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V. N. Karazin Kharkiv National University

ORGANIZATION AND PROVISION OF EMERGENCY MEDICAL CARE IN THE PRACTICE OF A FAMILY DOCTOR. PROVIDING EMERGENCY CARE BY A FAMILY DOCTOR IN CASE OF SUDDEN DEATH, CONVULSIONS AND LOSS OF CONSCIOUSNESS IN THE PREHOSPITAL STAGE.

Educational and methodological guide to practical classes
and independent work of students of the 6th year for the discipline
"General practice - family medicine"

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Organization and provision of emergency medical care in the practice of a family doctor. Providing
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The educational and methodological guide contains general information and algorithms for providing emergency care in the practice of a family doctor. The educational and methodological guide to practical classes and independent work of students of the 6th year was compiled in accordance with the working program of the academic discipline "General practice - family medicine", specialty - 222 "Medicine". The purpose of the guide is to facilitate the assimilation of theoretical knowledge by students of the 6th year of the medical faculty during preparation for practical classes.

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Навчальне видання

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ОРГАНІЗАЦІЯ ТА НАДАННЯ ЕКСТРЕНОЇ МЕДИЧНОЇ ДОПОМОГИ В ПРАКТИКІ СІМЕЙНОГО ЛІКАРЯ. НАДАННЯ ЕКСТРИНТЕННОЇ ДОПОМОГИ СІМЕЙНИМ ЛІКАРЕМ ПРИ РАПТОВІЙ СМЕРТІ, СУДОМАХ ТА ВТРАТИ СВІДОМІСТІ НА ДОДОГОСПІТАЛЬНОМУ ЕТАПІ.

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CONTENT

INTRODUCTION

A general practitioner should have the skills to provide emergency care for various life-threatening conditions. A timely diagnosis and the ability to professionally provide emergency care at the pre-hospital stage helps to avoid severe complications, the development of irreversible changes in the body, the death of the patient and improves further treatment results.

The educational and methodical guide contains general information and algorithms for providing emergency care in the practice of a primary care physician in the provision of medical care in case of sudden death, convulsions and loss of consciousness in the prehospital stage.

The need to create such guide is due to the insufficient amount of educational and methodological materials that would fully correspond to these sections of the program in the educational discipline "General practice - family medicine".

The educational and methodological guide can be recommended for students of higher medical educational institutions of the IV level of accreditation. The guide contains useful information for interns, general practitioners - family medicine doctors and doctors of any specialty.

1. SECTION 1

CLASSIFICATION, ETIOLOGY AND PATHOGENESIS OF SUDDEN CARDIAC DEATH

In the recommendations of the European Society of Cardiology, sudden cardiac death is defined as "natural death due to cardiac causes, which is preceded by sudden fainting within 1 hour after the onset of acute symptoms; a previously diagnosed heart disease is possible, but the time and manner of death are unexpected."

In the classification of cardiovascular diseases of the Ukrainian Scientific Society of Cardiology, which was adopted at the VI National Congress of Cardiologists of Ukraine, the definition of sudden cardiac death was harmonized with the International Classification of Diseases 10 revision:

146.1 - sudden cardiac death (arrhythmic) (death that occurred within 1 hour after the appearance of the first symptoms of the disease or significant deterioration of the patient's condition against the background of a stable chronic course of the disease):

1. With the restoration of cardiac activity:
   - ventricular fibrillation;
   - asystole;
   - electromechanical dissociation (indicated if possible).
2. Cardiac arrest (death that occurred later than 1 hour after the onset or worsening of symptoms of the disease).
3. Sudden cardiac death (irreversible):
   - ventricular fibrillation;
   - asystole;
   - electromechanical dissociation (indicated if possible).

145.0 - with recovery of cardiac activity.

146.9 – cardiac arrest (irreversible).

An example of a clinical diagnosis: arrhythmogenic dysplasia of the right ventricle, persistent monomorphic ventricular tachycardia, sudden cardiac death (arrhythmic), irreversible (ventricular fibrillation, asystole, 06.12.2002).

Etiology. The clinical manifestation of sudden cardiac death often depends on a certain mechanism. According to the results of one of the studies, among the total number of 157 outpatients who suffered sudden cardiac death during Holter monitoring of the electrocardiogram, deaths were due to: ventricular fibrillation (62.4%), bradyarrhythmia (16.5%), "pirouette -tachycardia" (12.7%), primary ventricular tachycardia (8.3%). A change in the ST segment was observed in 12.6%. The more precisely the mechanism is established, the better preventive measures can be used. Although it has been proven that the main cause of sudden death after myocardial infarction is tachyarrhythmia, there are other mechanisms, such as aortic rupture, subarachnoid aneurysm rupture, heart rupture and tamponade, massive pulmonary embolism, and others. On the other hand, death may be arrhythmic in nature but not

sudden, such as a patient who dies in hospital from hemodynamic collapse and complications from persistent ventricular tachycardia.

The most common cause of sudden cardiac death is acute coronary syndrome; in 25% of patients with ischemic heart disease who died suddenly, sudden cardiac death was the first and only manifestation of the disease. Sudden cardiac death also accounts for 40–50% of deaths in patients with heart failure. In high-risk myocardial infarction patients (data from the EMIAT, CAMIAT, TRACE, SWORD, DINAMIT trials), the cumulative arrhythmic mortality was approximately 5% at 1 year and 9% at 2 years, while the rate of nonarrhythmic cardiac death was respectively 4 and 7%. Sudden cardiac death is caused by hypertrophy of the left ventricle.

At the same time, in almost 12% of cases, the cause of sudden cardiac death remains unclear, given the fact that at autopsy or after a comprehensive medical examination of patients who have experienced a cardiac arrest, no signs of heart disease are found. The percentage of patients who die suddenly without apparent heart disease is highest at a young age. From time to time there are reports of cases of sudden cardiac death among famous athletes - persons, it would seem, with an exemplary state of health. From an epidemiological point of view, sudden cardiac death at a young age, especially in individuals without clinical signs of heart disease, is of limited importance, since it accounts for only a small proportion of sudden cardiac death cases that occur in the general population. However, victims of sudden cardiac death are practically healthy people, whose premature death has tragic consequences for families and society.

Diseases and conditions in which sudden cardiac death most often develops:

- Acute coronary syndrome;
- Post-infarction cardiosclerosis;
- Heart failure;
- Hypertrophic cardiomyopathy;
- Dilated cardiomyopathy;
- Myocarditis;
- Aortic stenosis;
- Mitral valve prolapse;
- Violation of impulse conduction;
- Wolff-Parkinson-White syndrome;
- Syndrome of prolonged QT interval;
- Brugada syndrome;
- Arrhythmogenic right ventricular dysplasia;
- Abnormal development of coronary arteries;
- Myocardial bridges;
- "Sports heart".

Pathogenesis of sudden cardiac death. Arrhythmias, heart blocks and the development of electrical instability of the myocardium are believed to be the main cause of sudden cardiac death.

Trigger factors of sudden cardiac death include:

- acute myocardial ischemia caused by coronary spasm, thrombosis, including reperfusion, increased myocardial oxygen demand;
- imbalance of the vegetative status with a predominance of sympathetic;
- disorders of myocardial metabolism (hypokalemia, hypomagnesemia);
- toxic factors, including the use of sympathomimetics, bronchodilators, antiarrhythmic drugs that increase the excitability of the myocardium or prolong the QT interval (about arrhythmic reactions); blockade of sodium channels by novocainomid and etacizin increases the degree of hypopolarization of cardiomyocyte membranes, as a result of which hypopolarizing heart rhythm disturbances (extrasystoles, heart blocks, short episodes of flutter and ventricular fibrillation) occur when minimal arrhythmogenic doses of potassium chloride are administered.

The cause of sudden cardiac death in diseases of the heart and blood vessels is ventricular fibrillation (85%), asystole (10%) and electromechanical dissociation (5%), and the percentage of patients who survived and suffered at least one episode of fatal arrhythmia is insignificant and is 19% total amount

General principles of providing emergency care in case of sudden stoppage of blood circulation. Cessation of blood circulation occurs more often at home (2/3 of cases), in men over 50 years old (3/4 of cases), during the day (8–

18 hours), with witnesses (2/3 of cases).

The American Heart Association (ANA) proposed an algorithm for the organization of first aid, called the "chain of survival":

- the first link ("early attack") - includes recognizing the given situation, calling and delivering experienced personnel to the patient;
- the second ("early cardiopulmonary resuscitation") is usually sufficient to support the patient until the arrival of an experienced team. It should be noted that according to the literature, any cardiopulmonary resuscitation, even only chest compression, is much better than no cardiopulmonary resuscitation;
- the third - early defibrillation using automated external defibrillators (Automated external defibrillators - AED);
- the fourth is the early beginning of the stage of further life support, including intubation and the use of drugs.

2. CARDIOPULMONARY RESUSCITATION

One of the main factors affecting the survival rate is the length of the time interval from the moment of circulatory arrest to the start of cardiopulmonary resuscitation.

Therefore, after establishing the signs of clinical death (absence of pulsation on the carotid arteries, apnea, dilated pupils that do not respond to light), it is necessary to immediately start cardiopulmonary resuscitation according to the algorithm proposed by P. Safar. Safar divided the entire complex of cardiopulmonary resuscitation into 3 stages, each of which has its own purpose and successive stages. According to the new recommendations of the ERS (2010), the cardiopulmonary resuscitation (A-B- C) algorithm was modified to C-A-B, so the first stage is the immediate start of chest compression and only then restoration of airway patency and artificial respiration:

I stage. Basic life support. The goal is emergency oxygenation.

Stages:

C. Artificial support of blood circulation.

A. Restoration of airway patency.

B. Artificial respiration.

II stage. Further life support. The goal is to restore spontaneous blood circulation.

Stages:

D. Drug therapy.

E. Electrocardiography or electrocardioscopy.

F. Defibrillation.

III stage. Long life support. The goal is cerebral resuscitation and post-resuscitation intensive therapy of multiple organ dysfunction.

Stages:

G. Assessment of the condition (establishment of the cause of circulatory arrest and its elimination) and the possibility of a full rescue of the patient, taking into account the degree of damage to the central nervous system.

H. Restoration of normal thinking.

I. Intensive therapy aimed at correcting impaired functions of other organs and systems.

J. Stage of basic life support.

C. Artificial blood circulation support. The first (pre-hospital) stage of resuscitation should be started directly at the scene without delay by any person familiar with the elements of cardiopulmonary resuscitation. Its purpose is to maintain artificial blood circulation and artificial ventilation of the lungs (artificial ventilation of the lungs) with the help of elementary methods that ensure the extension of the period of reversal of changes in vital organs until the moment when adequate independent

blood circulation is restored. An indication for cardiopulmonary resuscitation is the presence of even two main signs of clinical death. It is inadmissible to start resuscitation measures without checking the pulse on the carotid artery, because indirect massage of the heart during its normal operation can cause the cessation of blood circulation.

Contraindications to cardiopulmonary resuscitation are as follows:

- biological death;
- social death;
- terminal stages of incurable diseases;
- inoperable malignant neoplasms with metastasis;
- if it is known for certain that more than 25 minutes have passed since the cessation of blood circulation in conditions of normothermia.

