MINISTRY OF HEATH OF UKRAINE
POLTAVA STATE MEDICAL UNIVERSITY

Department of general surgery

METHODICAL INSTRUCTIONS
FOR STUDENT SELF-DIRECTED WORK WHEN PREPARING FOR AND DURING PRACTICAL CLASS

Study discipline	**General surgery**
Module №1	**INTRODUCTION TO SURGERY. SURGICAL EMERGENCY CONDITIONS. FUNDAMENTALS OF ANESTHESIOLOGY AND INTENSIVE CARE**
Content module 4.	**Injury and damage.**
Lesson theme №15	Polytrauma. Multiple injuries, combined and combined injuries. Traumatic shock. Syndrome of prolonged compression: pathogenesis, clinic, diagnosis, first aid, principles of treatment.
Years of study	***III***
Faculty	**International**

Poltava

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Lesson theme №15	Polytrauma. Multiple injuries, combined and combined injuries. Traumatic shock. Syndrome of prolonged compression: pathogenesis, clinic, diagnosis, first aid, principles of treatment.

1. Relevance of the topic :

In recent years, the death rate from injuries in Ukraine has increased by 38.7%. According to the Ministry of Health of Ukraine, 31-44 thousand people die from injuries every year. 20-25% of them from a combined injury. The incidence of polytrauma in recent years is 5.5-35% of all traumatized patients. The fatality for polytrauma is 12.2-63.4%, of which 65.1- 70.0% of the victims die in the first 24-48 hours (35.0% in the first 15 minutes since the injury). In economically developed countries, injuries are the third leading causes of death after cancer and cardiovascular disease. International experience shows that 15-20% of injuries are prevented each year if the emergency medical service is improved. The level of fatality and disability in polytrauma is inversely proportional to the speed and quality of care provided.

Polytrauma - a severe pathological process caused by damage to several anatomical areas or segments of the limbs with a pronounced manifestation of mutual deterioration syndrome, which includes the simultaneous onset and development of several pathological conditions, vascular diseases, is always accompanied by shock.

Traumatic shock (TS) is a frequent and formidable complication of gunshot wounds and peacetime injuries. During the fighting, natural disasters, large-scale terrorist acts, the importance of this problem increases significantly. While during the Second World War traumatic shock reached from 2.6% to 11.6% with various injuries, in the Afghan war 1979-1989. its share increased to 25-30%. This is due to changes in weapons, their improvement, the increase in their destructive power and the increase in the number of heavy and multiple injuries.

In the use of nuclear missile and missile weapons, shock was found in 25% of all victims. After the atomic bomb explosion in the cities of Hiroshima and Nagasaki, one in five victims (20%), in Korea, with napalm - 21.4% shock.

The fatality in shock remains high and reaches 30-40%. Therefore, the organization of care, diagnosis and treatment of victims of polytrauma with shock and long-term compression syndrome is an important and complex problem.

So, mastering the skills and skills to provide medical care, care for patients who have undergone surgery, is important for all employees of the surgical service.

2.Specific goals:

- Definition of polytrauma, classification.
- Definition of traumatic shock (TSh), long-term compression syndrome (LTCS).
- Assimilate the classification of TSh and LTCS.
- Assimilate the etiology of the occurrence of TSh and LTCS.
- Know the pathogenesis of TSh and LTCS.

- Know the methods of diagnosing the severity of TSh and LTCS.
- Assimilate complex therapy of TSh and LTCS depending on the severity of the condition of the victim and the severity of the injury.
- Assimilate first aid in emergencies.
- Assimilate the prevention of LTCS.
- Know the complications of TSh and LTCS.
- To be able to provide first aid for TSh and LTCS.
- To master the equipment of transport immobilization.
- To master the technique of overlaying the harness.
- To be able to conduct a clinical examination of the victim with TS and LTCS.
- Be able to interpret data from hemodynamic indicators in TSh and LTCS
- Be able to interpret data from laboratory blood tests (general analysis, biochemical indicators, electrolyte indicators).
- The algorithm of first aid for long-term compression syndrome and traumatic shock.
- Principles of treatment in hospital conditions.

3. Basic knowledge, skills, skills necessary to study the topic (interdisciplinary integration).

Discipline	Skills learned
Normal anatomy	Osteology, myology, syndesmology, joint structure. anatomy of the circulatory organs and nervous system.
Histology	Cytology, morphology and blood function, cardiovascular system, endocrine system, respiratory system, digestive system, liver, pancreas, urinary organs.
Physiology	The physiology of the circulatory organs. The structure and function of the microcirculator vascular bed. General perceptions of central and peripheral blood flow. The physical and chemical mechanisms of fluid exchange between blood and tissues. Neuromoral regulation of filtration, reabsorption and kidney secretion.
Surgery and topographical anatomy	Topography of vascular-nervous formations of limbs. Topography of internal organs.
Propedevtics	Care for lying patients.
Biophysics	Energy transfer laws, kinetic energy. The laws of hydrodynamics.
Pathologycal Physiology	Disruption of microcirculation, ischemia, stasis, thrombosis, water-electrolyte metabolism, hypoxia, heart failure, respiratory pathology, impaired kidney function, impaired function of the hypothalamus, pituitary gland, adrenal glands, thyroid glands.
Pathological anatomy	Shock organs.

