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Gomel State Medical University

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

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Topic on Combined intensive care

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

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Gomel 2020

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

1. Background.
2. The purpose of training (skills and knowledge).
3. Basic sections.
4. Suggested Reading.
5. Questions for self-study
6. Topics of educational and research work of students.
7. Learning material.
8. Self-study.
9. Case studies and test control.

Relevance of the topic

The dream of a return to life knead or deceased people have long worried. The timely and proper conduct of the cardio-pulmonary resuscitation (CPR), even by the witnesses - lay people, allows two or three times to reduce deaths from sudden cessation of cardiac activity and return to the normal life of the victims. At present, these methods are constantly improving. Therefore, medical students and doctors of all specialties should receive continuous information about new attitudes and achievements in this field. Mastering the elements of diagnosis of terminal states of emergency and intensive care techniques - the most important task of the medical school. On the quality of training and the number of people trained in these methods depends directly impact resuscitation.

Purpose of the lesson

The development of clinical signs of terminal states and principles of resuscitation and intensive care.

The student should know:

- the types of terminal conditions, their clinical manifestations
- pathophysiological mechanism of terminal states
- principles for a comprehensive cardio-pulmonary-cerebral resuscitation
- pharmacokinetic and pharmacodynamic characteristics of drugs used in cardio-pulmonary-cerebral resuscitation (CPCR)
- pathogenesis, clinical manifestations, intensive care of postresuscitative disease

The student should be able to:

- identify clinical terminal state
- justify the tactics for different reasons of death
- justify the termination of resuscitation
- conduct defibrillation
- apply basic groups of drugs in the complex CPCR

Sections studied before and needed for the session

- anatomy and physiology of the central nervous system, cardiovascular system, respiratory and excretory systems
- biochemistry of carbohydrate, protein and lipid metabolism
- Pathophysiology of dying
- basic pharmacology of drugs used for resuscitation

Recommended Reading

Books on anatomy, normal physiology, pathophysiology, pharmacology for medical students.

Suggested Reading on lessons:

Main Reading

1. Bunyatyan, AA Anaesthesia and Intensive Care / AA Bunyatyan [and others] Ed. AA

Buniatian. - M., Medicine. - 1994. - 565 p.

2. Dale, OA Anaesthesia and Intensive Care / O. Valley [and others] Ed. OA Valley - M., Medicine - 1998. - 574 p.

3. Lecture material.

Further Reading

1. Koryachkin, VA Critical Care threatening conditions / VA Koryachkin [and others] Ed. Ed. VA Koryachkina - Saint-Petersburg, Medical Publishing House. - 2002. - 283 p.

2. Sumin, SA Emergencies / SA, Sumin. - M., pharmaceutical world. - 2000. - 464 p.

Questions for self-study

Questions on basic knowledge

1. Pathophysiological mechanisms of dying body.
2. Biochemical abnormalities in terminal states.
3. Electrophysiological mechanisms of arrhythmias
4. Drugs used in cardio-pulmonary-cerebral resuscitation, their pharmacokinetic and pharmacodynamic characteristics.

Questions about the topic studied

1. Classification of terminal states: preagonic state, the agony, clinical death. Pathophysiology of terminal states. Clinic terminal states.
2. Circulatory arrest. Causes, precursors, symptoms, diagnosis. Types of heart failure. Clinic, diagnostics.
3. The concept of integrated cardio-pulmonary-cerebral resuscitation. Indications and contraindications.
4. Primary methods of cardiopulmonary-cerebral resuscitation. How to restore the airway. Methods of mechanical ventilation (MV): "mouth to mouth", "mouth-to-nose," Ambu bag type, the S-and T-shaped airway.
5. Cardiac massage. Massage types - direct (open), indirect (closed). Technique, complications. Performance indicators resuscitation.
6. Electropulse therapy: defibrillation, cardioversion, electrical pacing. Indications, technique, performance and complications.
7. Drug therapy. Pharmacology of the substances used for the restoration of the heart, the indications for their use. Dose, route of administration and procedure. Infusion therapy during resuscitation
8. Postresuscitation disease. Keeping patients in the early postresuscitation.
9. Biological death. Clinical signs of "brain death" biological death.

Topics of educational and research work of students

1. The history and formation of resuscitation as a science.
2. Academician VA Negovsky role in the global resuscitation.
3. Biological death - the transition or the end of existence?
4. Ethical-deontological problems of resuscitation.

Teaching tools for organization of independent work of students

1. Computer database.
2. Simulator for revitalizing the body, face masks, airways, defibrillator, Ambu bag.
3. Scheme for the first phase of resuscitation.
4. Slides: insertion of airways, laryngeal masks, intubation tubes.
5. Tables 1.1-1.8.
6. Objectives, test control.
7. Bank of tasks for self-study.
8. Medical history.
9. Thematic patients.

Training Material

Resuscitation - the science of reviving the body ("re" - again; «animare» - animate), pathogenesis, prevention and treatment of terminal states.

Terminal state - a short-term, a sharp weakening of the body's defenses leading to the dying. This condition may result from the development of any disease or serious injury, each of which has its nosological specificity. But the process of dying is common patterns unique to the terminal state as a kind of pathological forms. For a terminal condition characterized irreversible processes without intensive care.

Terminal conditions include three stages:

1. Preagonal state;
- Terminal break (since it does not always happen, - the classification is not included, but it is still worth considering);
2. Agonal state (agony);
3. Clinical death.

Pathogenesis.

Common pathophysiological link in thanatogenesis is the presence of some form of hypoxia, which then becomes mixed with a predominance of circulatory disorders. During the process of dying depends on which of the vital functions previously violated the original pathological process - breathing or blood circulation. In primary respiratory arrest hypoxia stops the heart and affects the brain, and vice versa - during the initial poor circulation heart stops first, then breathing and affects the brain.

Causes of respiratory failure may be asphyxia, ie disorders of the airway, ventilation disturbance of the central and peripheral origin, and lung disease, etc.

The causes of circulatory disorders (cardiac arrest): heart disease, severe hypovolemia, reflex effects, electrolyte disturbances, and others.

Original disease process in the body launches a series of compensatory and adaptive responses to restore blood flow and oxygenation of the central nervous system (CNS) in the form of more frequent and deeper breathing, increase circulation (increased minute volume of blood and blood pressure (BP)), centralized circulation by peripheral spasm. This leads to a temporary increase in blood supply to the central nervous system, myocardium. However, the hypoxia of other organs and tissues develops (kidney, liver, intestine, muscle, etc.). Under hypoxic conditions develops acidosis. Acidosis expands precapillaries - develops decentralized circulation. Amplified abnormalities in the blood system and hemostasis, which are responsible for violations of volume, viscosity and flow of blood, the formation of aggregates of its corpuscles, the phenomenon of sludge, blood clots. Prolonged stress compensatory mechanisms, especially the respiratory center (as in the work of the respiratory muscles consumes up to 50% of oxygen entering the body), leads to the depletion of energy resources and reserves of glycogen in the brain and depression of the respiratory center. Brain hypoxia begins to progress. Metabolism switches to anaerobic glycolysis pathway, disrupted production of ATP. This in turn affects the function of Na-K pump cell membrane, leading to a delay in cell sodium and water, ie, brain edema and paralysis neurons. If the supply of oxygen to the brain can not be restored, death is imminent.

Clinic.