The fundamental problem of artificial maintenance of blood circulation is a very low level (less than 30% of the norm) of cardiac output (CH), which is formed during chest compression, as a result of which blood is expelled not only from the heart, but also from the large vessels and vessels of the lungs. After stopping the pressure on the sternum, it returns to its original position due to the elasticity of the rib cage of the chest. At the same time, the compressed cavities of the heart and blood vessels straighten out and are filled with blood again. To ensure sufficient blood flow in the brain, it is necessary to perform at least 100 compressions in 1 minute (according to ANA recommendations, 2015).

Correctly performed compression ensures maintenance of systolic blood pressure at the level of 60-80 mm Hg, while diastolic blood pressure rarely exceeds 40 mm Hg and, as a result, causes a low level of cerebral (30-60% of normal) and coronary (5- 20% of normal) blood flow. Therefore, there are significant changes in the attitude of chest compression. Thus, when it is performed, the coronary perfusion pressure increases only gradually, therefore, with each successive pause necessary for "mouth-to-mouth" breathing, it rapidly decreases. However, carrying out several additional compressions leads to the restoration of the initial level of cerebral and coronary perfusion.

The position of the resuscitator's hands significantly affects the effectiveness of cardiac massage. The place of attachment of the hands is the lower part two fingers above the sterno-xiphoid joint (see Fig. 1.1):

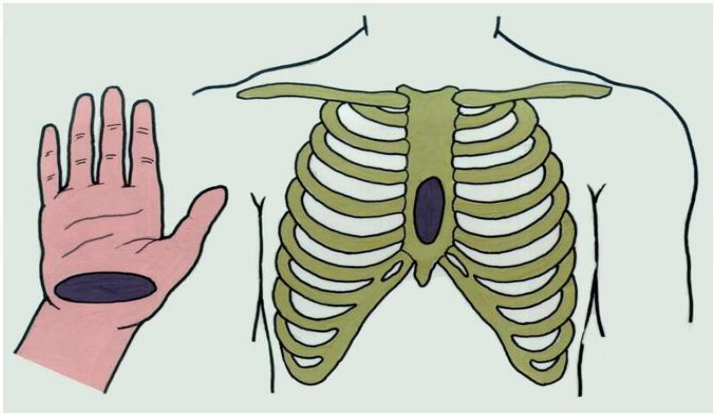

Fig. 1.1. Place of support of the palm and the area of application of force in the case of indirect heart massage

In the classic version of performing indirect heart massage, the resuscitator puts each of his hands on it so that the base of the palm is located above the compression site, and the fingers are parallel to the ribs. The second hand is placed on the back of the first so that its base also projects to the

place of compression. Fingers should not lie on the victim's chest, because this shifts the point of compression and increases the risk of fracture of the ribs and costal cartilages.

In addition to the classic arrangement of hands, the method of crossed fingers is also used, in which the fingers of the upper hand pass between the fingers of the lower hand and cover the palm. Pressing in this version is carried out with the entire surface of the palm. At the same time, along with the increase in the compression surface, the pressure per unit area decreases. Today, there are no clear recommendations for choosing one or another method of indirect heart massage.

One of the main conditions for the correct performance of indirect heart massage is pressure not due to the hands, but due to the weight of the resuscitator's body. To do this, you need not to bend your arms at the elbow joints. According to the American Heart Association (2015) recommendations, during compression, the sternum should bend at least 5 cm in adults and at least 1/3 of the chest diameter in children (about 5 cm) and infants (about 4 cm).

In the recommendations of the American Heart Association (2015), it is proposed to replace the sequence of basic life support measures A-B-C (ensuring the patency of the respiratory tract, artificial lung ventilation and oxygenation, artificial blood circulation support) with C-A-B for adults, children and infants (except for newborns).

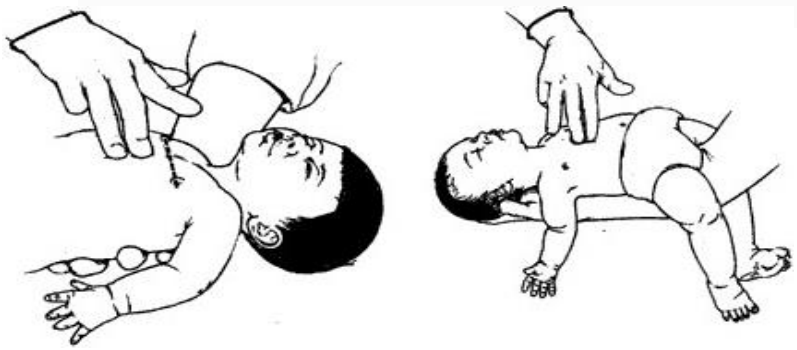

Fig. 1.2. Resuscitation of an infant

Increase the filling of the heart cavities with blood by raising the foot end of the bed or by simply placing a roller under your feet. Against the background of the absence of vascular tone, this provides artificial centralization of blood circulation.

Direct heart massage remains a later alternative. Despite the fact that direct cardiac massage provides a higher level of coronary and cerebral perfusion pressure (50% and 63-94% of normal, respectively) than chest compression, there are no data on its ability to improve the outcome of cardiopulmonary resuscitation, in addition, its use is associated with more frequent complications. Nevertheless, there are a number of direct indications for its implementation:

1. Presence of an open chest in operating room conditions;
2. Suspicion of intrathoracic bleeding;
3. Suspicion of a violation of abdominal blood circulation due to compression of the descending part of the thoracic aorta;
4. Massive pulmonary embolism;
5. Cessation of blood circulation against the background of hypothermia (allows direct warming of the heart);
6. Failure of chest compression to generate a pulse on the carotid and femoral arteries due to deformation of the bones of the chest or spine;
7. Suspicion of a long period of unnoticed clinical death;

8. Inability of properly performed chest compression in combination with other measures of the stage of further life support to restore spontaneous normotension.

In order to facilitate long-term cardiopulmonary resuscitation, mechanical chest compression devices such as "AutoPulse" and "Life-State" (Michigan Instruments) and others are widely used abroad.

Errors during cardiac massage are tactical and procedural in nature. Errors of the first group include: late start of indirect massage or continuation of its performance in cases where direct heart massage was recommended. Procedural errors include violations of the sequence and techniques of cardiopulmonary resuscitation, incorrect selection of the mode of artificial lung ventilation and the place of heart compression.

Complications of artificial maintenance of blood circulation are mostly associated with fractures of the ribs and sternum with subsequent damage to the lungs by fragments of the ribs, pneumo- and hemothorax, damage to the liver and spleen, hematoma of the mediastinum, tamponade of the heart. Complications of direct heart massage include bleeding from dissected vessels, crossing of nerves, lung damage. Along with the signs of the correctness and adequacy of conducting each of the stages of the first stage of cardiopulmonary resuscitation, it is also necessary to check the signs of the effectiveness of resuscitation measures as a whole. These include a change in the color of the skin from pale and cyanotic to pale pink, narrowing of the pupils, and sometimes the appearance of independent breathing. These signs indicate the restoration of metabolism in the tissues, at least at the minimum acceptable level.

In parallel with the assessment of signs of the effectiveness of resuscitation measures, possible reasons for their absence are analyzed in the presence of signs of correctly performed stages of the first stage of cardiopulmonary resuscitation . Preservation of the paleness of the skin can indicate massive internal bleeding, preservation of cyanosis (especially in the upper part of the trunk, neck and head) - about thromboembolism of the pulmonary artery, wide pupils - about traumatic or non-traumatic damage to the deep parts of the brain, damage to the oculomotor centers, the effect of drugs that expand the pupil

The absence of signs of the effectiveness of resuscitation measures when using all available methods within 30 minutes is an indication to stop resuscitation.

Resuscitation measures of the first stage of cardiopulmonary resuscitation allow maintaining gas exchange in tissues at a minimum level. They are carried out before the restoration of independent blood circulation, however, cardiopulmonary resuscitation is not enough for the full recovery of the vital activity of the body using methods of the first stage. They are supplemented by measures of the II stage of resuscitation, which are performed by the resuscitation team, which has the experience of conducting them and the appropriate equipment.

A. Restoration of airway patency. The "gold standard" of ensuring patency of the respiratory tract remains the triple reception of Safar (head tilt, mouth opening, lower jaw extension) and tracheal intubation (Fig. 1.3).

As an alternative to endotracheal intubation, it is recommended to use a laryngeal mask or a double-lumen Combitube as technically simpler, compared to intubation, but at the same time reliable methods of airway protection.

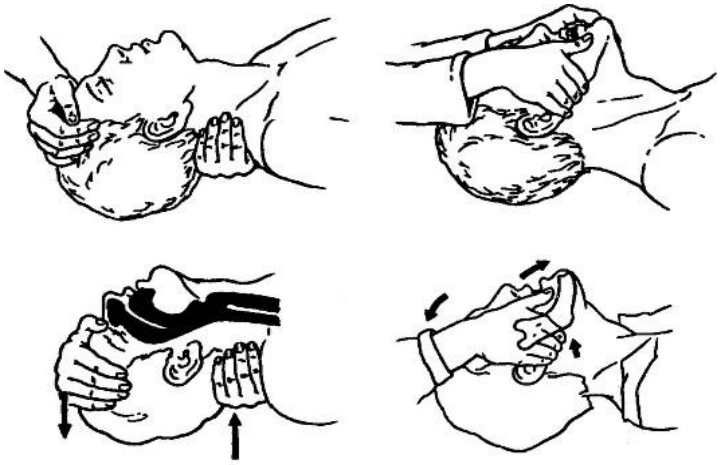

Fig. 1.3. Restoration of airway patency

In victims with a suspicion of a fracture of the spine in the cervical region or a fracture of the base of the skull, manipulations of the head are carried out very carefully.

It is undesirable to turn the head to the side and throw it sharply. A moderate extension of the cervical spine is sufficient.

Airway patency can also be maintained with the use of airways, a laryngeal mask, and other devices. Due to the fact that irritation of the root of the tongue and the entrance to the trachea can cause vomiting and laryngospasm, the introduction of airways and tracheal intubation is performed only in victims who are in a coma or under the influence of sedative drugs.

In case of impossibility of performing the manipulations listed above, there is a need for surgical methods of restoring airway patency - cricotomy, microtracheostomy, tracheostomy. When the patency of the respiratory tract is disturbed due to the ingress of foreign bodies, the tactics depend on the level at which it stopped and on its consistency. If a foreign body is placed in the oral cavity, you can try to remove it with your fingers. Mucus, blood clots, vomitus and other liquid pathological contents from the oral cavity can be removed by wrapping the fingers with a hygroscopic tissue, using a gauze swab clamped in forceps, surgical suction or a rubber bulb. Solid foreign bodies from the oral part of the pharynx can be removed with the help of two fingers, tweezers or forceps. In the groove where a foreign body or liquid enters the vocal cords, it is necessary to create a drainage position in which the exit from the trachea will be lowered lower than the parts of the lungs where the pathological content is located. If this is not enough, it is fashionable to promote the foreign body with the help of vibration massage. To do this, with a hand clenched into a fist, blows of moderate force are applied to the other hand, located on the victim's chest. At the same time, blows begin to be applied over the distal parts of the lungs, gradually moving them towards the trachea.