4. Self-employed tasks in preparation for class and class.

4.1. List of the main terms, parameters, characteristics that a student should learn in preparation for the class:

Term	*Definition*

Traumatic shock (TS)	Response of an organism to a severe injury. it is shown in disorders of the vital functions of an organism (blood circulations, gas exchange, a metabolism, neurohumoral regulation).
Primary shock	Arises directly after influence causal a factor (a consequence of a trauma).
Secondary shock	Arises under the influence of auxiliary aggressive factors (to an insufficient (wrong) transport immobilization, heavy and long transportation, it is prematurely executed surgery).
Recurring shock	Arises after a conclusion from shock with unstable compensation of the vital functions of an organism.
Syndrome of long compression (SLC)	Symptoms complex of local changes, owing to long compression of a segment of a body of the person with disorder of blood supply with hypostasis, a necrosis of tissues and the general symptoms of traumatic shock, intoxication of an organism and a acute renal failure.
Haemo dynamics	It is the movement of blood in cardio vascular system
First aid	A set of simple, appropriate actions aimed at maintaining the health and life of the victim
Hypoxia	Reduced oxygen content in tissues.
Hypoxemia	Lack of oxygen in the blood.
Anoxemia	Lack of oxygen in the blood
Microcirculation-qi i	Blood circulation in the system of small vessels (with a diameter of less than 100 microns) of an organ or tissue ensures the supply of oxygen and nutrients to them.
Necrosis	The death of individual cells, tissue, parts or an entire organ in a living organism.
Acidosis	Violation of the acid-core equilibrium in the body, characterized by the appearance of an absolute or relative excess of acids in the blood and an increase in the concentration of hydrogen ions.
Spasm	Involuntary contraction of cross-striped or smooth muscles.
Dilatation	Sustained diffuse expansion of any hollow organ (vessels).
Oliguria	Reduced discharge of urine.
Hemodialysis	The method of correction of water-electrolyte, acid-core condition and removal of various harmful substances from the body.
Hemosorbtion	The method of removing toxins from the body by extracorpolyse perfusion of blood through sorbents.

4.2. Theoretical questions to occupation.

- Definition of polytrauma, classification;
- definition of TSh;
- definition of the concept of LTCS ;

- classification of TSh;
- classification of LTCS ;
- etiology of TSh;
- etiology of the occurrence of LTCS ;
- pathogenesis of TSh; pathogenesis of LTCS ;
- phase and severity of TSh;
- periods of LTCS;
- methods for diagnosing the severity of TSh;
- methods for diagnosing the severity of LTCS;
- complex therapy of TSh depending on the severity of the condition of the victim and the severity of the wound;
- complex therapy of LTCS, depending on the severity of the condition of the victim and the severity of the wound;
- first aid on the battlefield, Normal display Maintenance and emergency care;
- indications and contraindications for surgical intervention in case of TSh at the stage of qualified surgical care;
- prevention of LTCS ;
- complications of TSh;
- complications of LTCS ;
- principles of transport immobilization;
- rules for applying a tourniquet, bandaging with an elastic bandage;
- clinical examination of a victim with TSh;
- clinical examination of the victim with LTCS ;
- tactics of treatment of TSh at various stages of medical evacuation;
- Tactics of treatment of LTCS at various stages of medical evacuation;
- means of transport immobilization;
- The tire structure of Diterichs and Cramer.

4.3. Practical work (tasks) that are performed in the lesson:

- technique for carrying out a complex of emergency measures for shock and prolonged compression syndrome (mouth-to-mouth breathing, air ducts administration, oxygen supply by devices, administration of drugs;
- technique of local anesthesia and novocaine blockade (vagosympathetic, perinephral, paravertebral, intercostal, intrathoracic according to L.G. Shkolnikov and V.P. Selivanov, case, conductive);

- The technique of venosection and venipuncture of the central (subclavian, large saphenous) and peripheral veins.
- compliance with the rule of three catheters (catheterization of the nasal passages, veins, urinary bladder);
- the appointment of comprehensive anti-shock therapy ;
- diagnosted of shock and syndrome and crush, acute renal disease based on clinical symptoms: consciousness, sedation, eye and tendon reflexes, blood etc. The pressure, pulse rate, depth and breathing rate, temperature, color, sweating, diuresis, urine color ;
- temporary stop of bleeding ;
- superimposing aseptic dressings;
- the imposition of transport tires from improvised materials, splint of Cramer, Diterichs;
- selection of medicines for the treatment of traumatic shock and prolonged compression syndrome.

5. The content of the topic:

Injury classification:

An isolated injury is each individual damage in any anatomical - functional area of the body or organ.

Multiple trauma is two or more of the same type of damage within the same anatomical region.

In traumatology, 6 anatomical sites are distinguished: 1) head; 2) neck, chest; 3) the spine; 4) the stomach; 5) retroperitoneal space with a basin and perineum; 6) limbs (for example, fractures of the hip and leg bones).

A well- documented injury - several injuries within different anatomical - functional areas.

Combined injury - damage resulting from the simultaneous or sequential exposure of several traumatic agents to the body.

The short word " **polytrauma** " is an alarm, it determines the complexity of the injury, the severity of the patient's condition, the presence of blood loss, shock, etc. At the same time, it is a call to readiness to provide assistance to the victim at all stages: ambulance, emergency room, resuscitation service, operating room.