Preagonal state lasts from minutes to days and is characterized by a sharp decrease in blood pressure, tachycardia, and tachypnea at first, and then bradycardia and bradypnea, progressive depression of consciousness, decreased electrical activity of the brain and brainstem reflexes due to increasing hypoxia. All this corresponds to the clinic torpid phase of shock. Especially typical of the change in the process of dying breath, as it reflects the functional state of the CNS at different levels of hypoxia involved in its regulation. In the beginning of dying due to cardiovascular reflexes the respiratory center is stimulated and breathing deepens and becomes more frequent as a result of increased activity of inspiratory muscles and the inclusion of the auxiliary muscles. But then, under the influence of hypoxia breathing slows down,

becomes superficial, there are various types of periodic breathing (Kussmaul, Cheyne-Stokes, Biot). At the end of the pre-agony, the activity of the exhalation center one disappear, as a more younger structure. Preagony ends with a terminal pause in respiration (primary anoxic apnea), a slowing of the pulse lasting several minutes, which indicates that all levels of respiratory regulation above the medulla oblongata are turned off and the tone of the parasympathetic nervous system, which is more resistant to hypoxia, is increased.

Agony - can last from several minutes to several hours and is characterized by the last outbreak of life. This occurs due to the inhibition of hypoxia of the vagal reflex and the restoration of the activity of the respiratory center. But breathing is rare, with a short breath and a quick active exhalation. As a result of the irradiation of excitation from the inspiratory center to the expiratory, as well as to the motor neurons of the skeletal muscles, which makes the inhalation active, there is a violation of the reciprocal relationship between the centers of inspiration and exhalation, which leads to the occurrence of inspiration and expiration at the same time, and breathing becomes completely ineffective. Cardiac activity, freed from vagal influence and activated by stem formations, temporarily intensifies, but then falls even more. Bradycardia, blockade of the conduction system develops. A temporary increase in blood pressure is accompanied by a short-term increase in the electrical activity of the central nervous system and even the restoration of conditioned reflex activity (sometimes consciousness) with subsequent suppression, i.e., the development of clinical death. A prolonged period of agony is subsequently unfavorable for the restoration of blood circulation. The increase in clogging of microcirculation vessels is a formidable phenomenon during resuscitation measures. All of these changes lead to cardiac arrest. The cessation of cardiac activity begins with the suppression of contractility (at first the left ventricle and in the same sequence - the atria), and then the conduction and excitability, but the electrical activity of the heart persists even after 30-60 minutes after it stops.

Clinical death is characterized by a complete absence of vital functions of the body (respiration, blood circulation and central nervous activity), inhibition of metabolic processes, but irreversible changes in the brain have not yet occurred. The duration of clinical death is determined by the ability of the brain to undergo reversible hypoxia and is 4-6 minutes. However, a decrease in body temperature to 10 - 8 degrees lengthens this period up to 2 hours. After which there comes an irreversible damage to the central nervous system - **biological death**, and the revitalization of the body, as an integrated system, is no longer possible.

Sudden cardiac arrest.

Clinical death may come as no prior long process of dying. The causes of sudden cardiac arrest can be divided into two groups - cardiac and extracardiac.

Cardiac causes: coronary heart disease (CHD), including acute myocardial infarction, angina, coronary spasm, arrhythmias of different nature and origin; electrolyte imbalance, valvular heart disease, endocarditis, myocarditis, cardiomyopathy, cardiac tamponade, pulmonary embolism, rupture and dissection of the aortic aneurysm.

Extracardiac causes: airway obstruction, acute respiratory failure, shock of any etiology; reflex cardiac arrest, embolism of different genesis and localization, overdose of drugs, electrical shock, injury to the heart, drowning, exogenous poisoning.

Mechanisms of sudden cardiac arrest. Cardiac output falls to zero or is very inefficient. Clinic sudden cardiac arrest can occur due to:

- 1) ventricular fibrillation or pulseless ventricular tachycardia
- 2) asystole
- 3) EMD (electromechanical dissociation)

According ambulatory Holter electrocardiography (ECG) at the time of sudden death, the latter, as a rule, due to ventricular fibrillation, and transforming it into ventricular arrhythmias (about 80%). During ventricular fibrillation on an ECG, instead of ventricular complexes, waves of various shapes and amplitudes are recorded, the frequency of which is 250-400 per minute. Depending on the amplitude of the waves, large and small-wave fibrillation is distinguished.

With large-wave fibrillation, the height of the waves exceeds 5 mm; with small-wave fibrillation, the wave amplitude does not reach this value. When ventricular flutter on the ECG, instead of the ventricular complexes, a sawtooth curve with rhythmic wide waves with a frequency of usually more than 250 per minute and without an isoelectric interval between them is observed.

More rarely a sudden cardiac arrest due to bradyarrhythmia turning into asystole heart. When asystole heart on an electrocardiogram recorded straight line, perhaps with occasional ventricular complexes or P wave.

Ventricular fibrillation and asystole are separated by gravity prior state of patients for primary and secondary. Mechanisms of sudden arrhythmic death are primary fibrillation and asystole, resulting in individuals who were in good or relatively good order, with no pronounced signs of heart failure, hypotension, and other aggravating symptoms. Atrial and ventricular asystole, which developed in patients with these symptoms, called secondary. They are the mechanisms are not sudden, but the so-called foreseen death of patients with various diseases.

A rare mechanism of sudden cardiac arrest is the *electromechanical dissociation* of the heart. With electromechanical dissociation, recorded the electrical activity on the ECG as a sinus node, idioventricular rhythms, and paroxysmal atrial fibrillation or tachycardia, not accompanied by effective contractile activity of the heart (ie, there is no pulse on the main arteries). Causes of electromechanical dissociation (pulseless electrical activity (PEA) may be called potentially reversible causes of cardiac arrest (4 "D" and 4 "T"): hypoxia, hypovolemia, hypo / hyperkalemia, hypothermia, cardiac tamponade, chest (tension pneumothorax), toxic / therapeutic disorders (overdose of tricyclic antidepressants, digitalis, β -blockers blockers, calcium channel blockers), thromboembolic / mechanical obstruction.

Clinic and diagnosis of sudden cardiac arrest. The clinic is partly dependent on the cause. In general, primary cardiac arrest patient loses consciousness (8-15 sec), possible convulsions (rare), breathing is disrupted (by airway obstruction and secondary depression of the respiratory center) and eventually fades away completely (some sighs, observed after 3 minutes). Skin color changes (pale, appearance gray or cyanotic color of skin, especially nasolabial triangle), the pupil dilates and does not respond to light (40-60 sec). A relaxation of all voluntary muscles with relaxing the sphincter. Involuntary urination, involuntary defecation. Diagnostics is carried out sequentially by ABC, i.e. determine contact, mark the time and activate assistance, unbend the head and extend the lower jaw, evaluate airway and breathing, pulse on the carotid artery, pupil. The most basic, early and sufficient sign is the absence of a pulse on the main arteries (carotid, femoral, in infants the pulse is checked on the brachial artery). To determine the tactics of restoring blood circulation, ECG monitoring is important, which allows you to determine the mechanism of circulatory arrest.

Integrated Cardiopulmonary-cerebral resuscitation (CPCR)

Indications for initiation complex CPCR is:

- lack or sudden loss of consciousness (assessed as cardiac arrest until proven otherwise),
- cardiac arrest,
- states with weak circulation or breathing.

Refusal of resuscitation is possible in decompensation irreversible pathological processes (such as stage IV malignant cancer) that are defined by objective research and documented in the medical record (medical card).

Treatment. In most cases the treatment is carried out in stages. Distinguish three phases.

Stage I: Basic maintenance of vital functions. Includes diagnostics stop and three points A, B, C.

A (airway open) - restoration and control of the airway.

B (breath for victim) - ventilation of the victim.

C (circulation his blood) - the artificial maintenance of blood circulation through the cardiac massage.