B. Artificial respiration. Having ensured the possibility of air entering the lungs, artificial ventilation of the lungs is started. In cases of providing emergency care outside the hospital or in the absence of the necessary equipment, artificial respiration using mouth-to-mouth and mouth-to-nose methods is used. Newborns and infants use the mouth-to-mouth and nose-to-mouth method. Factors such as the patency of the nasal passages, the integrity of the maxillofacial skeleton and the soft tissues of the oral part of the mouth and nose affect the choice of one or another method of artificial lung ventilation.

When performing artificial ventilation of the lungs (artificial ventilation of the lungs) by the "mouth-to-mouth" method, each artificial breath should be carried out for 2 seconds (not forced) while simultaneously observing the excursion of the chest in order

to achieve the optimal respiratory volume and prevent air from entering the stomach (Fig. 1.4).

At the same time, the resuscitator must take a deep breath before each artificial breath to optimize the concentration of O2 in the exhaled air, since the latter contains only 16-17% O2 and 3.5-4% CO2. Respiratory volume should be 500-600 ml (6-7 ml/kg), respiratory rate

- 10/min to prevent the development of hyperventilation, which causes an increase in intrathoracic pressure, which causes a decrease in venous return to the heart and reduces cardiac output, which is associated with a poor survival rate of patients.

Fig. 1.4. artificial lung ventilation by the mouth-to-mouth method

At the same time, the resuscitator must take a deep breath before each artificial breath to optimize the concentration of O2 in the exhaled air, since the latter contains only 16-17% O2 and 3.5-4% CO2. The respiratory volume should be 500-600 ml (6-7 ml/kg), the respiratory rate - 10/min to prevent the development of hyperventilation, which causes an increase in intrathoracic pressure, which causes a decrease in venous return to the heart and reduces cardiac output, associated with a poor level survival rates of patients.

In the case of mouth-to-mouth artificial lung ventilation, airway patency is maintained by tilting the head and raising the lower jaw. As in the previous method, it s desirable to have a spacer separating the victim from the resuscitator. The resuscitator takes a breath, then covers the victim's nose with his lips with a force sufficient to create a seal, but not allowing the nasal passages to overlap, and takes a breath. The resuscitator then moves away from the victim, allowing air to passively escape from the lungs.

The mouth-to-mouth and nose-to-nose method is used in newborns and infants, because due to the small size of the mouth and nose, only this ratio allows to achieve sufficient tightness.

Control of the correctness of artificial lung ventilation is carried out based on the excursion of the chest and the presence of elastic resistance to exhalation. At the same time, they pay attention to the raising of the sternum during inhalation and its lowering during exhalation. Absence of lowering on exhalation can be observed in the case of air entering the stomach, which is accompanied by a characteristic sound that resembles muffled gurgling. In this case, the distended stomach, raising the edge of the costal arch, can create a false impression of an excursion of the chest. The danger of this complication lies not only in the fact that air does not enter the lungs, but also in the risk of regurgitation due to increased pressure in the stomach, which is accompanied by the development of aspiration pneumonia in the post-resuscitation period.

It is possible to prevent air from entering the stomach and regurgitation by taking Selik: pressing on the cricoid cartilage, it is pressed against the spine, which

leads to compression of the esophagus, thereby preventing the flow of stomach contents into the oral part of the pharynx.

Recently, methods of passive inflow of air into the lungs are used only in those cases when, due to a severe facial injury, ventilation by mouth-to-mouth or mouth-to-nose methods is impossible and at the same time it is not possible to perform tracheal incubation or tracheo- or conictomy.

Disadvantages of device-free methods of artificial lung ventilation are: the need for resuscitator contact with the victim (a gauze napkin or handkerchief does not protect against infection!); the impossibility of long-term artificial ventilation of the lungs by one resuscitator, which is especially important in the case of delaying the help of a specially equipped resuscitation team. Some of these disadvantages can be eliminated with the help of such devices as the "key of life" and Laerdal's mask. With its help, artificial ventilation of the lungs is performed by the mouth-mask method with additional oxygen supply through the nipple valve present in it. Disadvantages of device-free methods of artificial ventilation are eliminated by conducting artificial ventilation of the lungs with the help of manual devices. The most typical among them is the Ambu bag, capable of self-expanding after compression. It can be used with a face mask and other devices for maintaining the patency of the respiratory tract, which have standard disconnections for attaching respirators (laryngeal mask, endotracheal tube, esophageal obturator, etc.).

The use of hand-held devices prevents the adverse effect of artificial lung ventilation on the resuscitator, ensures that air with a normal amount of oxygen (and with an increased supply) enters the victim's lungs.

Another type of manual respirators are devices with a trigger ventilation system, which consist of a bleed valve on the oxygen or air mixture supply hose. In services that provide resuscitation care in out-of-hospital conditions (in areas of natural disasters, man-made disasters, military operations), trigger-type respirators are used, in which the inhalation and exhalation phases are switched automatically after reaching a certain pressure in the respiratory tract.

Oxygenation during resuscitation is one of the conditions for adequate oxygen supply to the brain, so it should be started as soon as possible. If possible, 100% oxygen is initially used, reducing its concentration in the future under the control of pO2.

Complications and errors during artificial lung ventilation: free passage of the respiratory tract is not ensured; air tightness is not ensured when blowing air; underestimation (later onset) or overestimation (beginning of cardiopulmonary resuscitation with intubation) of the value of artificial lung ventilation; lack of control over excursions of the chest; lack of control over air entering the stomach; attempts at medical stimulation of breathing.

The following is a brief overview of the elements of quality cardiopulmonary resuscitation for persons performing basic life support measures (according to 2015 ANA recommendations):

Table 1.1

A brief overview of the elements of quality cardiopulmonary resuscitation

Element	Adults and teenagers	Children (aged 1 year to puberty)	Babies (under the age of 1 year, excluding newborns)
Safety of place events	Ensure that the environment is safe for resuscitators and victim		
Recognition cardiac arrest	Check for consciousness. Not breathing or suffocating (i.e. breathing abnormally). Not breathing or suffocating. The pulse is not clearly determined for 10 seconds (Assessment of breathing and pulse can be carried out simultaneously for at least 10 seconds).		
Correlation "compression-inhalation" for absence incubation tubes	1 or 2 resuscitator 30:2	1 resuscitator 30:2 2 resuscitators or more 15:2	
Correlation "compression-inhalation" for availability incubation tubes	Continue chest compressions with a frequency of 100-120/min. Take 1 breath every 6 seconds (10 breaths/min.)		
Frequency compression squeeze	100-120/min.		
Depth depressions chest	At least 5 cm	At least 1/3 anteroposterior ore diameter cells Approximately 5 cm	At least 1/3 anteroposterior ore diameter cells Approximately 4 cm
Laying hands	2 hands on the bottom half of the sternum	2 hands or 1 hand (for very small children) on the lower half of the sternum	chest just below the nipple line 2 resuscitators or more: hands covering body, 2 thumbs in the center of the chest, just below the nipple line
Rectification chest	Wait until the chest is fully expanded after each compression, do not lean on the chest after each compression		
Reduction to minimum intervals	Intervals between chest compressions should not exceed 10 seconds		

3. UNIFIED CLINICAL PROTOCOL OF EMERGENCY MEDICAL AID "SUDDEN CARDIAC DEATH"

3.1. Assessment of the patient's condition - according to the avsde algorithm

1. A - airway patency (Airway)

1.1. Determine the symptoms of airway obstruction: violation of airway patency contributes to the emergence of paradoxical breathing and the participation of additional respiratory muscles in breathing; central cyanosis is a late symptom of airway obstruction; in patients who are in a critical condition, a disturbance of consciousness often causes a violation of the patency of the respiratory tract (sinking of the tongue, soft palate).

1.2. Oxygen in high concentration: using a mask with a tank; make sure the oxygen supply is adequate (> 10 L/min).

2. B - breathing (Breathing)

During the assessment of breathing, it is important to identify and treat conditions that are an immediate threat to life - a severe asthma attack, pulmonary edema, tension pneumothorax, hemothorax.

2.1. Identify the symptoms that may indicate breathing disorders: excessive sweating, central cyanosis, work of additional muscles or abdominal type of breathing.

2.2. Determine the frequency of breathing - normally it is 12-20 breaths per minute.

2.3. Assess the way of breathing, the depth of breaths and check whether the movements of the chest are symmetrical.

2.4. Pay attention to the excessive filling of the neck veins (for example, with severe asthma or tense pneumothorax), the presence and patency of pleural drainage, etc.

2.5. Carry out auscultation and percussion of the lungs.

2.6. Determine the position of the trachea - its displacement may indicate a tense pneumothorax, lung fibrosis or fluid in the pleural cavity.

3. C - blood circulation (Circulation)

3.1. Assess the color of the skin on the exposed parts (hands): blue, pink, pale or marbled.

3.2. Assess the temperature of the extremities: cold or warm.

3.3. Assess capillary filling - normally up to 2 seconds. Increased capillary refill may indicate reduced peripheral perfusion.

3.4. Assess the filling of the veins - they can be moderately filled or inflamed with hypovolemia.

3.5. Determine the heart rate. Find the peripheral pulse and pulse on a large artery, assess its presence, frequency, quality, regularity and symmetry.

3.6. Measure blood pressure.

3.7. Listen to the tones of the heart.

3.8. Pay attention to other symptoms that would indicate a decrease in cardiac output, such as loss of consciousness, oliguria (urine volume < 0.5 ml/kg/h).

4. D - impaired state of consciousness (Disability)

Most often, the causes of disturbances in the state of consciousness are severe hypoxia, hypercapnia, brain ischemia or the use of drugs with a sedative effect or analgesics;

4.1. Assess the pupils (diameter, symmetry and reaction to light).

4.2. Quickly assess the patient's state of consciousness on the AVPU scale: Alert (orienting), Vocal (responds to voice), Pain (responds to pain), Unresponsive (does not respond to any stimuli). You can also use the Glasgow ComaScale.

4.3. Determine the level of glucose to rule out hypoglycemia. If the glucose level is less than 3 mmol/L, provide IV 50.0 mL of 20% glucose solution.

5. E - additional information (Exposure)

5.1. Collect a detailed anamnesis from the patient, his relatives, friends.

5.2. Familiarize yourself with the patient's medical documentation: check the indicators of vital parameters and their changes in dynamics, check which medicines the patient is prescribed and which he takes.

4. PROTOCOL: "ORGANIZATION AND PROVISION OF MEDICAL AID AT THE PRE-HOSPITAL STAGE"

4.1. Diagnosis: sudden cardiac death Code icd-10: i 146.0, i 146.1, i 146.9, r 96.0, r 96.1, r 98, r 99

Justification and main provisions of the protocol

1. The pre-hospital stage includes the provision of pre-medical, according to the recommendations of the dispatcher, and emergency medical care to patients with sudden cardiac death from the moment the patient is identified or when relatives or witnesses seek medical help until the moment of hospitalization.

2. Provision of emergency medical care at the pre-hospital stage is carried out: by emergency (emergency) medical aid teams of Centers of emergency medical aid and disaster medicine, emergency (emergency) medical aid stations, doctors of emergency (urgent) medical aid departments of multidisciplinary hospitals that are part of the emergency medical aid system.