Polytrauma is characterized by:

- syndrome of mutual burden;
- atypical symptoms of damage;
- the complexity of the diagnosis;
- the need for a continuous assessment of the severity of the victim;
- urgent need for adequate medical measures;
- the development of a traumatic disease;
- a large number of complications;
- high mortality.

Damage to the abdominal organs especially aggravates the condition of patients with polytrauma. In case of a traumatic disease, events develop according to the following scheme: severe trauma (mechanical damage) - shock (shockogenic injuries) - general

inflammatory response syndrome (damage resulting from the systemic action of inflammatory mediators) - multiple organ dysfunction - infectious complications (infectious damage).

Polytrauma - heavy multiple and associated injuries, in which there is a traumatic illness and requiring e care for health reasons. A prerequisite for the use of the term "polytrauma" is the presence of traumatic shock, and one of the injuries or a combination of them pose a direct threat to life and health.

With polytrauma, a **traumatic disease** always develops. The basis of the pathogenesis of traumatic disease is a combination of damage reactions and defense reactions. The first includes traumatic shock, blood loss, impaired function of damaged organs, traumatic toxicosis, catabolism, tissue necrosis, decreased immunity; to the second (adaptive reactions) - redistribution of blood flow, increased erythropoiesis, extravasal fluid entering the vascular bed, anabolism, tissue regeneration. In general, an acute onset is always characteristic of a traumatic disease; lack of a latent period; hypoxia of the circular anemic type; systemic reaction is aggressive (fever, psychasthenia, aseptic and septic inflammatory foci).

In the course of a traumatic disease, 3 periods are distinguished:

I - shock;

II - detailed clinical picture;

III - the period of rehabilitation.

Traumatic shock is the body's response to severe trauma and is manifested in disorders of the vital functions of the body (blood circulation, gas exchange, metabolism, neurohumoral regulation).

The duration of the acute period of traumatic disease (shock) is about 2 days.

The occurrence of traumatic shock among the civilian population with multiple and combined injuries ranges from 13.3 to 86%.

The definition of traumatic shock.

In case of injury of varying severity and intensity, a reaction manifests itself - the response of the body in the form of an acceleration of the pulse, respiration, pallor of the skin and mucous membranes due to spasm of peripheral vessels, and a decrease in blood pressure. These manifestations may be different and depend on the severity of the injury.

The reaction-response is based on mobilizing defense reactions to maintain homeostasis, first in the form of compensation. If the traumatic factor, by the strength of intensity, severity and localization, continues to affect the body, the compensation stage can go into the decompensation stage. Depending on the severity of homeostasis disorders and related circumstances, the body can cope on its own or as a result of depletion of protective mechanisms, lack of therapeutic measures, a gradual transition to a terminal state occurs.

Tramatic shock is the response of the body to severe trauma, which manifests itself in disorders of the vital functions of the body (blood circulation, gas exchange, metabolism, neurohumoral regulation).

The occurrence of traumatic shock among the civilian population with multiple and combined trauma ranges from 13.3 to 86% .

Etiology and pathogenetic factors of shock.

etiology : closed and open traumatic injuries of any localization.

Contributing factors of shock:

- hypothermia, overheating;
- fatigue, malnutrition;
- additional trauma in the provision of medical care, removal from the battlefield, evacuation in case of poor transport immobilization or in its absence (secondary shock); \
- repeated bleeding
- poor quality late medical care.

The pathogenesis of shock.

Shock - a pathophysiological symptom, which is based on a decrease in blood flow to the tissues below the level needed to ensure the metabolism , which leads to intracellular disorders am and massive cell death.

Pathogenetic factors of shock

1. Massive blood loss (38%).
2. Damage to important organs: chest, abdomen, brain (30%).
3. Acute respiratory distress (21%;).
4. Pain and excessive afferent impulse (6.2%).
5. Intoxication: infections, prolonged with tissue pressure , intoxication with damage to the gastrointestinal tract, fat embolism (4.8%).

There are always 2-3 factors in the occurrence of traumatic shock (45%).

The pathogenesis of hemodynamic disorders.

BCC is characterized by relative constancy. 70-80% of blood circulates in the veins, 15% in the arteries and 5-7.5% in the capillaries. In general, 80% of all blood circulates in the cardiovascular system, and 20% of blood is in the parenchymatosus organs. The stability of the BCC is supported by the self-regulation mechanism. Hemodynamic changes cause disturbances in the stability of the internal environment, as a result of which the protective mechanisms are triggered.

With shock in hemodynamic disturbances, two phases are distinguished: the compensation phase and decompensation.

Compensativ phase. The formation of the main reactions to trauma is carried out in the subcortical centers of the brain, caused by a reaction to pain, which causes dilatation of blood vessels, a decrease in cardiac output, hypotension, and decentralization of blood circulation. At the same time , a decrease in bcc as a result of blood loss, hypoxia leads to a violation of blood chemistry and the development of postganglionic sympathetic endings, which causes the release of catecholamines into the bloodstream. Simultaneously occurs excitation of the pituitary-adrenal-renin system, which causes additional release into the blood pre quarrel in vessels. In aggregate, a vasospasm occurs. Excitation of β-receptors of the heart leads to an increase in cardiac contractions.