With the help of these measures, it is possible to provide a certain level of perfusion of the brain, heart and other organs with blood containing an acceptable amount of O₂. In rare cases, only these measures are enough to restore spontaneous circulation, but, as a rule, additional measures are required. In any case, the event of the 1st stage begins immediately and are used continuously as a base for all stages. It is desirable that the primary cardiopulmonary resuscitation possessed all adults. Doctors should be able to do it perfectly and be ready to teach.

Stage II: Restoration of spontaneous circulation, the stage of sustaining life. Also includes 3 points.

- 1) O₂, medications and fluids
- 2) ECG diagnosis of cardiac arrest type
- 3) cardioversion (most defibrillation, pacing is less).

You need to start this stage as early as possible. With this rare exception, 20-30 minutes are allotted to this stage, taking into account the time from the start of the stop. If during this time with the help of these measures it is not possible to restore spontaneous blood circulation, resuscitation is terminated. The fact is that oxygenation even with adequate mechanical ventilation and indirect heart massage is 6-30% of the due. So with indirect massage, the maximum cardiac output is 1.5 l / min, systolic blood pressure rises to 80 mmHg, but diastolic blood pressure is 0-40 mmHg, which causes a low level of brain (30-60% of normal) and coronary (5-20% of normal) blood flow. Such insufficient brain oxygenation during such a time leads to necrosis. In some cases, for example, with hypothermia (reduce the need for O₂), resuscitation takes much longer. If within 30 minutes from the start of resuscitation, despite all the measures taken, asystole or a rare idioventricular rhythm without a pulse is observed, then we can talk about "death of the heart", and not just about gross brain damage. Regarding ventricular fibrillation, there is a saying: "the heart is not ready to die if fibrillates," because as soon as the reserves of the heart are exhausted, the fibrillation fades into asystole. The duration of resuscitation in each case is considered individually (age, patient's condition before stopping, causes, duration of a complete stop, adequacy of resuscitation measures, etc.) with an understanding of what is happening. As a rule, after 30 minutes from a stop, gross brain damage occurs, but exceptions are possible.

If you are able to restore spontaneous circulation, begin Phase III - "further support life", "treatment postresuscitative disease", "cerebral resuscitation".

At stage III release as three points.

- 1) diagnosis and, if possible, treatment, the reasons for stopping (if it is not yet obvious, as with electrical injury, drowning, hanging) and the prevention of relapse.
- 2) diagnosis and treatment of complications arising after stopping (aspiration) and in the process resuscitation (injury from massage, problems with central venous catheterization, aspiration, etc.)
- 3) proper maintenance of a long life:
 - stabilization of the transport systems of O₂ (cardiac output (CO) and blood gases) and the entire homeostasis (blood glucose, osmolarity, acid-base balance, electrolytes, proteins, etc.)
 - proper cerebral resuscitation (treatment of cerebral edema, correction of intracranial pressure (ICP)).

In some cases, the sequence may vary somewhat. This applies to cardioversion. Defibrillation is more effective the sooner it is done (while large-wave fibrillation).

Stage I - basic life support (BLS)

Airway (A)

Gold standard for airway management are triple maneuver of P. Safar (extension of the head, removal of the lower jaw forward, mouth opening), and endotracheal intubation. It should be noted that, according to the study, tracheal intubation in patients with circulatory arrest is associated with a delay in chest compression lasting an average of 110 seconds.

As an alternative to endotracheal intubation, it is recommended to use technically simpler compared with tracheal intubation, but at the same time reliable methods of respiratory tract protection:

a) the use of a laryngeal mask, however, it must be remembered that in comparison with tracheal intubation, the risk of developing aspiration is increased. In this regard, in order to reduce the risk of aspiration, it is necessary to pause chest compression during mechanical ventilation (mechanical ventilation) through the laryngeal mask. In addition to standard laryngeal masks, it is allowed to use the I-gel mask, which has a larynx-like non-inflating cuff of a thermoplastic elastomeric gel, the formulation of which requires basic skills;

b) use of a double-lumen Combitube airway; with this method of ensuring airway patency, it will be guaranteed for any location of the airway — both in the esophagus and in the trachea;

c) if intubation is not possible, then a trained person can use a naso- or oropharyngeal airway. Oral and nasopharyngeal airways are easy to install with minimal experience. The most common and simplest to install is the Guedel oropharyngeal airway. The oropharyngeal airway has dimensions corresponding to the distance from the angle of the mouth to the angle of the lower jaw. The nasopharyngeal airway before administration should be well lubricated and equal to the diameter of the victim's little finger. Do not use the nasopharyngeal duct if a skull base fracture is suspected.

If necessary, you must also clean the oral cavity and oropharynx from foreign bodies (or use Heimlich's technique). With irremovable blockade of the upper respiratory tract, cricothyrotomy is performed.

Rules of mechanical ventilation (B)

Mechanical ventilation can be carried out:

- «mouth to mouth" or "mouth-to-nose"
- manual methods (Ambu bag mask, Ambu bag via an endotracheal tube)

During cardiopulmonary resuscitation (CPR), artificial respiration is started with two breaths. Each breath should last 1 sec. In order to avoid over-inflation of the lungs, the second breath begins only after exhalation has occurred, i.e. air out of the lungs. In order to ensure adequate ventilation, the resuscitator should tightly cover his mouth with his lips (pinched nose) or the patient's nose. If the patient's head is not sufficiently bent, then the airway is impaired, and air enters the stomach. Respiratory rate - 8-10 in 1 min, i.e. one respiratory cycle every 6-8 sec. The volume of inflated air depends on the age, constitutional features of the patient and is 400-600 ml (6-7 ml / kg) for adults. Too much inflated air increases the pressure in the oropharynx, increases the risk of bloating, regurgitation and aspiration; barotrauma; increases intrathoracic pressure, reduces venous return to the heart and reduces cardiac output.

Mechanical ventilation is carried out very carefully and methodically in order to avoid complications. When conducting mechanical ventilation, it is still possible to inflate the stomach. The prevention of this complication in the absence of tracheal intubation is achieved by maintaining the airways open, not only during inspiration, but also during passive exhalation. When conducting mechanical ventilation, you can not press on the epigastric region: with a full stomach, this causes vomiting. If, nevertheless, the contents of the stomach are thrown into the oropharynx, it is recommended to turn the resuscitated one on its side, clean its mouth, and then turn it on its back and continue CPR.

It is strongly recommended that protective equipment be used to reduce the risk of transmission of disease. When breathing from mouth to mouth or from mouth to nose, a face mask or protective film is used. During mouth-to-mouth breathing, there is the likelihood of hepatitis B infection. In human immunodeficiency virus or as a result of CPR is minimal, there is a risk of transmission of herpes simplex virus, meningococcus, mycobacterium tuberculosis and some other pulmonary infections, although also very slight. If the patient is suspected of using contact poisons or if he has infectious diseases, the carer should protect himself from direct contact with the victim and use additional devices (airways, Ambu bag, masks) for mechanical

ventilation, which have valves directing passively exhaled air away from the resuscitator . Optimal is endotracheal intubation. This is the final stage of recovery and full provision of airway patency: reliable protection against aspiration, prevention of gastric expansion, effective ventilation.

Signs of adequate ventilation.

When air is injected into the lungs, the chest rises and expands. During exhalation, air comes out of the lungs (listened to by the ear), and the chest takes its former position. Pressure on the cricoid cartilage in order to prevent air from entering the stomach and regurgitation (Selick maneuver) is recommended only for people with medical training.

