg • min) improves heart function and reduces vascular resistance, and a dose of 10.0 mg / (kg • min) and causes vasoconstriction, tachycardia and arrhythmia; koronarolitiki and antiplatelet agents (0.5% chimes st and 2 ml izotopin 0.25% to 2 ml of a dilution with 150 ml of 5% glucose intravenous infusion); rheological agents (reopoliglujukin to 1500 ml to 500 ml neogemodez intravenously), heparin (5000 U every 4 hours or continuously drop to 30 000 units a day) - only in the absence of the threat of bleeding, or a fraction of heparin (0.3 ml fraxiparine, Clexane 40 fragmin mg or 5000 IU per day), which are safe, even with the threat of bleeding.

Provide adequate ventilation, replenishment of fluid loss, high-energy (not less than 4000 kcal) parenteral nutrition due to amino acids and carbohydrates (fat emulsions are not used because of the threat of blockade RES).

Carefully adjust to glucose metabolic changes in rate of 1 g / (h • kg) supplemented with insulin at 1 U 2.5-3 g glucose anabolic hormone (testosterone propionate 2 ml a day, 1 ml retabolil every 7-10 days), glucose-alcohol mixture to inhibit ADH, the release of which is increased in septic shock; protease inhibitors (contrical to 60 000 IU per day), large doses of vitamins C, B1, B6, B12, cocarboxylase (0.05-1.00 g per day intramuscularly or intravenously), fosfadenu 2 mL of 2% solution of 3- 4 times a day.

Were treated AKI intestinal paresis (PEO normalization, stimulation of motility hypertonic enemas, diodinamikom introduction of neostigmine 0.05% solution of 1 ml twice daily subcutaneously).

Correct immunity (immunoglobulin antistaphylococcal to 4 doses every other day for 5 days intramuscularly, intravenously antistaphylococcal plasma, biologics - pentaglobin, sandoglobin, monoclonal antibodies).

Inject antibiotics mainly bactericidal activity (penicillins, in t. Ch. Semisynthetic, aminoglycosides, cephalosporins), derivatives fluorinolonu, high doses of carbapenems (gentamicin-up to 240-400 mg / day), cephalosporins (up to 12 g / day).

Bacteriostatic agents (tetracyclines, levomitsetini, macrolides) inappropriate. For the prevention of superinfection - antifungal drugs (oral nystatin 500,000 IU 3-4 times a day, 500,000 units levorin 2-4 times a day), intestopan 1-3 tablets 4-6 times daily, amphotericin B, diflukan. To prevent dysbacteriosis prescribe biologics.

- Anaphylactic shock occurring as the pronounced manifestation of anaphylaxis (allergic reaction negative type on parenteral administration of the allergen) and atopy (allergic diseases with hereditary predisposition to sensitization).

Pathogenesis is the presence in the body reagenovih antibodies that promote the release of histamine from mast cells, which develop under the influence of severe respiratory disorders, and vascular tone.

A true anaphylactic shock is preceded by sensitization - an immune response as a result of which there reagenovi antibodies, ie the patient to shock occurrence should at least once in contact with the allergen. But sometimes shock develops, and at the first contact, because certain substances can cause the release of histamine without the participation of antibodies (eg, X-ray contrast agents containing iodine), although such a shock treatments did not differ from the present anaphylactic.

The clinic anaphylactic shock are the following forms: fulminant form is characterized by sudden development of pallor or cyanosis, dilated pupils, agonal breathing, and clinical death occurs in the next 10 minutes;

- severe with harbingers of impending disaster (in the form of complaints of shortness of breath and blood circulation), after which developed the same symptoms as in the form of lightning;
- moderately severe shock occurs in several different ways:
 - a) cardiac (the most common) - spasms or peripheral vascular expansion violates the peripheral and then central hemodynamics with a fall in blood pressure, respiration is not suffering;
 - b) asphyxial suffocation due to edema of the larynx, trachea or bronchospasm;
 - c) with cerebral symptoms, reminiscent of status epilepticus and acute cerebrovascular accident;
 - d) with abdominal symptoms perforation cavity organ or bowel obstruction.

Recognition of anaphylactic shock medium gravity promotes a skin rash that appears in this form of shock.