Hemodynamics are compensated in three directions: an increase in resistance to blood flow, an increase in bcc, an increase in the volume of shock and minute ejection of blood.

Vascular spasm contributes to:

- a decrease in the capacity of the vascular bed and a relative increase in bcc ;
- spasm suck rows α-receptor zone and ejection part deposited blood ;

- increase in blood pressure ;

- a spasm of pre-postcapillary sphincters lowers the pressure in the capillaries and, according to the Starling hypothesis, promotes the transition of intercellular fluid into the vascular bed;

- activation of renin-angiotensin-aldesterone, an increase in blood pressure causes an increase in the reabsorption of water and sodium ions in the kidneys. That is, a decrease in urine output does not lead to a decrease in bcc. In addition, sodium ions increase the sensitivity of arteries to the action of spores and blood vessels. Angiotensin causes a vasoconstrictive effect 10 times more active than norepinephrine;

- spasm of the lymphatic vessels increases bcc and the level of blood proteins due to lymph.

Hemodynamics compensation ensures proper circulation of the heart and brain.

With the elimination of shockogenic factors, the restoration of BVC, there is a need to relieve vasospasm and restore blood circulation in the tissues, providing oxygenation and metabolism.

Hemodynamic decompensation phase.

Prolonged pre- and post-capillary Single vasoconstriction reduces tissue perfusion: blood flow slows down, comes aggregation of erythrocytes, thrombocytes, increases viscosity of blood. There is a "shock-specific e" microcirculation disturbance.

Enhanced hypoxia activates leukocytes with the treatment of arachidionic acid products .

Locally activated cytokines IL-1, IL-2, and L-6, tumor necrosis factors, coagulation factors, free radicals, proteolytic enzymes. The cellular response is manifested in the activation of lymphocytes, neutrophils, monocytes. Cellular and humoral factors interact with each other and with adhesive molecules that contribute to the adhesion of platelets and platelets with adhesion to the capillary endothelium, increasing the permeability of capillaries. The slowdown of blood flow in the capillaries is promoted by the activation of nitric oxide, which causes dilatation of the capillaries.

Increased hypoxia disrupts carbohydrate metabolism with the formation of lactic, pyruvic acid. Hemoconcentration, blood stasis, acidosis, aggregation of cellular components, increase the permeability of the capillaries contributes sequestration blood and fluid into the exit interstcial space.

Acidosis causes damage to the membranes of lysosomal inclusions with the release of serotinin , histamine, bradykinin, proteolytic enzymes, lipases, amylases, which cause autolysis of cells. Aude simultaneity disclose camping precapillary sphincters , arterio-venous shunts. As a result, the output of blood vessels and blood vessels increases , which causes thickening of the blood, a decrease in bcc, as well as expansion of veins, venostasis and decentralization of blood circulation, a drop in blood pressure, a decrease in blood flow to the heart and blood flow. Transport ischemia enhances metabolic acidosis, irritating the chemoboreceptors of the aortic arch and carotid sinuses, increasing tachycardia to 140-150 beats per minute. This is negatively reflected in the coronary circulation , causing acute coronary insufficiency, myocardial depletion and cessation of cardiac activity. So the vicious circle closes:

At the same time, hypocalcemia and hyperkalemia negatively affect the myocardium, increasing myocardial dystrophic phenomena. At this time, on the ECG, there is high tachycardia, an increase in the R and T wave, and a decrease in the ST line.

This "hemodynamically bewitched circle" is closely intertwined with other "circles", in particular with respiratory disorders.

Pathogenesis of respiratory distress.

Injury and acute hemorrhage reflexively cause hyperventilation (1.5-2 times higher than normal), providing minute ventilation of the lungs, compensating for oxygen saturation in the period of compensation of hemodynamics. The brain, the heart receives tons of oxygen-rich blood.

As a result of reduced organ blood flow, low perfusion in the capillaries, oxygen delivery to tissues decreases, metabolism changes from aerobic to anaerobic, lactic, pyruvic acid is formed instead of CO_2 and water. The amount of buffer bases is rapidly decreasing.

Under the influence of acidosis, breathing becomes more frequent, but superficial, causing ventilation disorders and, accordingly, blood oxygenation. Hemodynamic decompensation, the opening of arteriovenous shunts, pulmonary edema, blood hemostasis in the lungs causes a critical deterioration in blood oxygenation and blood flow to the heart. The decompensation of hemodynamics is combined with decompensation of respiration and, as a consequence, a critical deterioration in hypoxemia and an increase in acidosis. This leads to the depression of the respiratory center and the autostopping.Another one , the “bewitched circle” closed, a second component of clinical death appeared: respiratory arrest joined the cardiac arrest.

The pathogenesis of metabolic disorders.

Under the influence of activation of the adrenal system, the liver glycogen goes into glucose to repay energy costs, causing hyperglycemia. In the phase of decompensation of hemodynamics, hypoxia, metabolic acidosis occurs. Simultaneously, the phase compensation of hemodynamics and metabolism is increased depletion of vitamins, in particular vitamin C. Reduced aetsya calcium - potassium index in the direction of hyperkalemia. The metabolism of fats is disrupted with the formation of intermediate products of ketone bodies, β-hydroxybutyric acid. Also Hard Of aetsya protein metabolism due to impaired renal excretory function, accumulate urea, uric acid, creatinine. The content of proteolytic enzymes increases, negatively affecting the cells, causing their proteolysis. Thus, decompensation of metabolism occurs.