Artificial maintenance of circulation (C)

A precordial thump is performed when a resuscitator directly observes the onset of ventricular fibrillation / ventricular tachycardia (VF / VT) on a cardiac monitor without a pulse, and the defibrillator is currently unavailable. It makes sense only in the first 10 seconds of stopping blood circulation. The victim is laid on a hard surface. The index finger and middle finger must be placed on the xiphoid process. Then, with the edge of a palm clenched into a fist, hit the sternum above the fingers, while the elbow of the striking hand should be directed along the body of the victim. If after this the pulse on the carotid artery does not appear, then it is advisable to switch to indirect heart massage. Contraindication - the child is less than 8 years old, body weight less than 15 kg.

According to the results of the work, a precordial thump sometimes eliminates the VF / VT without a pulse (mainly VT), but is most often ineffective and, conversely, can transform the rhythm into asystole. Therefore, if the doctor has a defibrillator ready for use, it is better to refrain from precordial shock. Precordial thump is also not recommended at the prehospital stage.

Chest compressions. Blood circulation can be restored by clicking on the chest. In this case, the heart is compressed between the sternum and the spine, and the blood is pushed out of the heart into the blood vessels. Rhythmic compressions mimic heart contractions and restore blood flow. This massage is called indirect because the lifesaver acts on the heart through the chest.

The victim lies on his back, always on a hard surface. If he is lying on the bed, he should be laid on the floor. Clothes on the patient's chest are unfastened, freeing the chest. The rescuer stands (in full growth or on his knees) on the side of the victim. He puts one palm on the lower half of the patient's sternum so that the fingers are perpendicular to it, the compression point is two to three transverse fingers above the base of the xiphoid process. The other hand is on top. Raised fingers do not touch the body. The lifesaver's straight arms are perpendicular to the victim's chest. Massage is done with the help of quick strokes weighing the whole body without bending the arms at the elbows. In this case, the patient's chest should bend 4-5 cm. During squeezing, hands should not be torn off the sternum. The displacement of the base of the palms relative to the sternum is unacceptable. For newborns, an indirect heart massage is performed with one finger. Babies with two fingers, older children with one palm. Depression is 1/3 of the height of the chest.

The main problem of artificially maintaining blood circulation is a very low level (less than 30% of the norm) of cardiac output (SV), created by squeezing the chest. With compression of the chest, coronary perfusion pressure rises only gradually, and, therefore, with each subsequent pause necessary for breathing from mouth to mouth, it rapidly decreases. However, several additional compressions lead to the restoration of the initial level of cerebral and coronary perfusion. In this regard, it was shown that the ratio of the number of compressions to respiratory rate, equal to 30: 2, is the most effective:

a) the ratio of the number of compressions to the respiratory rate without protecting the respiratory tract or with the protection of the laryngeal mask or respiratory tract of Combitube for one or two resuscitators should be 30: 2 and should be performed with a pause for mechanical ventilation (risk of aspiration!);

b) with respiratory tract protection (tracheal intubation) - chest compression should be performed at a frequency of 100 / min, ventilation should be performed at a frequency of 10 / min (in case of using an Ambu bag - 1 breath every 5 seconds) without a pause during mechanical ventilation (since squeezing the chest while simultaneously inflating the lungs increases coronary perfusion pressure).

To facilitate long-term CPR, mechanical chest compression devices are recommended.

The effectiveness of indirect massage is controlled by a second resuscitator performing mechanical ventilation, pulse on the carotid arteries.

Complications of indirect heart massage.

Possible complications of indirect heart massage include fractures of the ribs, sternum, ruptures of the rib-sternum joints, pneumothorax, hemothorax, ruptures of the liver and spleen, fat embolism. Careful adherence to indirect heart massage techniques reduces the risk of these complications, but does not exclude them.

Direct heart massage.

In children and young people, the mobility of the sternum is very high, while in the elderly it can be severely limited. With sternum deformities, the heart cannot be effectively pressed against the thoracic vertebrae. If the heart is displaced from its anatomical middle position between the sternum and vertebrae, then indirect massage is not effective. Spinal deformities (lordosis, kyphosis and scoliosis) can also interfere with indirect cardiac massage. In such cases, direct massage is performed. Direct massage is also carried out during intrathoracic operations, with suspected cardiac tamponade, intense pneumothorax, pulmonary embolism.

For direct cardiac massage under aseptic conditions, a thoracotomy is performed in the fifth intercostal space. The fingers of the left hand are placed on the back wall of the ventricles. The palm should cover the apex of the left ventricle. The thumb (I) of the left hand should be located on the front wall of the left ventricle, the index (II) and middle (III) fingers of the right hand are across the back wall of the ascending aorta, about 7 cm above the aortic valves, and I finger of the right hand is on the front aortic wall. Sequential ventricular-aortic compression causes a greater increase in blood flow in the coronary sinus and cerebral perfusion compared to conventional resuscitation methods.

On October 18, 2010, the European Resuscitation Council published the new European Recommendations for Cardiopulmonary Resuscitation (ERC 2010). They are based on new scientific data obtained since the preparation of the previous review five years ago. The most important measure for resuscitation is compression of the sternum. Such actions can be performed by any person, even a child. A simple and safe procedure significantly increases the victim's chances of life. For nonmedics, when finding a compression point, it is possible to position the hands in the center of the chest, between the nipples. Chest squeezing is an extremely important measure, even without the use of artificial respiration. Therefore, the sequence of resuscitation was changed to "C-A-B." Witnesses of the event who have special training and want to help should chest massage in combination with artificial respiration, in a ratio of 30 compressions to 2 breaths.

In the past 10 years, the use of automatic external defibrillators (AED) has also become more widespread in Europe. These devices not only allow you to determine the need for defibrillation and discharge power, but are usually also equipped with voice instructions for the entire cycle of cardiopulmonary resuscitation. These defibrillators are installed in the most crowded and most visited places, since the effectiveness of defibrillation drops sharply after 7 minutes after the occurrence of circulatory failure.

The standard method of using the AED is as follows: when a person is found unconscious and an ambulance is called, disposable electrodes are applied to the skin of the chest (you don't even have to spend time checking the pulse and pupils). On average, after a quarter of a minute, the device (if there is evidence for discharge) offers to press a button and defibrillate or (if there is no indication) start an indirect heart massage / artificial respiration and starts the timer. The rhythm analysis is repeated after discharge or after the standard time allotted for CPR.

This cycle continues until the arrival of the medical team. When the heart is restored, the defibrillator continues to work in observation mode.

Table 1. Key elements of the basic resuscitation adults, children and infants *

	Recommendations		
element	adult	children	babies
Recognizing	Unconscious		
	Not breathing or breathing is not right	Does not breathe or single breaths	
	Pulse is not detected for 10 seconds (For medical staff)		
The sequence of CPR	C-A-B		
Frequency of compressions	Least 100 compressions in one minute		
Depth of compression	Not less than 5 cm	Not less than one-third of the diameter of the chest (approximately 5cm)	Not less than one third of the diameter of the chest (about 4 cm)
Chest expansion	Full chest expansion between contractions. Compressors change every 2 minutes.		
The intervals between contractions	Intervals are minimal (less than 10 seconds)		
Airway	Tilting the head and raising the chin (in case of suspected injury - extension of the lower jaw)		
The ratio of compression-breath (before intubation)	30:2 1 or 2 rescuer	30:2 1 Recovery 15:2 Two rescuer	
MV (If not trained)	Only compressions		
Ventilator with endotracheal tube	1 breath every 6-8 seconds (8-10 breaths per minute) Asynchronously with compression About 1 second to breath Visible chest excursion		
Defibrillation	As soon as possible, apply and use the AED. Shorten the intervals between contractions before and after discharge. Continue with CPR after each discharge		
* Note: with the exception of newborns whose cardiac arrest is associated with asphyxia			

II stage - Advanced Life Support (ALS)

Route of administration of drugs.