Treatment of anaphylaxis depends on its shape. With lightning and severe forms of anaphylactic shock immediately begin resuscitation - ventilation and chest compressions with the introduction of appropriate medicinal preparations. Diluted in saline after cardiac resuscitation with lightning and heavy forms, as well as other embodiments of anaphylactic shock offer moderate fraction (bolus) administration at 0.1-0.2 ml of 0.1% epinephrine, every 5-10 min to hemodynamic stabilization, as the adrenaline acts as an antagonist of humoral factors contributing to the development of anaphylactic shock. A mixture of adrenaline can be administered as a drip infusion.

When adrenaline is administered antihistamines (diphenhydramine, suprastin, Promethazine, tavegil dose of 0.5-1.0 mg / kg intravenously). Then injected corticosteroids (hydrocortisone 125-500 mg, or other drugs in adequate doses), bearing in mind that they do not exert their effects immediately.

When kardialnomu embodiment except the above, supplement bcc infusion of crystalloid. Avoid colloidal solutions due to their potential allergic effects.

When asphyxial embodiment through the trachea and larynx edema particularly recommended administration of antihistamines, epinephrine, and corticosteroids that are anti-edema and bronchospasm when administered as 2.4% aminophylline th dose of 5 mg / (kg • h) (20 ml for adults) for 15 min, and then 0.5 mg / (kg • h) (grown at 15 ml / h). In the absence of effect of this medication is performed endotracheal intubation or tracheostomy.

When cerebral embodiment except epinephrine, antihistamines and corticosteroids, diazepam administered 3.2 mL 0.5%, after 8 hours, - re Lasix (furosemide) with 2% - 3.2 ml intravenously.

When abdominal variant conduct a thorough differential diagnosis in order to avoid unjustified surgical intervention.

After the elimination of the symptoms of shock, antihistamines and corticosteroids continue to be administered within 2-3 days, and finally reveal the allergen to prevent further contact with the victim him.

Recommended literature:

A. Main:

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2. CURRENT Medical Diagnosis and Treatment 2012, Fifty-First Edition (LANGE CURRENT Series) by Stephen McPhee, Maxine Papadakis and Michael W. Rabow (Paperback - Sep 12, 2011)
3. Davidson's Principles and Practice of Medicine: With STUDENT CONSULT Online Access, 21e (Principles & Practice of Medicine (Davidson's)) by Nicki R. Colledge BSc FRCP(Ed), Brian R. Walker BSc MD FRCP(Ed) and Stuart H. Ralston MB ChB MD FRCP FMedSci FRSE (Paperback - Mar 11, 2010)Kumar and Clark's Clinical Medicine, 7e (Kumar, Kumar and Clark's Clinical Medicine) by Parveen J. Kumar (Paperback - Jul 2, 2009)
4. 1000 Questions and Answers from Kumar & Clark's Clinical Medicine, 2e [Paperback] Parveen Kumar CBE BSc MD FRCP FRCP(Edin) (Editor), Michael L Clark MD FRCP (Editor)
5. Differential Diagnosis in Internal Medicine: From Symptom to Diagnosis by Walter Siegenthaler (Mar 21, 2007)
6. Symptom to Diagnosis: An Evidence Based Guide, Second Edition (LANGE Clinical Medicine) by Scott D. C. Stern (Sep 16, 2009)
7. CURRENT Diagnosis and Treatment Emergency Medicine, Seventh Edition (LANGE CURRENT Series) by C. Keith Stone (May 23, 2011)
8. Harrison's Gastroenterology and Hepatology by Dan Longo and Anthony Fauci (May 3, 2010)
9. Mayo Clinic Gastroenterology and Hepatology Board Review (Mayo Clinic Scientific Press) by Stephen Hauser (Jun 23, 2011)
10. Clinical Nephrology 2012 (The Clinical Medicine Series) by M.D., C. G. Weber (Sep 19, 2011) - Kindle eBook
11. Goldman's Cecil medicine / [edited by] Lee Goldman, Andrew I. Schafer.—24th ed. Elsevier Sanders. Rev. ed. of: Cecil medicine. 23rd ed. – 2012. p.
12. Sonographer's Handbook of Diagnostic Ultrasound by Jason R. Young M.D. (Feb 23, 2011)

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