The pathogenesis of disorders of the endocrine system.

In order to enhance compensation and adaptation of vital functions of the body, the pituitary-adrenal system is primarily activated reflexively and humorally , taking part in the compensation of hemodynamics. In the phase of hemodynamic decompensation, adrenal exhaustion occurs, causing acute adrenal insufficiency. At the same time, the function of the secondary glands is depleted : thyroid, parathyroid, and gonads.

Pathogenesis of disorders of the nervous system.

Pain, excessive afferent impulse cause the wounded suffering, excessive psycho-emotional stress, and with prolonged exposure and impaired formation of adaptation reactions, the regulation of the cardiovascular system is disrupted. First comes the activation of the sympathetic part of the nervous system, and after its depletion - the advantage of the parasympathetic. There is a depletion of reflex activity in the form of hypoareflexia, decortication, decerebration of clinical and biological death.

Traumatic shock and shock of a different genesis are a pathophysiological symptom-complex in which the blood flow to tissues decreases below the level necessary to ensure normal metabolic processes, which leads to intracellular disorders and mass cell death. That is, the shock is based on microcirculation disorders, in which neurocirculatory, immune, metabolic and endocrine components take part.

Thus, shock is a polyetiological and monopathogenetic pathophysiological syndrome with a microcirculation crisis in the end.

Classification of shock.

Primary - occurs immediately after exposure to a causal factor (consequence of an injury).

Secondary - under the influence of auxiliary aggressive factors (insufficient (incorrect) transport immobilization, heavy and prolonged transportation, prematurely performed surgical intervention).

Recurrent - repeated - after removal from shock with unstable compensation of vital functions of the body.

"Superimposed" on the corresponding reactions of the injured organism, it is more difficult than the primary shock, and mortality from it is much higher.

The clinical picture of shock. Phases of shock.

In the clinical course of traumatic shock, three phases are distinguished: erectile, torpid, and terminal.

The ***erectile phase*** is not always (9-12%), expressed with damage to the head. It depends on the nature of the damage, the strength and duration of the pain irritation, the type of nervous system, the previous mental state, the state of the combat situation at the time of the wound.

Symptoms:

- short duration; excessive, more than 2-3 hours, the duration is an unfavorable prognostic sign with the transition to a severe degree of the torpid phase;
- psychomotor, linguistic arousal;
- the skin is pale, dry, sometimes can be replaced by hyperemia;
- blood pressure is elevated or normal;
- increase in blood sugar;
- increased hyperesthesia, hyperreflexia and muscle hypertension.

The torpid phase is characterized by a certain dynamics of state changes: relative compensation, decompensation, and the formation of irreversible changes.

In the torpid phase, disorders of all vital body functions are observed:

- retardation with preserved consciousness;
- pallor of the skin and mucous membranes;
- decrease in blood pressure;

- increase in heart rate;
- hypothermia;
- decrease in tendon reflexes;
- oliguria;
- rapid breathing, choking.

II period of a traumatic disease is a period of a developed clinical picture. It is divided into two phases - catabolic and anabolic. The catabolic phase is characterized by a predominance of local manifestations over the general ones, and for the anabolic phase, on the contrary

Treatment of **polytrauma** is carried out in stages. At the prehospital stage, they provide emergency care: they stop bleeding, provide respiratory tract obstruction and artificial ventilation of the lungs when breathing is disturbed, they give an indirect massage and use medications when the heart stops carry out transport immobilization and anesthesia. At the hospital stage, measures are taken to combat shock, including the stabilization of hemodynamics, complete pain relief; reliable immobilization, oxygen therapy, correction of all impaired functions. Surgical interventions for polytrauma are divided into urgent, urgent and delayed. Emergency interventions (for health reasons) are carried out together with antishock therapy for massive blood loss (damage to the spleen, liver), lung crushing, cardiac tamponade, intracranial compression. Urgent interventions are carried out after stabilization of the patient's condition and its removal from shock. This is surgical treatment of wounds, amputations, osteosynthesis (with combined trauma, external fixation is preferred). Delayed interventions (necrectomy, skin grafting, etc.) are performed during the catabolic and anabolic phases of traumatic disease.

During the rehabilitation period of a traumatic disease, treatment is aimed at the most complete restoration of functions..

Long-term compression syndrome (CTC) is a separate type of traumatic disease that develops as a result of prolonged compression of tissues with impaired blood flow and is a general reaction of the body to degenerative necrotic processes in ischemic tissues with subsequent reperfusion toxic damage to vital organs and systems.

The clinical manifestations of the crash syndrome are stereotyped and do not depend on the localization of the compression site and its causes, but are determined by the degree of tissue ischemia and endotoxemia. The severity of the condition of the victim is determined by the severity of the injury, the amount of plasma loss and the severity of endotoxemia after decompression.