Endotracheal way. According to the recommendations of ERC'2010, the endotracheal route of administration of drugs is no longer recommended. Studies have shown that in the process of CPR, the dose of adrenaline administered endotracheally, which is equivalent to the dose when administered intravenously, should be 3 to 10 times greater. At the same time, a number of experimental studies indicate that low concentrations of adrenaline during the endotracheal route of administration can cause transient β -adrenergic effects, which lead to the development of hypotension and a decrease in coronary perfusion pressure, which, in turn, worsens the effectiveness of CPR. In addition, injected endotracheally large volumes of fluid can degrade gas exchange. In this connection, the new recommendations use two main accesses for drug administration:

a) **the intravenous route** to the central or peripheral veins. The determining method is central treatment. Drugs should be diluted in 20 ml of saline;

b) **the intraosseous pathway** - the intraosseous injection of drugs into the humerus or tibia providing an adequate plasma concentration, comparable in time with the introduction of drugs into the central vein. It is most often used in pediatrics when it is impossible to find the peripheral vein. The tibia is within the internal cavity of the body. In children, the cavity in the tubular bones is not yet filled, both in adults and anastomose with veins. The use of mechanical devices for intraosseous administration of drugs allows for an almost acceptable rate of infusion. You can put a catheter into the central vein (connected, poor).

Pharmacological support of resuscitation

1. Adrenaline:

- a) with electrical activity without a pulse / asystole (EABP / asystole) - 1 mg every 3-5 minutes intravenously;
- b) with VF / VT without pulse, adrenaline is injected only after the third ineffective discharge of electrical defibrillation at a dose of 1 mg. Subsequently, this dose is administered every 3-5 minutes intravenously (i.e., before every second defibrillation) for as long as VF / VT without pulse. The higher the wave amplitude during fibrillation, the higher the probability of success with defibrillation. The amplitude increases with improved oxygenation of the heart and the introduction of adrenaline.

As an alternative to epinephrine, vasopressin in a dose of 40 units can be used, once intravenously.

2. Atropine. In cases of severe bradycardia, atropine injection can restore adequate blood circulation by reducing the tone of the vagus nerve. When the administration of atropine does not give an effect for 1-2 minutes, the administration of adrenaline is indicated. Dosage: 0.5-1 mg of atropine intravenously, if necessary, re-administered after 3-5 minutes to a total dose of 0.04 mg / kg (2-3 mg). Higher doses (single dose - 1 mg) are indicated for severe bradycardia.

The use of atropine during CPR is no longer recommended. Studies have shown the absence of the effect of atropine during circulatory arrest by the mechanism of EABP / asystole.

3. Amiodarone is a first-line antiarrhythmic drug in VF / VT without pulse, refractory to electro-pulse therapy after the 3rd ineffective discharge, in an initial dose of 300 mg (diluted in 20 ml of physiological saline or 5% glucose), if necessary, re-enter 150 mg before each subsequent discharge of the defibrillator. After the restoration of self-circulation, it is necessary to provide intravenous drip of amiodarone at a dose of 900 mg in the first 24 hours of the postresuscitation period in order to prevent refrillation.

4. Lidocaine - in the absence of amiodarone (while it should not be used as a supplement to amiodarone) - an initial dose of 100 mg (1-1.5 mg / kg) iv, if necessary, an additional bolus of 50 mg (at the same time total dose should not exceed 3 mg / kg for 1 hour).

5. Sodium bicarbonate. Routine use during CPR or after restoration of self-circulation is not recommended.

Circulatory arrest is a combination of respiratory and metabolic acidosis. The optimal method for correcting acidemia during circulatory arrest is to compress the chest, an additional positive effect is provided by ventilation.

The routine administration of sodium bicarbonate during CPR due to the generation of CO₂ diffusing into the cells causes a number of adverse effects:

- increased intracellular acidosis;
- negative inotropic effect on the ischemic myocardium;
- circulatory disturbance in the brain due to the presence of highly osmolar sodium;
- shift of the oxyhemoglobin dissociation curve to the left, which can reduce oxygen delivery to tissues.

Indications for the introduction of sodium bicarbonate are cases of circulatory arrest associated with hyperkalemia or an overdose of tricyclic antidepressants at a dose of 50 mmol (50 ml - 8.4% solution) iv.

6. Calcium chloride - at a dose of 10 ml of a 10% iv solution (6.8 mmol Ca²⁺ +) with hyperkalemia, hypocalcemia, an overdose of calcium channel blockers.

7. Magnesium sulfate. The drug is especially necessary if circulatory arrest has occurred on the background of hypomagnesemia. The latter can be assumed with hypokalemia, treatment with diuretics, digoxin. It has an antiarrhythmic effect, the introduction of the drug is indicated for stable VF / VT. The initial dose is 2 g per 40-50 ml of a solution of 5% iv glucose in a bolus for 1-2 minutes. If there is no effect, the administration is repeated after 10-15 minutes.

8. For infusion therapy, saline solutions without glucose are most often used (hyperglycemia with insufficient oxygenation has a damaging effect), "quick drops". When circulatory arrest is an empty heart type, infusion therapy is crucial. Use crystalloids, colloids, blood and its preparations. With blood loss, it is possible to use up to 500 ml of blood from a universal donor. I (0) Rh "-". Use rapid infusion of several veins through needles or catheters with a wide clearance.

9. Oxygen therapy. In order to successfully fight hypoxia, it is necessary to use high (up to 100%) oxygen concentrations as soon as possible. Mechanical ventilation with high concentration of oxygen through a mask or endotracheal tube reduces hypoxemia in conditions of minimal circulation.

Electropulse therapy.

Defibrillation is a method of interrupting ventricular fibrillation. Most often, this term refers to electrical defibrillation (although chemical and mechanical are possible). Electrical defibrillation is a form of electropulse therapy (along with cardioversion and pacemaker). The essence of the method is to bring a powerful email to the heart. discharge. With ventricular fibrillation, each myocardial myofibril is reduced asynchronously (at the heart of electronic processes is the Re-entry phenomenon). Powerful email. the discharge leads the entire myocardium simultaneously to a state of depolarization, after which the myofibrils exit this state at the same time. If after this the sinus node generates a pulse, then its further propagation will go in the usual way. Those. defibrillation stops the electric chaos, literally stopping the heart so that it again beats efficiently. Spontaneous fibrillation does not pass, but only fades into asystole. The earlier defibrillation is performed, the more effective it is; this is a priority in resuscitation. Currently, only direct current is used for defibrillation. There are monopolar and bipolar impulses. Distinguish between external and internal defibrillation (when the electrodes are applied directly to the heart). With internal defibrillation, the discharge is much smaller (almost 10 times). Defibrillation is indicated for paroxysmal ventricular tachycardia or for ventricular fibrillation. With external defibrillation, the plates are well lubricated with a special gel to reduce electric. resistance or apply napkins moistened with nat. solution (less often). One electrode is "placed on the right under the collarbone -", and the second "+" is not the top of the heart (somewhat more lateral than the nipple). On many defibrillators on the plates APEX is written - the top, STERNUM - the sternum, so as not to confuse. When the desired charge has accumulated on the defibrillator capacitor, which is judged by a special signal, chest compression (impedance decreases) and a discharge is produced after the "everyone has departed" command. It is important for the prevention of electrical injury so that no one touches the patient directly or through any conductor.