3. Medical care at the pre-hospital stage should be provided to patients with sudden cardiac death in the first minutes of the onset of symptoms.

4. Patients with sudden cardiac death must be urgently hospitalized, first of all, in centers (departments) where highly qualified treatment in the intensive care unit (after resuscitation) and the possibility of primary coronary intervention are possible.

5. Rapid diagnosis of signs of sudden cardiac death at the pre-hospital stage reduces the time of initiation of resuscitation measures and increases the probability of spontaneous restoration of blood circulation.

6. To ensure the consistency of providing medical care to patients with a diagnosis of sudden cardiac death in each hospital, it is advisable to develop and implement local medical care protocols that define the patient's clinical route and the scope of medical and diagnostic measures in accordance with material, technical and personnel support. Interaction between health centers that provide emergency, primary and secondary medical care is determined by the order of the territorial body on health care.

7. local medical care protocols should be brought to everyone who is involved in providing medical care to patients with sudden cardiac death at the pre-hospital stage.

4.2. For healthcare facilities providing emergency medical aid

4.2.1. For the dispatcher of the operational dispatch service of the center for emergency medical aid and disaster medicine

Provisions of the protocol

1. Reception of a call by the dispatcher of the operational dispatch service of the center of emergency medical aid and disaster medicine by the single telephone

number of the emergency medical aid call 103 or by the single telephone number of the emergency medical aid call 112.

2. The dispatcher of the operational-dispatch service of the center of emergency medical aid and disaster medicine must accept the call in accordance with the approved algorithm and send the emergency (urgent) medical aid team to the patient suspected of sudden cardiac death.

Necessary actions (see appendix #6 and #8)

Mandatory:

1. Advice to the subscriber who called the operational dispatch service of the center of emergency medical assistance and disaster medicine:

- make sure of your own safety and assess the situation;
- check the patient's consciousness by shouting and lightly shaking the shoulders;
- call for help from others;
- place the patient on a firm, flat horizontal surface;
- open the airways and check for breathing and pulse;
- in their absence, or uncertainty about their presence - immediately start cardiopulmonary resuscitation;
- if there is an automatic external defibrillator nearby, immediately bring it or entrust it to others, perform indirect heart massage and artificial respiration in parallel (or only heart massage if artificial respiration is impossible). The ratio of chest compressions to artificial breaths is 30:2, starting with chest compressions;
- change the one who performs chest compressions every 2 minutes, if possible;
- continue cardiopulmonary resuscitation until the patient breathes, moves, and opens his eyes; before the arrival of the EMD brigade; before the onset of physical exhaustion of the resuscitator;
- in case of resuscitation of the patient - transfer him to a stable position on his side and wait for the arrival of the EMD team, while constantly monitoring the presence of breathing (pulse check is mandatory only for medical personnel);
- do not leave the patient unattended.

2. After registering the call, the dispatcher urgently sends the EMD team in a class C car (in its absence, a car of a lower class) to the scene, with an indication of the high probability of sudden cardiac death.

4.3. For the emergency (first) medical aid brigade

Provisions of the protocol

4.3.1. The standard for the arrival of an emergency (ambulance) medical aid team at the scene of the incident is 10 minutes in cities, and 20 minutes in settlements outside the city limits from the moment of receipt of the request to the dispatcher of the operational dispatch service of the center of emergency medical aid and disaster medicine.

Taking into account meteorological conditions, seasonal features, the epidemiological situation and the condition of the roads, the specified standards may be exceeded, but not by more than 10 minutes.

4.3.2. The diagnostic and clinical examination of the patient is recorded in the emergency medical aid departure card (form 110/o). It is necessary to attach an ECG to the ambulance departure card (form 110/o), and in the case of transmission of biometric ECG signals to the telemetry consultation center, a cardiologist's conclusion should be recorded.

Justification

Early diagnosis and hospitalization of patients with sudden cardiac death in specialized hospitals for the purpose of high-quality post-resuscitation treatment and primary coronary intervention reduces mortality and disability due to this disease, improves the results of patient treatment.

Necessary actions of the head of the emergency (urgent) medical care team

Mandatory:

1. History collection (should be carried out during cardiopulmonary resuscitation)

1.1. Collection of medical history:

1.1.1. Establish the exact time of deterioration of the patient's general condition.

1.1.2. Establish the most important reasons for the deterioration of the general condition.

1.1.3. Determine whether pre-medical assistance was provided in case of sudden cardiac death.

1.1.4. Set the time of onset of sudden cardiac death.

1.2. Collection of life anamnesis:

1.2.1. To establish what diseases the patient had, especially related to the cardiovascular system and the presence of the most possible diseases that could cause it.

1.2.2. Find out what medicines the patient took before the arrival of the emergency (ambulance) medical team.

1.2.3. Identify other concomitant diseases in the anamnesis: heart rhythm disorders, cerebral blood circulation disorders, oncological diseases and others.

1.2.4. Collect a general allergy history and find out if there are allergic reactions to taking medicines.

2. Examination and physical examination

2.1. Detection of signs of circulatory arrest should be carried out according to the SAV system.

2.2. Assessment of the general condition and vital functions: consciousness, breathing, blood circulation according to the ABCDE algorithm (Appendix No. 1) is carried out after restoration of spontaneous blood circulation.

3. Instrumental examination

Mandatory:

1. It is necessary to register an ECG in 3 leads or assess the rhythm directly from the defibrillator spoons.

2. Pulse oximetry (determination of blood oxygen saturation, the norm is 95% and above).

Wanted:

1. Capnography.

2. 12-channel ECG.

4.3.3. Treatment tactics

1. Necessary actions of the emergency (fast) medical care team at the pre-hospital stage (appendix No. 7):

Mandatory:

1. Before providing emergency medical aid, it is necessary to make sure that there are no threats to the team and the patient.

2. Quickly diagnose sudden cardiac death (no more than 10 seconds to check for signs of life).

If the patient has signs of life or they appeared during the resuscitation process, perform an examination of the ABCDE, give oxygen, connect a monitor, establish IV or IV access.

3. In the absence of vital signs - immediately start cardiopulmonary resuscitation (cardiopulmonary resuscitation): chest compressions and artificial respiration in a ratio of 30:2, starting with compressions, parallelly apply electrocardiograph electrodes or defibrillator spoons.

If the defibrillator is automatic, perform chest compressions while applying the electrodes.

If the defibrillator is a spoon and ventricular fibrillation without a pulse is registered, perform chest compressions while the defibrillator charges. The team leader orders not to touch the patient and conducts defibrillation.

In the presence of confirmed asystole, defibrillation is not performed, the main elements of cardiopulmonary resuscitation are continued.

4. If possible, determine and treat the cause of sudden cardiac death (appendix #9).

5. Ensuring the patency of the respiratory tract by incubating with an endotracheal tube or supralaryngeal airway devices (NZP laryngeal mask, laryngeal tube, combitube or oropharyngeal (nasopharyngeal) airways with manual fixation of the tilted head and neck).

6. Provide venous or intraosseous access. Administer medication.

Give humidified oxygen in the maximum available concentration of 10-15 l/min. or until reaching pulse oximetry indicators of 95% and above.

Epinephrine (1 mg) every 3-5 minutes. during cardiopulmonary resuscitation under a monitor-confirmed rhythm that does not require defibrillation (asystole).

Epinephrine (1 mg) after 3 defibrillations and thereafter every 3-5 minutes. during cardiopulmonary resuscitation in a rhythm requiring defibrillation.

Amiodarone (300 mg.) after the third defibrillation once immediately after the administration of epinephrine.

Items 5 and 6 should not delay or stop the main elements of cardiopulmonary resuscitation (defibrillation, chest compressions and artificial ventilation).

1. Immediately after determining the rhythm in case of sudden cardiac death, if necessary, perform defibrillation and immediately continue cardiopulmonary resuscitation until the next rhythm analysis in 2 minutes.

2. It is necessary to change the person performing chest compressions every 2 minutes at the time of rhythm analysis with a defibrillator, and in its absence - every 2 minutes, avoiding interruptions in cardiopulmonary resuscitation.

3. The team leader must constantly monitor the quality of cardiopulmonary resuscitation performed, and in case of poor performance, adjust the necessary elements of cardiopulmonary resuscitation to the appropriate level.

4. The team leader should consider the possibility of transporting the patient to the hospital without stopping cardiopulmonary resuscitation or after the appearance of signs of life.

Contraindicated and not recommended interventions in patients with sudden cardiac death:

1. It is not recommended to perform a preventive inspection of the oral cavity before starting cardiopulmonary resuscitation, if there are no objective reasons for this.

2. Administration of atropine is not recommended.

3. Endotracheal administration of drugs is not recommended.

4. Precardial shock is performed only under the condition of all the circumstances listed below:

– On the patient's monitor, ventricular fibrillation without a pulse was recorded.

- No more than 10 seconds have passed since the onset of ventricular fibrillation without a pulse.

- There is no defibrillator under the arms.

- In addition to you, there are also persons present in the room who can deliver a defibrillator. Conducting a precardial shock should not delay the delivery of the defibrillator: if there is no one nearby, you carry the defibrillator without applying a precardial shock.

5. Avoid pauses between chest compressions and artificial breaths, continue chest compressions while applying defibrillator electrodes. Pause in cardiopulmonary resuscitation only at the time of rhythm analysis with a defibrillator.

6. Avoid excessive ventilation.

7. Avoid overfatigue during cardiopulmonary resuscitation - the person performing chest compressions should be changed every 2 minutes.

4.3.4. Hospitalization

Justification

Urgent hospitalization of the patient in designated health care institutions of secondary medical care

Necessary actions of the head of the emergency (urgent) medical care team

Mandatory:

1. All patients with sudden cardiac death, regardless of gender, age and other factors, are subject to urgent hospitalization. It is necessary to take the patient's medical documentation and previous cardiograms from the hospital. The priority task of the emergency (urgent) medical care team is to transport patients to the center (department), where primary angioplasty (stenting) is possible.

2. During transportation, it is necessary to ensure the monitoring of the patient's condition, the implementation of medical measures and the readiness to carry out resuscitation measures.

3. Transportation is carried out on a stretcher after the rhythm is restored to the emergency (urgent) medical care department of a multidisciplinary hospital, or bypassing the reception department, directly to the intensive care unit, intensive care unit, heart attack unit, cardiac intensive care unit.

4. The territorial health authority must develop and approve an order, a local protocol (of the appropriate level), which ensures the organization of assistance to patients with sudden cardiac death, and interaction between health care institutions.

II. Defibrillation

The automatic external defibrillator independently determines the required shock energy. In the spoon biphasic defibrillator, the energy of the first and second defibrillation is set at the level indicated by the manufacturer of the specific device, the third and all subsequent defibrillations are performed with the maximum available energy. In the presence of a monophasic defibrillator, starting from the first defibrillation, the maximum possible discharge energy is set.

If a normal rhythm was restored by defibrillation, which again changed to a rhythm requiring defibrillation, a discharge is applied with the energy that first restored the normal rhythm. If it is ineffective, the energy is increased.

III. Ventilation

As soon as the patient's trachea is intubated, chest compressions must be resumed and continued without interruption (with the exception of defibrillation and pulse checking - if indicated), with a frequency of at least 100 min-1 and ventilation of the lungs with a frequency of approximately 10 breaths min-1 (times in 5 sec.) asynchronously. If it is not possible to ensure adequate oxygenation, in this way, pauses in compressions should be made for ventilation.