In the clinical course of STS, three periods are distinguished: 1) early (1-3 days) - an increase in edema and hemodynamic disorders. It begins with the termination of the traumatic agent. The general condition of the patient at this time may be satisfactory. They mainly complain of general weakness, nausea, pain in a damaged organ, and loss of function. Pulse is rapid, blood pressure is reduced; 2) acute renal failure (from the 3rd to the 9th-12th day). During this period, the general condition of patients improves slightly. The pain and swelling of the affected areas are gradually reduced. Blood pressure is normal or slightly elevated, body temperature is from 37.5 to 38.5 ° C. The pulse corresponds to temperature. The signs of acute renal failure, which on the 5th - 7th day are already pronounced, are gradually increasing. Diuresis gradually decreases, the amount of urea in the urine increases. Death can occur on the 8th - 12th day. The development of

acute renal failure and the severity of its clinical manifestations are affected not so much by the length of time the victim remains under the rubble, but by the length of the interval from the moment of release from compression to surgery; 3) late (from the 9-12th day to the end of the second month). Local changes prevail over general ones. A gradual restoration of sensitivity is accompanied by increased pain. In the presence of a wound, necrotic muscles protrude from it. Gangrene of the distal extremities is sometimes observed.

According to the severity of clinical manifestations, four forms of STS are distinguished:

1) very severe - develops in case of compression of both lower limbs for 6 hours or more. Death occurs on the first or second day after injury; ¬

2) severe - one (or both) lower limb is compressed for 6-7 hours;

3) moderate - occurs in the case of compression of soft tissues for less than 6 hours. It proceeds without visible hemodynamic disorders, with moderate impaired renal function ¬

4) light - individual segments of soft tissues are compressed for a duration of less than 4 hours. Disorders of the functions of the cardiovascular system and kidneys are slightly expressed.

Locally in areas subjected to compression, they show deformations, abrasions,

Conflicts, or blisters filled with serous or hemorrhagic contents. After elimination of compression, soft tissue edema quickly develops.

With compensated ischemia (when there was no complete ischemia or ischemia was short for 2-4 hours), active movements, tactile and pain sensitivity remain in the limbs. With uncompensated ischemia, the limbs lose active movements, tactile and pain sensitivity. The listed local clinical signs are temporary. Compression time no more than 6-10 hours.

Irreversible limb ischemia is characterized by the loss of both active and passive movements. Muscles acquire a ligneous density: rigor mortis, lack of all types of sensitivity. Necrosis of the extremities is indicated by signs of dry or wet gangrene. From day 4-5, the severity of the condition of the victims is determined by acute renal failure, less commonly, respiratory distress syndrome.

Prehospital care for STS includes the following actions:

1) narcotic analgesics are administered to the victims at the scene of the incident before being released from the rubble;

2) before release from the rubble, a tourniquet is imposed on a squeezed limb;

3) immediately after the release, the viability of the limb is assessed: with compensated and uncompensated ischemia, the tourniquet is removed. With irreversible ischemia and crushing of the limb, the latter is subject to amputation, the tourniquet is not removed or re-laid closer to the compression zone.

After releasing and removing the tourniquet, an aseptic dressing is applied, the limb is bandaged with an elastic bandage, immobilized, covered with ice, snow (reduces the absorption of toxic substances), anti-shock measures begin.

Treatment of patients with STS should be aimed at restoring coordination of the processes of excitation and inhibition, correcting hemodynamic disorders, restoring

general homeostasis by conducting early intensive and adequate infusion therapy, reducing plasma loss and toxemia, and normalizing kidney function.

6. Materials for self-control:
Tests for self-control (initial level of knowledge) .

1. Algover's shock index is the ratio:
A) heart rate / blood pressure system;
B) BP syst / central VP;
C) BP syst. / Heart rate;
D) central VP / cardiac rate
E) BVC / centralVP.

2. A characteristic sign of concussion:
short-term loss of consciousness;
paraorbital hematomas,
mild nosebleeds;
prolonged loss of consciousness;

3. First aid for valvular pneumothorax:
translation of valve pneumothorax into the open;
thoracotomy;
thoracocentesis;
translation of valve pneumothorax into closed.

4. Place of drainage of the pleural cavity with pneumothorax:
2-3 intercostal space along the midclavicular line;
7-8 intercostal space in the front axillary line;
7-8 intercostal space along the scapular line;
doesn't really matter.

5. How quickly does the pattern of traumatic toxicosis develop?
after releasing the limb from compression;
immediately after compression of the limb;
4-8 hours after the liberation of the limb;
within 24-48 hours. after the liberation of the limb;
2-4 days after limb release.

6. What are the main pathogenetic factors in the development of traumatic toxicosis:
pain irritation, plasma and blood loss, traumatic toxemia.
fat embolism of internal organs; traumatic toxemia;
plasma and blood loss; traumatic toxemia;
coagulation disorders traumatic toxemia;
traumatic toxemia.

7. The main periods of the clinical course of traumatic toxicosis:
period of increase in edema and vascular insufficiency, acute renal failure, recovery;
period of increase in edema and vascular insufficiency;
toxic period;
period of acute renal failure:
recovery period, reactive period.

8. Express the symptoms of the torpid phase of traumatic shock of the II degree:
lethargy, adynamia, hypotension, tachycardia;
hypertension, tachycardia, adynamia;
hypotension, tachycardia;
tachycardia, patient agitation;

9. The main sign of the torpid phase of shock are:
coma, hypotension;
vomiting
increased white blood cell count;
pupil myosis;
tachycardia.

10. Name the phases of shock ::
erectile, torpid;
erectile, terminal;
initial, intermediate, terminal;
lightning, acute, subacute;
erectile, terminal.