The first-discharge energy currently recommended by ERC'2010 should be 360 J for *single-phase defibrillators* (they are no longer being produced), both for the first and all subsequent discharges (children: 3-5 J / kg). The initial energy level *for biphasic defibrillators* should be 150-200 J for the 1st discharge, followed by an energy escalation of up to 360 J with repeated discharges. The research results showed that biphasic defibrillation using less energy is much more effective and to a lesser extent causes damage and postresuscitative myocardial dysfunction compared to the equivalent energy of a monophasic pulse.

Immediately after applying the defibrillator discharge, it is necessary to continue compression of the chest and other components of CPR for 2 minutes and only then conduct an

ECG rhythm assessment, if sinus rhythm is restored, assess its hemodynamic effectiveness by the presence of a pulse on the carotid and radial arteries (by simultaneous palpation of these vessels). Even if defibrillation is effective and restores, according to ECG, the sinus rhythm, it is extremely rarely immediately after defibrillation that it is hemodynamically effective (i.e., capable of generating a pulse, and hence blood circulation). It usually takes about 1 minute of chest compression to restore self-circulation (pulse). When restoring a hemodynamically effective rhythm, additional compression of the chest will not cause re-development of the VF. Conversely, in the case of restoration of only organized bioelectric activity of the heart, but hemodynamically ineffective, the cessation of chest compression will inevitably lead to ventricular refrillation. The above facts justify the immediate start of chest compression after applying a defibrillator discharge for 2 minutes and only subsequent assessment of the rhythm by ECG, and in the case of restoration of the sinus rhythm, pulsation assessment in the carotid and radial arteries. The interval between defibrillation discharge and the beginning of chest compression should be less than 10 seconds. Cardioversion is a type of EIT. The principle of action, as with defibrillation. The difference is that discharges of lower energy are used, but synchronously with the cardiac cycle. It has been shown that cardioversion in tachyarrhythmias is based on the re-entry mechanism (atrial fibrillation, atrial flutter, supraventricular tachycardia paroxysm, etc.). A smaller discharge causes a lesser side effect on the myocardium. And synchronization is needed in order to ensure the exact hit of the pulse in the appropriate place in the cardiac cycle. Otherwise, when the impulse, for example, enters the so-called vulnerable area (for example, the T wave), ventricular fibrillation.

Cardioversion along with drug therapy is used during resuscitation for the treatment of secondary (after stopping) arrhythmias. Technically, cardioversion is performed as follows, you must first apply the ECG electrodes of the monitor-defibrillator to the patient, select the appropriate lead for a clear visualization of the QRS complex, and press the "synchronization" button. After that, further actions as with defibrillation. But the discharge will not happen immediately as you press the "discharge" button, but automatically in synchronization with the cardiac cycles. For cardioversion with atrial flutter, sometimes 5–25 J discharges are required, which is incomparable with a discharge during defibrillation (The following scheme is usually used: 50 → 100 → 200 → 300 J). If the patient is conscious, sedation or anesthesia is necessary. Often, the success of EIT is better against the background of drug antiarrhythmic therapy. Adequate oxygenation and ventilation, pH and electrolyte levels are crucial.

Cardiac pacing (CP) is a type of EIT for the treatment of bradyarrhythmias against the background of low automatism and conduction disturbances. Sometimes it is used for asystole. When asystole is not a transient disorder due to disorders of the functions of automatism (for example: AMI, poisoning with some poisons), but is a reflection of the irreversible process of "dying" of the heart, pacemaking is not effective and is not shown. During resuscitation, external cardiac pacing is used, in the absence of the effect of drug therapy for grade III AV block, and sometimes with asystole.

With external CP (pacing), one electrode is placed on the top of the heart, and the other under the scapula. The pace frequency is set, and then it is selected according to the increasing current strength, which ensures reliable excitation of the ventricles. To assess the adequacy of pacing, it is necessary to monitor the ECG. The current strength with external CP is large, which can cause a reduction in the patient's muscles, as well as pain and discomfort, when the patient regains consciousness against the background of pacing. If necessary, sedation and analgesia are used. After stabilization, they switch to a more complex method - internal endocardial pacemaking. In both external and internal CP, the demand mode is possible, i.e. inclusion on demand, when stimulation is activated only with paroxysms of inadequate rhythm, when "own rhythm" is stable, stimulation is not performed.

Monitoring during CPR

In the process of CPR, it is necessary to monitor the effectiveness of ventilation of the lungs, electrocardiographic monitoring of heart rhythm, skin color, the presence and frequency

of the pulse on the main arteries, the size and reaction of the pupils to light, muscle tone, the presence of attempts to spontaneously breathe, the simplest reflexes (ciliary, corneal, reaction to pain), level of consciousness.

Additionally, capnometry (capnography) is carried out, a study of body temperature, blood gases, indicators of CBS, hemoglobin, electrolytes (Na, K, Mg), glucose.

Termination of resuscitation

CPR must be performed for as long as ventricular fibrillation is maintained on the ECG, since it minimizes metabolism in the myocardium, which provides the potential for the restoration of self-circulation.

In case of circulatory arrest by the EABP / asystole mechanism in the absence of a potentially reversible cause (according to the "four G - four T" algorithm requiring etiological treatment), CPR is performed for 30 minutes, and if it is ineffective, it is stopped.

CPR is performed for more than 30 minutes in cases of hypothermia, drowning in ice water, and an overdose of drugs.

The cessation of resuscitation is recorded as the time of the biological death of the patient.

III stage (stage long-term maintenance of life)

According to the National Registry for Cardiopulmonary Resuscitation USA (National Registry of Cardiopulmonary Resuscitation - NRCPR), among 19,819 adults and 524 children after restoration of spontaneous circulation hospital mortality rate was 67 and 55%, respectively. According to epidemiological studies in the UK, including 24,132 resuscitated patients in the UK mortality rate was 71%. It should be noted that among the survivors only 15-20% have rapid restoration of adequate level of consciousness, the other 80% of the patients go through postresuscitation disease. The causes of death in the postresuscitation stage: 1/3 - cardiac (highest risk in the first 24 postresuscitation hours), 1/3 - dysfunction of various extracerebral organs and 1/3 - neurological (causes of death in the long-term postresuscitatorial disease).

Postresuscitation disease

In case of successful resuscitation restoration of central nervous system functions occurs in reverse order compared to the dying, primarily begins operations more ancient system of the central nervous system - the center of inhalation through the resumption of blood circulation in the medulla oblongata. Early appeared breath demonstrates and promotes more stable and complete recovery of the remaining functions. In the beginning there some breaths agonistic type (convulsive character) and then breathing becomes normal character. The first begins operation inspiratory center, later - expiratory, which coincides with the restoration of corneal reflexes. Later than other parts of the CNS restores function of the cortex. Cardiac resuscitation: first - function automatism, excitability and conductivity, and then (later) - contractility, which is also associated with resistance older ontogeny and phylogeny formations to hypoxia. If the recovery creates a sufficient blood circulation and gas exchange, then the recovery of the heart function is a gradual recovery of central nervous system functions. However, disturbance of microcirculation, acidosis and associated brain edema violates the oxygen supply, which may be the cause of new lesions - postresuscitatorial disease.

Postresuscitation disease - a syndrome of multiple organ failure in the early postresuscitation stage arising in connection with the formation of deep hypoxia, especially circulatory nature, in all organs and tissues (primarily the central nervous system, liver, kidney, lung, and bowel infarction) during a terminal condition and resuscitation, and by pathological intoxication products of metabolism during this period.