When using devices for artificial lung ventilation, it is first necessary to set the respiratory volume at the level of 6-7 ml per kg of body weight (at 10 breaths per minute), then titrate according to Pa02.

VI. Drug therapy

Epinephrine. Epinephrine increases the excitability of the myocardium and therefore is a potentially arrhythmogenic substance, especially in conditions of ischemia or hypoxia of the myocardium. After resuscitation, epinephrine can cause repeated ventricular fibrillation.

Epinephrine is available most often in two concentrations: 1 to 1000 (1ml-1mg of epinephrine) for parenteral administration; 1 to 10,000 (10 ml-1 mg of epinephrine) for external use. In the case of treatment of victims with defibrillation rhythms FSH/SHT without a pulse, epinephrine in a concentration of 1 mg. It is administered after 3 defibrillations, then every 3-5 minutes. After one defibrillation). In the treatment of non-defibrillation rhythms, asystole/pulseless electrical activity - 1 mg of epinephrine immediately after intravenous (intraosseous) access is performed, and then every 3-5 minutes. during the entire time of resuscitation.

Amiodarone. Amiodarone is an antiarrhythmic drug that stabilizes cell membranes, prolongs the duration of the functional potential and the refraction time

of cardiomyocytes of the atria and ventricles. Slows down atrioventricular conduction; a similar effect is also observed in additional conductive pathways. Amiodarone has a negative isotropic effect and the reason is the expansion of peripheral vessels by non-competitive blocking of α-receptors.

If ventricular fibrillation persists after the third defibrillation, it is necessary to introduce an initial dose of amiodarone 300 mg IV (or IV), dissolved in 20 ml of 5% glucose (which will reduce foaming of the drug in the syringe). An additional dose of 150 mg of amiodarone can be administered in case of recurrence of persistent ventricular fibrillation.

Lidocaine. Lidocaine is an antiarrhythmic drug that stabilizes membranes and acts by prolonging the refraction time of myocytes. Reduces the automatism of the ventricles and reduces their ectopic activity. Reduces the activity of depolarized arrhythmogenic tissues, while minimally affecting the electrical activity of normal tissues.

In the absence of amiodarone, it is possible to apply lidocaine in an initial dose of 100 mg (1-1.5 mg/kg) in the case of ventricular fibrillation without a pulse, resistant to three times defibrillation. If necessary, you can additionally enter a painful 50 mg. The total dose should not exceed 3 mg/kg during the first hour of treatment.

Magnesium sulfate

IV (or IV) administration of magnesium sulfate is a safe and often effective method of treating ventricular tachyarrhythmias.

With ventricular fibrillation resistant to defibrillation, it is necessary to start with a dose of 2 gv/v (4 ml of 50% magnesium sulfate = 8 mmol); the dose can be repeated after 10-15 minutes. For other types of tachyarrhythmia, 2 g should be administered over 10 minutes.

Sodium bicarbonate

Routine use of sodium bicarbonate during cardiopulmonary resuscitation is not recommended. The introduction of sodium bicarbonate (50 mmol) is recommended for cardiac arrest against the background of hyperkalemia or poisoning with tricyclic antidepressants. Repeated doses are administered according to clinical indications or repeated blood gas analysis.

V. Indicators of the quality of medical care.

5.1. List of indicators of the quality of medical care

5.1.1. Availability of a local protocol for the management of a patient with sudden cardiac death by a doctor and paramedic from emergency medicine.

5.1.2. Percentage of patients with sudden cardiac death who were defibrillated for pulseless ventricular fibrillation.

5.1.3. Percentage of patients with sudden cardiac death admitted to a specialized hospital with a hemodynamically significant rhythm after sudden cardiac death.

5.1.4. Percentage of emergency medicine doctors and paramedics who were trained in short-term cardiopulmonary resuscitation courses.

5.1.5. Percentage of patients who experienced sudden cardiac death and were discharged from the hospital with a satisfactory neurological picture.

5. SECTION 2

PROVISION OF EMERGENCY ASSISTANCE IN THE PRACTICE OF A FAMILY DOCTOR FOR SEIZURES

5.1. Seizures and loss of consciousness

Convulsions are sudden involuntary attacks of tonic-clonic contractions of skeletal muscles, which are often accompanied by loss of consciousness.

General information about convulsive syndrome. Convulsive syndrome is a pathological condition that manifests itself in the form of attacks of involuntary contractions of striated muscles caused by irritation of certain brain structures that control movements. The convulsive syndrome has a local character (localized convulsions) or can be generalized (in case of involvement of many muscle groups).

Seizures are distinguished:

- fast (clonic) – rapid change of contractions and relaxations;
- long with slow muscle contraction (tonic);
- of a mixed nature (clonic-tonic).

Seizure syndromes are divided by origin into epileptic and non-epileptic (symptomatic convulsive attacks), which are secondary, but can later transform into epileptic ones.

For the most part, convulsions are a manifestation of epileptic attacks. They can be generalized or local. Recent convulsions are observed on one side of the body or in one limb and indicate an area of the brain affected by excitement. In the case of generalized attacks, the entire cerebral cortex is irritated with the involvement of departments responsible for movement.

Epileptic seizures are possible in the presence of congenital defects in the development of the central nervous system, focal lesions of the brain (tumor, abscess), hereditary diseases of the metabolism, pathology of the cardiovascular system (congenital heart defects), as well as in the case of blood diseases (capillarotoxicosis, hemophilia, leukemia, thrombocytopenic purpura). Seizures can occur as a result of strong emotions.

Secondary seizures can be caused by:

- suffered injury;
- both endogenous and exogenous intoxication (uremia, liver failure, coma, poisoning with drugs and salts of heavy metals, household poisoning);
- vascular disorders (cerebral thromboembolism, arteriovenous aneurysm);
- brain tumors;
- physical injuries (electrical injury, thermal damage, sunstroke);
- metabolic disorders (hypoglycemia, hyponatremia, hypocalcemia, pyridoxine deficiency);
- infections (meningitis, encephalitis).

5.2. Classification of convulsions

Due to the cause of occurrence:

1. Convulsions, which are a nonspecific reaction of the brain to irritating factors: injuries, infections, intoxication, etc. These are encephalic or episodic epileptic reactions.

2. Symptomatic convulsions or symptomatic epilepsy against the background of an active current cerebral process (tumorous, inflammatory, parasitic, etc.).

3. Epilepsy - seizures against the background of organic lesions of the central nervous system.

According to the mechanism:

1. Epileptic
2. Non-epileptic

According to the duration of muscle contraction:

1. Tonic,
2. Clonic
3. Tonic-clonic.

By prevalence:

1. Generalized,
2. Local (focal).

According to the state of consciousness: with or without loss of consciousness.

Causes of seizures

1. Idiopathic epilepsy.
2. Infectious diseases of the brain.
3. Intoxication.
4. Fever (febrile convulsions).
5. Alcohol abstinence.
6. Metabolic disorders.
7. Side effects of medicinal products (neuroleptics, metoclopramide, etc.).
8. Physical factors (electrical injury, overheating).
9. Affective respiratory attacks in younger children.
10. Fainting.
11. Psychogenic (demonstrative) attacks.
12. Trunk spasms (hormeotonia, decortication, decerebration spasms)
13. Multifocal myoclonus.
14. The "rigid person" syndrome.
15. Tetanus.
16. Tetany (spasmophilia).
17. Metabolic myopathies.

If a seizure syndrome is suspected, the family doctor should refer the child to a specialized pediatric department for a thorough examination.

Algorithm of examination of a child by a family doctor.

1. Find out the reasons for the court:

evaluate the course of pregnancy, childbirth, family history;

conduct a thorough assessment of neurological status;

estimate the size of the head circumference (to rule out hydrocephalus);

perform an examination with an ophthalmoscope (to detect signs of intrauterine infections, stagnant changes, hemorrhages on the fundus).

2. Send the child for biochemical tests (blood and urine tests for the content of calcium, sodium, magnesium, bilirubin, urea, ammonium, glucose, acid-base status).

3. Send for functional and morphological studies (electroencephalography, neurosonography and electrocardiography, computer tomography, if necessary - angiography).

4. Send the child for an x-ray of the skull, which can reveal:

- small sizes or premature closure of the crowns and seams;
- foci of calcification;
- increase in the size of the skull;
- signs of intracranial hypertension (presence of finger depressions, changes in the contours of the Turkish saddle), which indicates the organic origin of the convulsive syndrome;
- examination for SARS-infection.

Algorithm for diagnosis of convulsive syndrome in children.

1. In the case of hyperthermia complicated by a convulsive syndrome (find out the presence of an increase in body temperature before the development of a convulsive attack):

- exclude intoxication due to infection;
- exclude neuroinfections.

2. At a normal body temperature, find out the cause of the seizure:

poisoning, injury to the central nervous system, rickets, spasmophilia, epilepsy, hysteria.

3. Assess skin color (cyanosis, pallor).

4. Find out if:

- disorders of cardiac activity (tachycardia, bradycardia, hemodynamic disorders);
- respiratory disorders (apnea, pathological types of breathing, shortness of breath);
- meningeal symptoms (rigidity of the muscles of the back of the head, positive symptoms of Kernig, Brudzinsky);
- vegetative disorders (widespread dermographism, anisocoria).

Algorithm of the sequence of actions in case of convulsive syndrome in children (according to the Unified clinical protocol of primary, emergency, secondary (specialized) and tertiary (highly specialized) medical care. Epilepsies in children. 2014)

1. Lay the child on a flat surface on his back, put the roller under the neck, turn his head to the side.

2. Remove all damaging items.

3. Provide fresh air.

4. Unbutton tight clothes.

5. Calm the child (remove sound and light stimuli).

6. Measure the body temperature (if it exceeds 38.5 °C, give the child antipyretic drugs and carry out cooling).

7. Conduct anticonvulsant therapy.

The algorithm for providing emergency care at the pre-hospital stage in case of convulsive syndrome in children:

1. Administer tranquilizers: benzodiazepine (sibazone, relanium, seduxen) - 0.5% solution intramuscularly at the rate of 0.3-0.5 mg/kg. If there is no effect, repeat the injection every 10-15 minutes three times. It is possible to introduce tranquilizers through the rectum using a catheter attached to a syringe (0.5 mg/kg in the first year of life; 2.5-5 mg at the age of 1-3 years, 5-7.5 mg at 3-6 years; 7.5-10 mg at school age, in case of ineffectiveness, the administration should be repeated after 10-15 minutes).

2. Dehydration therapy: Lasix 2-3 mg intramuscularly or intravenously.

3. If possible, oxygen inhalation through a nasal catheter or mask.

4. Hospitalization to the neurological department, in case of violation of vital functions - to the resuscitation and intensive care department.

Epilepsy is a chronic brain disease of various etiologies, characterized by repeated epileptic seizures resulting from excessive neuronal discharges, and accompanied by various clinical and paraclinical symptoms.