11. Periods of prolonged compression syndrome:
early, intermediate, late;
initial, intermediate, terminal;
Acute, subacute;
erectile, terminal;
erectile, intermediate, late, terminal.

12. In the pathogenesis of crash syndrome, the main role is played by:
compression and decompression;
compression;
decompression;
acute blood loss;
pain.

13. The main measures in the treatment of traumatic shock at the prehospital stage:
stopping bleeding, analgesic therapy, restoration of BCC;
the introduction of antibiotics;
restoration of electrolyte balance;

recovery of consciousness;
the introduction of analgesics.

14. The main sign of the torpid phase of shock are:
coma, hypotension;
vomiting
increased white blood cell count;
pupil myosis;
tachycardia.

15. What is the period of the syndrome of prolonged compression of the tissues of the limb:
intermediate;
erectile;
torpid;
acute;
terminal.

16. Indicate which of the symptoms is characteristic of the erectile phase of traumatic shock?
excitation;
fainting state;
injured in consciousness;
retardation;

17. Indicate which of the symptoms is characteristic of the torpid phase of shock?
acceleration and weakening of the filling of the pulse;
increase in blood pressure;
polyuria;
increase in body temperature;
increased metabolism.

18. Traumatic toxicosis occurs due to:
prolonged squeezing of soft tissues;
severe abdominal injury;
spinal fracture;
closed chest damage;
transfusion of incompatible blood.

19. In the case of prolonged compression syndrome, it is necessary:
applying a tourniquet or twist, tight bandaging, immobilization of the limb;
limb immobilization;
imposing a tourniquet or twist;
grinding limbs;
rubbing the limb, immobilization of the limb.

20. How many stages during acute renal failure with prolonged compression syndrome?

4 stages;
2 stages;
3 stages;
Stage 1;
5 stages.

21. In which of the stages of acute renal failure with prolonged compression syndrome is polyuria observed?

3 stages;
2 stages;
1 stage;
4 stages;
5 stages.

B. Tasks for self-control:

1. Patient B, 45 years old, riding a bicycle, received a fracture of his right thigh. Is it possible to reposition fragments at the time of first aid?

not;
Yes;
yes, only after stopping the bleeding;
yes, only after administration of analgesics;
Yes, after stopping the bleeding and the introduction of analgesics.

2. Patient P, 30 years old, fell from a tree and received a fracture of his left thigh. An ambulance was called. Which tire should be used at the time of first aid?

Diterichs splint;
Cramer splint
Elansky splint;
Shantz splint;
gypsum bandage.

3. Patient Zh, 50 years old, after an accident, was taken to the emergency department of the emergency hospital. During the examination, the patient is inhibited, adynamic. Pulse of weak filling, tachycardia. What phase of the traumatic shock is this picture typical for?

torpid;
erectile;
agony;
clinical death;
biological death.

4. Patient Yu, 20 years old, after an accident, was delivered with the following clinical picture: inhibited. The skin is pale, cold, clammy sweat. Severe cyanosis of the nail bed, when pressed with a finger, blood flow is restored very slowly. Arterial systolic pressure is reduced to 90-70 mm. Pulse of weak filling, increased to 110-120 in 1 min. Central venous pressure is reduced. The breath is shallow. What degree of shock does the patient have?

the second;
the first;
the third;
the fourth;
terminal.

5. The patient was admitted to the hospital 4 hours after falling from a height on his left side. Complains of pain in the left hypochondrium, general weakness. Objectively: the patient is pale, AD-90/50 mm, pulse-110 beats per minute, weak filling. The abdomen takes part in the act of breathing; upon palpation, it is soft, moderately painful throughout. With percussion, blunting of percussion sound on the flanks. What damage can be assumed in the patient?

damage to the spleen with internal bleeding, traumatic shock;
damage to the hollow organ by internal peritoneal bleeding;
damage to the parenchymal organ with peritonitis;
peritonitis, traumatic shock.

6. The patient fell and received a chest injury. Upon admission, he complains of hemoptysis, shortness of breath, increased pain in the chest with deep breathing. Objectively: the skin is pale, respiratory rate - 30 / min., Pulse - 100 beats. / min., AD-110/60 mm. Hg On palpation, crepitus is determined along the posterior axillary line in the region of VI-VII ribs on the left. Percussion - tympanitis over the left half of the chest. Auscultatory - the absence of respiratory sounds over the left half of the chest. What is the complication of injury in this patient?

fracture of ribs on the left, closed pneumothorax on the left, traumatic shock of the I st .;
rib fracture on the left, left-side closed pneumothorax;
rib fracture on the left, left-side closed hemothorax;
left-side closed pneumothorax, traumatic shock of the II Art.

7. The patient was admitted with severe subcutaneous emphysema of the upper half of the body and shortness of breath up to 30 breaths per minute. Objectively: pulse up to 120 beats / min., Blood pressure 100/60 mm Hg, pale skin. Over the left half of the chest - tympanitis, breathing is not audible. Heart sounds are deaf, on a panoramic chest x-ray signs of pneumothorax, mediastinal displacement to the right. What emergency care should be given to the patient?

drain the pleural cavity;
administration of analgesics;
stop bleeding and the introduction of analgesics;

surgical intervention;
immobilization, the introduction of analgesics.