Stages (periods) of postresuscitatorial disease:

Stage I - the period of unstable functions - for the first 6 - 10 hours of recovery. It is characterized by hypovolemia, small venous blood flow to the heart, centralization of blood

circulation, insufficiency of peripheral blood flow, ischemia and tissue hypoxia. External respiration is activated compensatory with the development of hypocapnia against the background of preservation of blood oxygenation. Circulatory hypoxia, acidosis, respiratory alkalosis leads to cell transmineralization, accumulation of Na⁺, cell edema, and blood thickening; aggregation and hypercoagulation (DIC).

Stage II - relative stabilization of functions (period of imaginary well-being) is characterized by a temporary improvement in the patient's condition - after 10-12 hours of treatment. But at the same time, microcirculation, metabolism, organ failure (acute renal failure (ARF), and so on) persist. Optimism is premature.

Stage III - re-deterioration, even if patients are then recovered. It starts at the end of the 1st beginning of the 2nd day of treatment. The most characteristic feature is the appearance of arterial hypoxemia due to the development of "shock lung" as a result of DIC, may develop acute renal failure (oliguria).

Stage IV - a period of improvement or progression of complications - to 3 - 5 days of joining and purulent inflammatory changes in the foci of ischemic and hypoxic cell damage.

Pathogenetically substantiated therapy is done with the characteristics of each of the steps that can prevent or ameliorate the symptoms of the postresuscitatory disease.

In Stage I - the main task - stop bleeding (e.g. surgery), restore blood volume (transfusion), the maintenance of respiratory function (oxygen and mechanical ventilation). In general, treatment is consistent both with and decompensated shock (hypovolemia eradication, improvement of blood rheology, anti-aggregation inhibitors of proteolysis, the treatment of DIC based on the stage). Required catheterization of the vessel. Monitor the impact on blood pressure, central venous pressure, urine output, blood gas and acid-base status. Protecting the brain from hypoxia (craniocerebral hypothermia, antihypoxants - sodium oxybate, barbiturates, benzodiazepines, cytochrome C).

In stage II - prolonged mechanical ventilation with O₂ for 4 - 6 hours, oxygen and improved tissue perfusion. Continued transfusion therapy, hormones (in high doses), antispasmodics (peripheral and central), antiplatelet agents (Courant, Trental). Prevention posthypoxic brain edema and acute renal failure (transfusion mannitol). Restores energy losses and hypokalemia (transfusion polarizing mixture).

In stage III - besides the above prevention and treatment of "shock lung", i.e., respiratory failure. Heparin (under the control of heparin clotting reduced by 2 times), fibrinolysis. Oxygen therapy. The use of parenteral nutrition. Antibiotic therapy.

In stage IV - reduces the amount of transfusions, the transfer to enteral nutrition, treatment of inflammatory and suppurative processes, and organ failure. Antibiotics, hyperimmune drugs. Control and correction of plasma osmolality (normal - 270 - 310 mmol/L).

Monitoring in postresuscitation stage. Patients with a history of heart failure, should be closely supervised. The first thing to take into account the full clinical assessment of the status of the patient and the data of non-invasive methods. Indications for other, more complex methods (intracranial pressure monitoring, measurement of pulmonary artery wedge pressure (PAWP), etc.) should be strongly argued.

Monitoring of the cardiovascular system includes continuous monitoring of blood pressure, heart rate, central venous pressure (CVP), ECG, blood volume. It is important to prevent hyperperfusion and maintain normotension. To eliminate stasis microcirculation recommended: light hypertension for a short time, the use of rheological and moderate hemodilution. It is important to detect and eliminate cardiac arrhythmias, depending on the initial pathology (ischemia, AV block, etc.) and catecholaminin-induced disorders associated with the use of inotropic and other means. ECG diagnosis of arrhythmia requires a clear interpretation of P-wave and the complex QRS (V, and standard lead II). To detect ischemia these indicators are not enough. Hidden episodes of ischemia may go unnoticed. Chest leads V5 or evidence of ischemia modified septum and left lateral wall, and the bipolar lead II limb ischemia shows the lower part of the myocardium in the pool right coronary artery.

Important information obtained by measuring the parameters of central hemodynamics. In order to prevent re-VF after successful cardioversion, and for the treatment of multiple ventricular premature beats can be assigned to amiodarone or lidocaine as an intravenous infusion of 1-4 mg / min. When bradyarrhythmia (sinus bradycardia, complete AV block) is not curable atropine, pacing may be required, especially in cases where the AV block or slow idioventricular rhythm accompanied by hemodynamic disorders. In cardiogenic shock, caused by reduced pumping function of the heart, dobutamine (3-12 mcg / kg / min) and dopamine (2 - 10 mg / kg / min) are indicated.

Target values necessary to achieve postresuscitation:

- MAP (mean arterial pressure) - 70 - 90 mm Hg;
- CVP - 8-12 cm H₂O;
- Hemoglobin -> 100 g / L;
- lactate - <2.0 mmol / L;

Monitoring of the respiratory system. In postresuscitation stage important to reduce the concentration of O₂ in inspired air to 94% or lower, if possible, to avoid the consequences hyperoxygenation. Necessary to maintain PaO₂ at near 100 mm Hg. The recommended level of PaCO₂ - 25-35 mm Hg, and an increase in intracranial pressure - on average 25 mm Hg. Correction of blood gases is achieved by mechanical ventilation in the mode of a small positive end-expiratory pressure (PEEP). Ventilator continues to full critical functions (consciousness, adequate spontaneous respiration, stable hemodynamics).

Fluid and electrolyte and acid-base balance. To monitor the amount of fluid infused, urine output and possible extrarenal losses. Infusion isotonic electrolyte solutions are recommended in combination with non-aqueous 10% glucose solution. Hematocrit maintained at 0.30-0.35, plasma osmolality and its content of electrolytes and glucose - within limits. Mild metabolic acidosis is valid and desirable (pH = 7,25-7,35), because in these conditions it is better oxygenizing tissue and increases cardiac output. In addition, the level of K⁺ in serum is often reduced after successful resuscitation, and excess correction of acidosis may enhance hypokalemia, and lead to a new cardiac arrest.

Monitoring of neurological function. Most important link in therapy - physiotherapy for CNS.

Sound basis for monitoring CNS in patients after cardiac arrest are the Glasgow Coma Scale with reaction eye opening, motor and verbal response, combined with data of the EEG. For therapeutic purposes, "to protect the brain" shows high doses of corticosteroids. When the EEG activity and shows a tendency to seizures diazepam (Seduxen, Valium, Relanium, Sibazon) - anticonvulsant, sedative. In marked seizures - sodium thiopental (5 mg / kg), muscle relaxants.

According to the testimony - sedatives and analgesics.

Currently, hypothermia is the most promising method of protection neuroprotective brain. Revealed that the decrease in body temperature by 1 ° C in the average decreases the rate of cerebral metabolism by 6-7%. According to current guidelines, all patients unconscious, suffered a cardiac arrest, you need to ensure that therapeutic hypothermia (TH) of the body up to 32-34 ° C for 12-24 hours. Method - external cooling with hypothermia or an intravenous infusion of normal saline or Ringer's lactate solution (4 ° C) at a dose of 30 ml / kg at a rate of administration of 100 ml / min. For the relief of cold shivering – analgesic, sedation, muscle relaxants, the use of vasodilators (nitrates), a slow warming - not faster 0,2-0,5 ° C / h. Then the constant maintenance of normothermia.

The outcome of treatment depends on the root cause of a cardiac arrest, the duration of its impact, timeliness and quality of resuscitation and intensive care in a highly postresuscitation period. CPR technique is constantly improving, and perhaps in the near future will undergo significant changes.