In adults, the following pathological conditions can cause convulsive attacks:

- violation of the use of anticonvulsants;
- acute disorders of cerebral circulation;
- acute inflammatory diseases of the brain;
- closed craniocerebral injury;
- brain tumors (primary and metastatic);
- alcoholism (alcohol withdrawal syndrome);
- acute poisoning with organophosphorus substances and psychotropic drugs;
- mental and physical fatigue;
- blood diseases (hemophilia, leukemia, capillarotoxicosis, thrombocytopenic purpura);
- uremia, liver failure;
- fainting, respiratory-affective convulsions due to strong emotions;
- hereditary metabolic diseases;
- congenital defects in the development of the central nervous system;
- pathology of the cardiovascular system (congenital heart defects, collapse);
- hysteria.

It is important to distinguish between an epileptic attack and epilepsy as a disease. Single or occasional epileptic seizures or an epileptic reaction, occurring in a certain situation, do not recur in the future. Epilepsy should not include repeated epileptic seizures in the case of acute cerebral diseases (in case of cerebral circulation disorders, meningitis, encephalitis).

5.3. Single and serial attacks and epileptic status are distinguished

In the case of observation of an attack by a specialist, the diagnosis is established quite easily. The issue of differential diagnosis of epileptic clonic-tonic convulsions and fainting must be resolved by analyzing clinical and anamnestic data from both the patient and eyewitnesses.

The patient should be asked about the presence of predictors of an attack. Typical manifestations of aura are a feeling of discomfort in the stomach, unusual sounds. The aura usually recurs, preceding another epileptic attack. The patient should be asked about how consciousness is restored. The presence of disorientation in space and time, drowsiness for several minutes or more indicate convulsions.

Epilepsy is also characterized by tongue-biting, muscle pain that lasts for several hours and even days. Involuntary urination cannot be considered as a differential diagnostic sign. Eyewitnesses should be interviewed about the nature of movements of individual parts of the body during an episode of loss of consciousness [29].

Other clinical information is less informative for the diagnosis of epilepsy:

- burdened heredity;
- nocturnal occurrence of the episode;
- urinary incontinence during a seizure episode;
- trauma during a seizure episode;
- headache and drowsiness after a seizure episode.

Status epilepticus occurs as a result of a prolonged epileptic attack or attacks that are repeated at short intervals of time, which is clinically characterized by progressive deterioration, the addition of respiratory, circulatory and metabolic disorders that grow, ultimately leading to the development of a comatose state.

Provocative factors of epileptic attacks are as follows:

- alcohol use,
- physical and mental fatigue,
- violation of regular use of anticonvulsants. Differential diagnostic criteria of an epileptic attack:
- suddenness of development;
- the possibility of development under any conditions, not always the presence of the influence of a previous psychogenic factor;
- absence of subjective and objective signs characteristic of fainting (noise and ringing in the ears, flickering of flies in front of the eyes, general weakness, pale skin, sharp drop in blood pressure);
- suppression of consciousness during an attack;
- the appearance of mydriasis with areflexia of the pupils to light;
- development of postparoxysmal symptoms up to epileptic coma.
- Differential diagnosis should be based on the following information:
- features of clinical manifestations;
- anamnesis results;

- results of examination of the fundus;
- examination results at the secondary and tertiary levels:

- CT scan of the brain;
- electroencephalography;
- lumbar puncture;
- angiography.

Convulsions during epileptic attacks must be differentiated from such conditions as cramps, hyperkinesis, syncope.

Algorithm of emergency care in case of an epileptic attack.

1. Prevention of killing the patient.

2. Ease of breathing.

3. Prevention of tongue biting: between the corner teeth, it is recommended to insert the handle of a tablespoon wrapped in a cloth, or, if there is none, a small wooden object (it is unacceptable to insert metal objects, especially between the front teeth, because this can cause damage to the teeth and asphyxiation if they get into the upper respiratory tract).

4. After the end of the attack, the patient should not be woken up and he should not be given any medicines.

Algorithm of emergency care in case of epileptic condition.

Considering the fact that the family outpatient clinic does not always have the necessary equipment at its disposal, the algorithm of actions of the family doctor is as follows:

1. Ensuring patency of the upper respiratory tract.

2. Elimination of the possibility of the tongue sinking.

3. Intravenous slow administration of 2-4 ml of 0.5% seduxen solution (if within 5-10 minutes the dose did not eliminate the convulsive syndrome, it is necessary to re-introduce this drug).

4. In case of inefficiency of re-introduction of seduxen, a transitional ultra-short-acting barbiturate is justified: hexenal or thiopental sodium (300-400 mg of 1% solution intravenously). These drugs have a depressing effect on the respiratory center, and in case of their overdose, breathing may stop (!).

5. Elimination of signs of acute cardiovascular insufficiency is carried out with cardiac glycosides (for example, intravenous administration of 0.5-0.7 ml 0.05 % solution of strophanthin or other drugs of this group) and vasoactive agents such as mesaton or norepinephrine hydrotartrate.

6. Elimination of cerebral edema (osmoduretics).

7. Improvement of the rheological properties of blood (low molecular weight dextran or heparin 2500-5000 units subcutaneously or intramuscularly 2-4 times a day).

8. Administration of antihypoxants (drugs such as GOMK).

9. As soon as possible, it is necessary to transfer the patient to artificial ventilation of the lungs with the introduction of muscle relaxants.

Syncopal states are states characterized by spontaneously occurring transient loss of consciousness and translational tone due to cerebral hypoperfusion followed by spontaneous recovery.

One of the most important problems in the practice of a family doctor is a patient's sudden loss of consciousness, which can be a manifestation of various cerebral, somatic, including cardiac pathologies and can be classified as one of the multidisciplinary problems of clinical medicine, which requires correct syndromological and nosological diagnosis.

The most common cases of loss of consciousness include fainting, coma, and collapse. The latter, in turn, can be an independent manifestation or a harbinger of fainting.

Fainting, or syncope (from Greek supkore – cutting off, contraction), is clinically characterized by generalized muscle weakness, a decrease in postural tone, blood pressure, and loss of consciousness. The most common reason is a decrease in blood circulation in the brain and brain centers.

Coma (from Greek kota – deep sleep) – complete loss of consciousness and orientation with manifestations of neurological and autonomic disorders. The duration of the coma depends on the degree of violations.

Coma of various etiologies have common manifestations, which include loss of consciousness, loss or reduction of sensitivity, reflexes, muscle tone, and impaired vegetative functions of the body.

The Glasgow coma scale is given in table. 2.1.

Table 2.1

Glasgow Coma Scale

Symptoms	Points
Opening the eyes	
- absent	1
- to painful stimuli	2
- by command/voice	3
- spontaneously with flashing	4
Motor response	
- absent	1
- extension of the arm to a painful stimulus	2
- bending of the arm to a painful stimulus	3
- opening the hand to a painful stimulus	4
- the hand localizes the place of the painful stimulus	5
- execution of commands	6
Verbal response	
- absent	1
- various sounds, but not words	2
- inappropriate words or phrases	3
- confused language	4
- norm	5

Total score on the Glasgow Coma Scale	Traditional terms
15	Clear consciousness
13-14	Muting
9-12	Sopor
4-8	Coma
3	Brain death

Causes of comatose states:

- Sharp reduction of the intracranial space - intracerebral hemorrhage, brain infarction, abscesses, brain tumors, post-traumatic intracerebral hematomas, epidural and subdural hematomas. Pronounced symptoms of brain damage are observed;
- Exogenous intoxications (alcohol, barbiturates, opiates, benzodiazepines, antidepressants, carbon monoxide, etc.), acute metabolic disorders (hypoxia, diabetes, renal, hepatic, adrenal, acute cardiovascular insufficiency); general infections (pneumonia, typhus, sepsis, infectious-toxic shock); shock of various etiology; sharp hyperthermia and hypothermia; status epilepticus; pronounced hypertensive encephalopathy. Focal symptoms of brain damage are not observed or are not expressed sharply;
- Subarachnoid hemorrhage, as a result of aneurysm rupture, severe forms of meningitis or encephalitis. Meningeal symptoms, changes in cerebrospinal fluid, as well as focal neurological symptoms are revealed.

Signs that you need to pay attention to when examining a comatose person is listed in the table. 2.2

Table 2.2

Examination of a comatose patient

Skin	Moist, dry, hyperemic, cyanotic, jaundiced
Head and face	Presence of injuries
eyes	Conjunctiva (hemorrhage, jaundice), pupil reaction to light; fundus (edema of the optic disc, hypertensive or diabetic retinopathy)
Nose and ears	Discharge of pus, blood, liquorice, acrocyanosis.
Tongue	Dryness, bite marks or scars
Breath	The smell of acetone, urine, alcohol
Neck	Stiffness of the occipital muscles, pulsation of the carotid arteries

Chest	Frequency, depth, rhythmicity of breathing
Heart	Rhythm disturbances (bradycardia), sources of vascular embolism brain (mitral stenosis).
Stomach	Enlargement of the liver, spleen or kidneys
hands	Blood pressure, hemiplegia, scars from injections
Brushes	Frequency, rhythm and filling of the pulse, tremor

Collapse (from the Latin coIabor, соллasus – weakened; the one who fell) is a vascular insufficiency that develops acutely.

30-50% of adults have experienced fainting or a similar condition at least once in their life. The prevalence of isolated syncope increases in the age group of people older than 75 years. About 35% of patients after a first episode of syncope are at risk of recurrence during the next three years of follow-up, with 82% of recurrences occurring within the first two years.

Fainting of a noncoronarogenic nature and syncopal states of unknown origin are mostly observed in women, and cardiogenic variants of syncope develop more often in men.

The family doctor must remember that the mortality within one year after the first episode of syncope in persons with cardiac fainting and organic heart damage is 18-33%, with non-cardiac - 0-12%, with fainting of unknown etiology - 6%.

The following syncopal states are related to the rubrics of the ICD-10:

- psychogenic vertigo (F48.8);
- sinocarotid syncope (G 90.0);
- thermal fainting (T 67.1);
- orthostatic arterial hypotension (L95.1), including neurogenic (G90.3);
- Adams - Stokes syndrome (L45.9).

The classification of syncopal conditions proposed by the experts of the European Society of Cardiology (ESS, 2009) deserves attention.

Recommendations on the diagnosis and treatment of syncopal states of the Working Group on the diagnosis and treatment of syncopal states of the European Society of Cardiology (ESC), 2009 edition.

Classification of syncopal states:

I. Reflex (neurogenic) fainting:

1) vasovagal:

- due to emotional stress: fear, pain, fear of blood, medical manipulations and instruments;
- orthostatic load.

2) situational:

- sneezing, coughing;
- stimulation of the gastrointestinal tract: swallowing, defecation, visceral pain;
- reaction to urination;
- after physical exertion;
- postprandial (after eating);
- others (laughter, playing brass instruments, lifting weights);

3) irritation of the carotid sinus;

4) atypical (unspecified trigger or atypical manifestations).

II. Fainting due to orthostatic hypotension:

1) primary vegetative insufficiency:

- pure autonomic failure, multiple system atrophy, disease Parkinson's disease with autonomic failure, Lewy dementia;

2) secondary vegetative insufficiency:

- diabetes, amyloidosis, uremia, spinal cord injury;

3) orthostatic hypotension provoked by chemical substances / medicines:

- alcohol, diuretics, vasodilators, phenothiazides, antidepressants;

4) deficiency of circulating blood volume:

- bleeding, diarrhea, vomiting, etc.