8. Patient K., 36 years old, in an accident, received a closed chest injury and a fracture of the left shoulder. The severity of the condition of the victim is due to traumatic shock of the first degree. Systolic blood pressure in a patient is:
90 mmHg;
80 mmHg;
120 mmHg;
100 mmHg;
70 mmHg

9. Patient L., 48 years old, was taken to the surgical department due to a combined injury: closed craniocerebral injury, closed chest injury, fracture of 3 ribs on the left. Traumatic shock of the II degree. Systolic blood pressure in a patient is:
80 mmHg;
90 mmHg;
70 mmHg;
100 mmHg;
60 mm Hg

10. Patient L., 21 years old, was taken to a surgical clinic after falling from the 5th floor with combined multiple trauma. The patient's condition is terminal. Blood pressure is:
not determined.;
40 mmHg;
70 mmHg;
100 mmHg;
60 mm Hg

11. Patient N., 47 years old, is in a specialized surgical clinic for a syndrome of prolonged compression. Indicate the main manifestations of the syndrome of prolonged compression:
acute renal failure, acute liver failure, anemia, pulmonary edema, hypovolemia;
acute liver failure, hypovolemia, anemia
acute renal failure, pulmonary edema, anemia
acute liver failure, hypovolemia, pulmonary edema.

12. A patient diagnosed with "Politrauma" stated clinical death. When conducting a closed heart massage, a specific crunch appeared, which indicates a fracture of the ribs. What are your next steps?
continue heart massage, strictly setting the base of the palms at the border of the middle and lower third of the sternum;
continue heart massage without changing the position of the hands;

stop external heart massage, go to an open heart massage; stop external cardiac massage, carry out electrical defibrillation;

perform novocaine blockade of fracture sites and continue massage.

13. Patient 36 years old, delivered without immobilization after 3 hours. after falling from the height of the 3rd floor. Arrhythmic pulse more than 150 beats per min. on the carotid artery, blood pressure 40/0 mm. Hg, Cheyne-Stokes breathing. The hips are deformed, the stomach is moderately tense, painful. Make a preliminary diagnosis:

multiple, combined trauma, hip fractures, predagonal condition;

clinical death;

multiple, combined trauma, intra-abdominal bleeding;

fractures of both hips, traumatic shock of the III century;

multiple trauma, coma fractures of both thighs, traumatic shock of the II st.

Situational Tasks

1. The patient was admitted with severe subcutaneous emphysema of the upper half of the body and shortness of breath up to 30 min. Objectively: pulse up to 120 beats / min., Blood pressure 100/60 mm Hg, the skin was pale. Over the left half of the chest - tympanitis, breathing is not audible. Heart sounds are deaf, on a panoramic chest x-ray signs of pneumothorax, mediastinal displacement to the right. What emergency care should be given to the patient?

2. A medical sister should feed patient S., who was operated on 2 days ago for traumatic injuries of the oral cavity, traumatic shock of the first degree. Feeding should occur through a nasogastric tube prescribed to the patient. To do this, the nurse connected the Jenet syringe with the nutrient mixture to the probe and began to slowly inject the latter through the probe. At the same time, the patient began to complain of a feeling of fullness behind the sternum, pain in this area. Why did the patient have these complaints? What mistake did the nurse make?

3. Patient M., 69 years old, was operated on for diffuse peritonitis that developed as a result of polytrauma with damage to the small intestine. After the operation, 4 days have passed. The patient is allowed to eat through the mouth (table 1a). The patient's condition is serious, the patient is weakened, is in a supine position. How to feed this patient?

4. Patient V., 77 years old, 1 day ago an operation was performed - amputation of the left lower limb due to wet gangrene against the background of prolonged compression syndrome. The patient's condition is serious. She cannot move independently. On examination, hyperemia of the skin was found in the areas of the corners of both scapula, sacrococcygeal region. What complication can develop in this patient? What needs to be done in such a situation?

5. A patient diagnosed with "Politrauma" has been diagnosed with clinical death. When conducting a closed heart massage, a specific crunch appeared, testifying to a fracture of the ribs. What are your next steps?

6. The patient fell and received a chest injury. Upon admission, he complains of hemoptysis, shortness of breath, increased pain in the chest with deep breathing. Objectively: the skin is pale, respiratory rate - 30 per min., Pulse - 100 beats. / min., AD-110/60 mm. Hg On palpation, crepitus is determined along the posterior axillary line in the region of VI-VII ribs on the left. Percussion tympanitis over the left half of the chest. Auscultatory - the absence of respiratory sounds over the left half of the chest. What is the complication of injury in this patient?

8. Patient M., 77 years old, who is in a supine position, in the sacrococcygeal region there is skin hyperemia, blisters with bloody contents. What complication developed in this patient, what needs to be done in this case?

9. Patient K., 35 years old, was operated on 1 day ago for multiple traumatic injuries of the face, oral cavity, and both lips. How to feed this patient in the postoperative period?

10 When introducing a probe for gastric lavage, the patient developed an irrepressible severe cough. What could have caused this, what to do in this case?

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8. The distribution points are awarded to students:

At mastering topic number 15 to content module 4 for training activities for students rated a 4-point scale (traditional) scale, which is then converted into points as follows:

rating	**Points**
5 (excellent)	5
4 (good)	4
3 (satisfactory)	3
2 (poor)	0

Guidelines prepared
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