Outcomes of resuscitation

After the restoration of spontaneous circulation, as already mentioned, the treatment begins postresuscitative disease. Outcomes are possible variety. System is the most vulnerable CNS. The following results are from best to worst.

1. Full recovery (possibly with an immediate start resuscitation with rapid effect. Faster easier coma better prognosis)

2. Encephalopathy of varying severity and varied nature.

3. Decortication or Apallic syndrome. New crust is most sensitive to hypoxia, so maybe the development of this state, when the crust was lost, and subcortical centers are functioning. It is a social death, "vegetative state." Patients show adequate spontaneous breathing and circulation if it is fed, he swallows. All physiological needs depart involuntarily retained only elementary reflex reaction to the subcortical and spinal level to external stimuli.

4. Brain death - the state, as a result of hypoxic episode killed the whole brain as a whole that is and bark and trunk. (These states are also found in the cranial injuries and other similar processes). Clinic:

- coma
- breathing is provided by mechanical ventilation (spontaneous breathing is absent).
- no stem reflexes (pupillary, corneal, oculocephalic, oculovestibular, pharyngeal and tracheal).
- Hemodynamics - may be relatively unstable.

Brain death is established by a commission of medical facility where the patient. Included in the commission certified physicians with experience in the specialty for at least 5 years of critical care medicine, a neurologist (neurosurgeon) and specialists for additional instrumental studies. For instrumental confirmation of brain death can be used: electroencephalography (EEG), cerebral angiography, nuclear magnetic resonance angiography, transcranial Doppler ultrasonography, or cerebral scintigraphy. The main document is the Protocol establishing brain death, which is set to stop resuscitation and suction bodies for transplantation in accordance with legislation. The Protocol establishing brain death should be given details of all studies, the names and patronymics medical committee members, their signature, date and hour of registration of brain death and, therefore, detection of biological death. Appointment of committee and approval of the Protocol establishing brain death is head of intensive care unit, where the patient, and in his absence - the duty doctor responsible institution. The commission may not include experts involved in the fence, and organ transplantation. Responsible for ascertaining biological death are doctors, the diagnosis of brain death, to hospital where the patient died.

Self-study

Task number one

Conducted a comprehensive CPR on a simulator.

- Conduct a determination of the need resuscitation
- Perform CPR
- Conduct ventilation methods "mouth to mouth" and the manual method using a mask and Ambu bag
- Identify tactics to use and dosage of essential drugs
- Conduct defibrillation.

Task number two

Assess the patient after successful CPR:

- Determine the period of the state of postresuscitative disease
- Identify tactics further monitoring
- Justify the treatment depending on the stage of state

Clinical problems

Objective number one

A patient, 86 years old (in history of CHD: stable angina FC3, CHF IIa, atrial fibrillation, tahiforma) mobile teams "emergency" set sudden cardiac arrest (the reason - ventricular fibrillation). CPR held for 10 minutes, its termination was motivated by age of the patient. The violation? Indications to stop CPR?

Objective number two

In the therapeutic department of the hospital was a receptionist resuscitation of the patient with suspected anaphylactic shock. The ward was a physician and a ward nurse performing resuscitation. The patient lies on his bed, his head on the pillow, pressing on the chest performed at the apex of the heart, the adrenaline injection is carried out in the muscle (the needle from the infusion system is removed from the vein). Note the error and enter the correct tactics CPR?

Objective number 3

A patient, 22 years old dived from a height of 3 meters, was pulled ashore unconscious. CPR started promptly after triple maneuver. Regained consciousness, the pulse of severe tachycardia, a rare independent breathing is shallow, there is no physical activity in the limbs. What a mistake was made at the stage of first aid? Further tactics department ICU?

Objective number 4

Child 8 years old, disappeared while swimming under water for 3 minutes, a lifeguard brought ashore unconscious. Following the CPR consciousness and vital functions are fully recovered. Was allowed to go home. After 10 hours taken to the emergency room with severe respiratory failure, signs of incipient pulmonary edema. Explain tactical error? What caused the rapid deterioration of the child?

Test control

1. Enter the correct order of the stages of dying

- a) preagony, terminal pause, the agony, the clinical and biological death.
- b) terminal pause preagony, the agony, the clinical and biological death
- c) the agon, preagony, terminal pause, clinical and biological death.
- d) agony, preagony, terminal pause, biological death, clinical death
- e) the agony preagony, clinical death, terminal pause and biological death

2. The principle of the "A" CPR means:

- a) cardiac massage
- b) determining the need resuscitation
- c) determination of the level of consciousness
- d) reduction and control of the airway
- e) ventilation

3. Pressure point when CPR is:

- a) in the apex of the heart
- b) in the upper third of the sternum
- c) at the base of the xiphoid process
- d) about the border of the lower and middle third of the sternum
- e) 2-3 cross fingers below the base of the xiphoid process

4. Force pressing on the chest of an adult shall be:

- a) 2-3 cm
- b) 8-10 cm

- c) 4-5 cm
- d) 1-2 cm
- e) 5-8 cm

5. Frequency compression when CPR is about:

- a) 20 per minute
- b) 30 per minute
- c) 40 per minute
- d) 80 per minute
- e) 110 per minute

6. The ratio of breaths / compressions by 2 rescuers in children:

- a) 2:10
- b) 2:15
- c) 1:5
- d) 1:1
- e) 1:20

7. The ratio of breaths / compressions by 1 rescuer in children:

- a) 2:10
- b) 2:15
- c) 2:30
- d) 1:1
- e) 1:20

8. Ventilation and chest compressions are performed independently in the event of:

- a) If the victim performed intubation
- b) the person is agonal phase
- c) the victim is pronounced scoliosis with chest deformity
- d) More than 30 minutes of cardiac arrest before resuscitation
- e) the victim is found to have chronic end-stage

9. Point of the electrodes at defibrillation:

- a) the two - at the top of the heart
- b) 1st - right parasternal line below the collarbone, 2nd - the apex of the heart
- c) 1st - on the left parasternal line below the collarbone, 2nd - the apex of the heart
- d) 1st - at the midline in the lower third of the sternum, the 2nd - the apex of the heart
- d) 1st - at the midline in the upper third of the sternum, the 2nd - the apex of the heart

10. When CPR SV is from the original:

- a) 80%
- b) 100%
- c) 50%
- d) 10%
- e) 30%

11. Regular alternation of periods postresuscitative states:

- a) the relative stabilization of functions, the period of instability features re deterioration period of improvement
- b) the period of instability features relative stabilization functions, re deterioration period of improvement
- c) a period of improvement, re deterioration relative stabilization functions instability period

features

- d) a period of improvement, the period of instability features re deterioration relative stabilization functions
- e) the period of instability features a period of improvement, re deterioration relative stabilization functions

12. Distinguish the following periods of the dying process:

- a) clinical death
- b) social death
- c) decortication
- d) decerebration
- e) natural death

13. The success of resuscitation due to the following factors:

- a) early diagnosis of clinical death
- b) a timely challenge resuscitator
- c) the timely start of resuscitation

Answers to clinical problems:

Number 1: Age is not a reason to stop CPR.

Number 2: The patient should be on a firm level surface, and if there are no contraindications, performed a triple maneuver, press right on the median line, intravenous (not distal ulnar vein), the needle should not be removed from a vein (changing the system itself).

Number 3. The probability of damage to the cervical spine, which should be considered when CPR and transportation.

Number 4. After successful CPR also required hospitalization. Deterioration caused by the development of respiratory distress syndrome.

Answers to test questions:

1) a 2) d 3) d 4) c 5) e 6) b 7) c 8) a 9) b 10) e 11) b 12) a, d, e 13) a, b, c