III. Cardiogenic syncope:

1) arrhythmogenic (primary cause):

a) bradycardia:

- sinus node dysfunction, including tachy-brad syndrome;

- violation of AV conduction;
- dysfunction of the implantable pacemaker;

b) tachycardia:

- supraventricular;
- ventricular (idiopathic, ion channel function pathology due to structural cardiac pathology);

c) drug-induced bradycardia and tachyarrhythmias;

2) structural pathology:

a) cardiac: valvular heart defects, acute coronary syndromes, hypertrophic cardiomyopathy, intracardiac volumetric formations (myxoma, tumors, etc.), pericarditis / tamponade, congenital anomalies of the development of coronary arteries, dysfunction of valve prosthesis, etc.;

b) others: embolism of a small circle of blood circulation, acute dissection of an aortic aneurysm, pulmonary hypertension.

Non-syncopal states that can be mistaken for fainting

1. Disorders with partial or complete loss of consciousness:

- metabolic disorders, including hypoglycemia, hypoxia, hyperventilation with hypocapnia;
- epilepsy;
- intoxication;
- transient ischemic attacks in the vertebral-basilar basin.

2. Disorders without loss of consciousness, resembling fainting:

- catalepsy;
- "drop attacks" (benign syndrome: sudden fall of middle-aged or elderly women without loss of consciousness);
- psychogenic "fainting" (psychosomatic disorders);
- transient ischemic attacks of carotid origin.
- Not always developed fainting can be attributed to one of the listed headings, since in some diseases syncope is based on several pathophysiological factors at once.

Below, the main causes of syncopal states will be considered from the perspective of a general practitioner - in the interests of his practical activity and taking into account the provision of emergency care.

Algorithm for initial assessment of the condition of a patient with episodic loss of consciousness. During the initial examination, after a thorough collection of history and complaints, it is necessary to get answers to three main questions:

1. To what category does loss of consciousness belong?
2. Whose patient has an organic disease of the heart and nervous system?
3. Are there anamnestic and clinical features that could indicate another pathological condition?

It is necessary to establish whether the patient belongs to the category of persons with a high risk of sudden death and whether he has:

- obstructive form of hypertrophic cardiomyopathy;
- myocardial ischemia;

- post-infarction cardiosclerosis with severe disorders of sinus node function and atrioventricular conduction;
- atrial myxoma;
- arrhythmogenic right ventricular dysplasia;
- Brugada syndrome;
- syndrome of prolonged Q-T interval;
- Wolff-Parkinson-Bate syndrome.

If the specified diseases can be excluded, it is worth directing the diagnostic search to identify other causes of fainting, followed by taking urgent medical measures, as well as improving the quality of life and preventing possible episodes of fainting.

It is necessary to warn family members about the possibility of relapses and the need to prevent injury during a fall, to teach first aid.

When assessing the condition of a patient with syncopal episodes, it is necessary to proceed from the fact that neurocardiogenic syncope occurs most often, arrhythmogenic fainting is observed somewhat less often.

It is necessary to take into account the age at which syncope debuts:

- neurocardiogenic syncopal states, conversion reactions (for psychiatric reasons), primary arrhythmia (Wolf-Parkinson-Bate syndromes, prolonged interval syndrome (Q–T, Brugada syndrome, obstructive form of hypertrophic cardiomyopathy) most often occur in children, adolescents and young people;
- in middle-aged patients, neurocardiogenic fainting is most often observed, the percentage of people with situationally determined syncope (during swallowing, coughing, urination, defecation), orthostatic fainting, syncope-like episodes associated with panic attacks is increasing;
- in elderly patients, the causes of syncopal states can be: orthostatic or postprandial hypotension, degenerative aortic stenosis, pulmonary embolism, carotid sinus hypersensitivity syndrome, transient myocardial ischemia, myocardial infarction, heart rhythm disturbances on the background of coronary heart disease, disorders of the functional state of the sinus node due to ischemia or sclerodegenerative processes, transient ischemic attacks in the vertebrobasilar pool.

5.6. Algorithm for providing emergency care during fainting

1. It is necessary to take measures aimed at improving the blood supply and oxygenation of the brain:

- eliminate provoking factors;
- transfer the patient to a horizontal position;
- give the legs an elevated position;
- provide access to fresh air;
- free from tight clothing;
- carry out a light body massage;

- provide a reflex effect on the centers of breathing and cardiovascular regulation (inhalation of ammonia vapors, splashing the face with cold water);
- turn the head to the side (prophylaxis of tongue depression) if there is no damage to the subclavian and carotid arteries.

2. In the absence of an effect from the measures taken and with a pronounced decrease in blood pressure:

- introduce sympathicotonic agents: 1% mesaton solution, 5% ephedrine hydrochloride solution;
- in case of heart rhythm disorders, prescribe antiarrhythmic drugs;
- in cases of bradycardia or cardiac arrest, introduce a 0.1% solution of atropine sulfate, apply indirect heart massage.

If prolonged unconsciousness is accompanied by significant disturbances of cardiac activity and breathing, in addition to carrying out the entire complex of resuscitation measures, it is necessary to ensure urgent hospitalization of the patient.

6. SECTION 3

OTHER EMERGENCY CONDITIONS OFTEN ENCOUNTERED IN THE PRACTICE OF A FAMILY DOCTOR

6.1. Asthmatic status

Asthmatic status - a severe attack of bronchial asthma lasting more than 24 hours, characterized by the development of acute respiratory failure, which is caused by obstruction of the airways in the conditions of the formation of resistance to bronchodilators (short-acting b 2-agonists), hypercapnia, hypoxemia, polycythemia.

Diagnostics:

A. Potential risk factors:

- exacerbation of the inflammatory process
- abrupt withdrawal of corticosteroids or cromons
- overdose (b2 -agonists of short action)
- significant contact with allergens, which increases bronchial hyperreactivity
- passive smoking, especially in children
- excessive exposure to occupational hazards
- abuse of narcotics, sedatives that depress the respiratory center
- psychoemotional stress
- climate and weather factor

B. Types of asthmatic status

- status that develops slowly (severe exacerbation of bronchial asthma, life-threatening asthma)
- anaphylactic (on the background of anaphylactic shock)

C. Clinical symptoms of asthmatic status:

- airway obstruction resistant to bronchodilators for more than 24 hours
- increase in shortness of breath, shortness of breath, wheezing, chest tightness
- Respiratory rate more than 30 in 1 minute, heart rate more than 120 in 1 minute
- Peak expiratory volume rate is less than 50% of the proper value
- shortness of breath at rest, while talking

Stages of the clinical course of asthmatic status

I. Stage of relative compensation (absence of ventilation disorders)

- reducing the amount of sputum
- consciousness and orientation are preserved
- forced position in bed
- pale cyanosis, sweating

- tachycardia 100-120 in 1 minute
- Respiratory rate 30-40 in 1 minute
- Hypertension
- RaO2=60-70 mmHg.
- RaCO2=40-45 mmHg.
- peak volume exhalation rate 30% of the appropriate values

II. Stage of decompensation ("no lung") - progressive ventilation disorders

- absence of sputum
- emergence of periods of excitement and apathy
- forced position in bed
- immobilization
- sharp diffuse cyanosis
- shortness of breath, hyperhidrosis, swelling of neck veins
- "no lung" (focal atelectasis)
- tachycardia 120-140 in 1 minute
- ChD40-50 in 1 minute
- Hypotension
- RaO2<50-60 mmHg.
- PaCO2>50-70 mmHg.
- Peak volume exhalation rate 20% of the proper values

III. Hypoxemic hypercapnic coma

- loss of consciousness, decreased reflexes
- labored, agonal breathing
- pulse 140-160 in 1 minute, paradoxical, pendulum-like
- hypotension, collapse, shock
- RaO2<40 mmHg.
- PaCO2>70-80 mmHg.
- organic neurological disorders

 Control methods for asthmatic status

- ECG
- intermittent diuresis
- hematocrit, gas composition of blood
- blood electrolytes
- CO2 saturation
- peak flowmetry or spirography

Algorithm of pharmacotherapy of patients with asthmatic status

I. Hospitalization in the intensive care unit, with stage 2 to the intensive care unit

II. Oxygen therapy (30-40% O2 through a nasal catheter or mask)

III. Infusion therapy (restoration of circulating blood volume, improvement of microcirculation, thinning of sputum):

- up to 3-4 liters in 1 day, up to 2.5 liters in 2 days
- 5% glucose, reopolyglukin, polyglukin, refortan

• per 500 ml of infusion solution – 0.5 ml of heparin (to prevent hypercoagulation, thin sputum, increase sensitivity to hormones)

IV. Hormonal therapy (prednisolone or methylprednisolone) painful or drip:

• 60-90 mg of prednisolone IV as a jet + 20-40 mg of prednisolone IV as a drip in 400 ml of physical. of the solution, in case of no effect, 90 mg every 2-3 hours (before reaching the peak volume exhalation rate of 50%), in case of improvement, 30 mg every 3 hours. The daily dose is 1-1.5 g

• II stage of the asthmatic status – the dose is increased to 1.5-2 g/day, if necessary, 1000 mg IV drip methylpred, when the stage of "silent lung" is removed, the dose is reduced by 25% and prescribed in a maintenance dose of up to 3- 5 days

V. Prescribing methylxanthines (do not prescribe with a heart rate of more than 120 in 1 minute!!)

• The first 30 minutes are a calculated shock dose

5-10 mg/kg (if the patient receives eufilin for the first time in his life) 3 mg/kg (if he received it earlier)

• subsequent administration of Euphilin within 3.5 hours

0.9 mg/kg/h (smoker)

0.6 mg/kg/h (non-smoker)

0.25 mg/kg/h (liver disease)

VI. Aggressive use of short-acting b2 -agonists via nebulizer every 20 minutes for 2-4 breaths until improvement and atrovent every 20 minutes via nebulizer

VII. Antibiotics according to clear indications (purulent sputum, infiltration in the lungs)

VIII. Indications for parenteral administration of adrenaline:

- anaphylactic form of asthmatic status
- ineffectiveness of inhalation administration of short-acting b 2-agonists
- rapid decompensation of the patient

IX. Ventilator for stage III asthmatic status

6.2. Emergency conditions of diabetes. Comatose states in diabetes

Hypoglycemic coma

Hypoglycemic coma is an acute complication of diabetes caused by a sharp drop in blood sugar with a subsequent decrease in glucose utilization by brain tissue and brain hypoxia.

Etiology. 1. Overdose of insulin or hypoglycemic drugs (chlorpropamide, tolbutamide), discrepancy between the dose of insulin and the need for it.

2. Insufficient intake of carbohydrates.

3. A long break in food.

4. Excessive muscle work.

5. A decrease in the insulin-inactivating capacity of the liver and kidneys due to a decrease in insulinase activity when diabetes is combined with liver or kidney diseases.

6. Labile course of diabetes.

7. State after ketoacidosis.

8. Alcohol intoxication.

9. B-cell adenoma (insuloma).

Pathogenesis. Hypoglycemia is accompanied by activation of the sympathoadrenal system, an increase in the level of adrenaline, norepinephrine. Hypoglycemia also leads to irritation of the hypothalamus, increased activity of the pituitary gland and adrenal cortex with an increase in the level of counterinsular hormones - ACTH, STH, glucocorticoids.

Hypoglycemia causes a severe violation of brain trophicity - hypoxia, hypofunction of the cortex and diencephalic zone. The development of irreversible changes is possible. Paresis of cerebral vessels, edema of the cerebral substance, thrombosis develops.

Clinical manifestations of hypoglycemic coma. Hypoglycemic coma develops quickly, often suddenly.

The severity of clinical manifestations depends on the sensitivity of the central nervous system to hypoglycemia.

Mild hypoglycemic state

Feeling hungry. Worry, anxiety, irritability, aggressiveness. Sharp general weakness, paleness, sweating, chills, tremors. Headache, dizziness, hyperesthesia. Numbness of the tongue and lips. Palpitation. Nausea, vomiting.

Pronounced hypoglycemic state

Sharp excitement, aggressiveness, hallucinations, fear, inappropriate behavior. Stunnedness, confused consciousness develops quickly. Increase of tendon, periosteal reflexes. Babinski's positive symptom. Tonic, clonic convulsions. The development of meningeal syndrome is possible - a decrease in tendon reflexes, hypotonia of muscles, anisocoria, nystagmus, sluggish reaction of the pupils. The pupils are wide, the tone of the eyeballs is normal. Temperature, breathing without features. The pulse is normal or accelerated. Angina attacks can transform into myocardial infarction. Strokes are possible.

Deep hypoglycemic coma

Complete loss of consciousness, deep coma. Areflexia. Termination by the court. Cessation of sweating. Hypothermia. Breathing is shallow. Bradycardia, hypotension.

Differential diagnosis is carried out with non-diabetic hypoglycemia. Primary hyperinsulinism (organic, absolute): severe deficiency

blood circulation, inflammatory and destructive liver damage, kidney failure.

Secondary hyperinsulinism (functional, symptomatic): surgical interventions on the gastrointestinal tract - gastrectomy, gastrostomy with accelerated absorption of carbohydrates and release of insulin.

Overeating after prolonged fasting. Hypersensitivity to insulin.

Hypoglycemia is possible with some endocrine diseases: Simmonds' syndrome, Shien's syndrome, pituitary dwarfism, hypothyroidism, Addison's disease, congenital hyperplasia of the adrenal cortex (silvtrashayusha form).

Deficiency of counterinsular hormones can be observed with renal glucosuria, malnutrition, pregnancy, lactation, liver cirrhosis, cholangitis, cholecystitis, extrapancreatic tumors.

Laboratory diagnostics. A hypoglycemic state can develop even with normal values of glycemia - with a sharp decrease in the level of sugar in the blood serum, adaptation of the central nervous system to high levels of blood sugar.

Against the background of hypoglycemia, "hungry" ketosis and acidosis can develop due to the activation of gluconeogenesis.

Treatment. 1. 40% glucose 20-40-100 ml is injected intravenously. The criterion of the sufficiency of the dose is the restoration of consciousness. Then switch to infusion of 5% glucose - hypoglycemia may recur.

2. Effective intramuscular administration of 1 ml of 1% glucagon, repeated administration through 10 minutes.

3. Subcutaneously inject 0.5-1.0 ml of 0.1% adrenaline solution, intravenously or intramuscularly 150-200 mg of hydrocortisone. With recurrent hypoglycemia every 2 hours, 1-2 ml of glucagon is administered intramuscularly, as well as glucocorticoids (75 mg of hydrocortisone or 30 mg of prednisolone) - intravenously as a drip 4 times a day.

4. To improve cerebral blood circulation, inject 5-10 ml of 25% intravenously magnesium sulfate solution.

5. Due to the danger of cerebral edema, in case of prolonged hypoglycemic coma, intravenous drip administration of 15-20% mannitol solution (0.5-1.0 g per 1 kg of body weight) is prescribed.

6. To improve glucose metabolism, 100 mg is administered intramuscularly cocarboxylase 5 ml of 5% ascorbic acid solution.

Oxygen therapy with humidified oxygen is carried out.

Cardiac and vascular drugs are used according to indications.

Ketoacidotic coma

Ketoacidotic coma is an acute complication of diabetes mellitus

accumulation of ketone bodies in the body, dehydration and acidosis. Ketoacidotic coma is the most frequent variant of acute disorders of carbohydrate metabolism, observed in 1-6% of patients with diabetes. The mortality rate from ketoacidotic coma in specialized medical institutions is 5%, since the body can tolerate significant hyperketonemia - up to 170 mmol/l.

Ketoacidosis - isolated biochemical changes without clinical symptoms.

Ketoacidotic state - pronounced biochemical changes and a number of clinical symptoms caused by them.

Etiology. 1. Late diagnosis of diabetes.

2. Inadequate treatment of diabetes - withdrawal of insulin or inconsistency of the dose of insulin with the need for the drug.

3. A sharp increase in the need for insulin, which causes decompensation of carbohydrate metabolism:

a) physical stress (injuries, operations, burns, frostbite);

b) mental stress;

c) severe intercurrent diseases (infections, inflammation, vascular disasters);

d) fatty infiltration of the liver;

e) lipid diet.

4. Prolonged vomiting of various genesis.

Pathogenesis. In the pathogenesis of diabetic coma, the main role is played by insulin deficiency, which reduces the utilization of glucose by tissues, increases gluconeogenesis in the liver, which leads to hyperglycemia and glucosuria. Insulin deficiency contributes to the increase of lipolysis and the accumulation of triglycerides, phospholipids, cholesterol, acetoacetic and beta-oxybutyric acids in the blood. Increased formation of ketone bodies in the liver with reduced utilization in extrahepatic tissues leads to ketoacidosis and ketonuria.

Hyperglycemia increases the osmotic pressure in the extracellular fluid, which promotes the flow of water and cellular electrolytes, especially potassium and phosphorus, from the cells into the intercellular spaces, the process of cellular dehydration develops. An increase in the concentration of glucose in the glomerular filtrate of the kidneys prevents its reabsorption in the tubules, causing osmotic diuresis. Extra- and intracellular fluid loss can reach 10% of body weight.

Accumulation of acetoacetic and beta-oxybutyric acids in the extracellular fluid strongly shifts the bicarbonate buffer system towards a decrease in pH, which causes an increase partial pressure of carbon dioxide and accumulation of hydrogen ions. These changes in the chemical composition of the blood lead to irritation of the respiratory center and the appearance of Kussmaul breathing, characteristic of diabetic acidosis. It is believed that the main role in the pathogenesis of acidosis an increase in the concentration of hydrogen ions, and not ketone bodies, plays a role, since their level does not correlate with the degree of severity of clinical manifestations. Elimination of excess hydrogen ions is achieved by oxidation of ketone bodies and excretion of hydrogen ions by the kidneys in the form of ammonia or by exchanging hydrogen ions for sodium ions in the renal tubules.

Since the utilization of ketone bodies in diabetic coma is sharply reduced, the balance of hydrogen ions is achieved as a result of their excretion by the kidneys. Functional disorders of the kidneys are characteristic features of diabetic acidosis. As a result of dehydration, glomerular filtration decreases and the supply of amino acids and glutamine to the cells of the renal tubules, where they are deaminated and ammonia is formed.

A change in the concentration of hydrogen ions disrupts the electrolyte gradient between the cellular and extracellular fluid, which leads to the loss of potassium, magnesium, and phosphorus ions in the urine. With a deficiency of potassium in the cells of the kidney tissue, the removal of hydrogen ions is disrupted, which creates a pathological cycle that leads to further depletion of sodium and potassium reserves.

Osmotic diuresis, like vomiting, contributes to the loss of sodium and chlorine. A study of the water-electrolyte balance in diabetic coma showed that the loss of extra- and intracellular fluid reaches 6 liters, sodium - about 500 meq, chloride - 390 meq, potassium - 350 meq. Decreased utilization of glucose by brain tissue, combined with cellular dehydration, cerebral anoxia and acidosis, is the cause of unconsciousness in

diabetic coma. Together with pronounced hyperglycemia, the content of sodium bicarbonate decreases to 15 meq/l and pH decreases to 7.3-6.8.

Scheme of the pathogenesis of ketoacidotic coma Decrease in the level of immunoreactive insulin. Decrease in the number of insulin receptors. Decreased affinity of insulin receptors.

Decreased hexokinase activity. Increase in the activity of glucose-6-phosphatase. A decrease in the utilization of glucose in the tissues increases the energy deficit in the tissues.

Compensatory increase in the secretion of counterinsular hormones (STH, ACTH, glucocorticoids, catecholamines). Activation of lipid metabolism:

the activity of lipolysis increases, the severity of lipogenesis decreases.

The main energy substrate in the body is non-alcoholic fatty acids.

The breakdown of NEG in the liver increases. The formation of acetoacetic acid in the liver increases. The resynthesis of acetoacetic acid into fatty acids decreases. The oxidation of acetoacetic acid in the Krebs cycle is reduced. Activation of gluconeogenesis: breakdown of glycogen in the liver; breakdown of proteins with transamination of amino acids in the liver.

Acute hyperglycemia. Pronounced glucosuria. Increased osmotic pressure of extracellular fluid, polyuria. Intracellular dehydration, up to 10% of body weight (6-7 liters) is lost.

Metabolic, respiratory, renal acidosis (renal blood flow decreases, glomerular filtration and renal excretion of H+ decreases).

Predictors of ketoacidotic coma. The clinical manifestations of coma are preceded by a period of decompensation of diabetes, manifested by polyuria, polydipsia, weight loss, anorexia, nausea and vomiting. The harbinger period can last several days, sometimes even weeks - the manifestations of decompensation of diabetes (polyuria, polydipsia, anorexia, weight loss) increase.

As ketoacidosis develops, the initial manifestations of intoxication with ketone bodies appear (nausea, vomiting, abdominal pain, the smell of acetone from the mouth), signs of dehydration of the body progress.

Clinical manifestations of ketoacidotic coma

Damage to the central nervous system. A throbbing, throbbing headache. Disturbance of consciousness:

Stage 1 (mild ketoacidotic state). Drowsiness, weakness, lethargy, fatigue, apathy, but the patient can adequately answer questions.

2nd stage (pronounced ketoacidotic state). Stunned: reactions are slowed down, inadequate, contact with the patient is difficult. Reflexes are preserved.

3rd stage (severe ketoacidotic state). Sopor: consciousness is absent, the patient can be awakened only with the help of strong stimuli, speech contact with the patient is impossible. Tendon reflexes are reduced, vital reflexes (swallowing, pupillary, corneal) are preserved. Pain sensitivity is preserved.

4th stage (ketoacidotic coma). Comatose state: complete loss of consciousness, does not respond to any stimuli. Loss of tendon, periosteal, skin reflexes, welcome reflexes are weakened.

Damage to the respiratory organs. Rare, noisy Kussmaul-type breathing (prolonged inhalation, short exhalation, long pause), caused by acidosis. The smell of acetone in the air that the patient exhales.

Damage to the cardiovascular system. Blood pressure decreases or is normal. The pulse is frequent, with weak filling. Rhythm disturbances caused by hypokalemia (ventricular extrasystole, atrial fibrillation).

Damage to the gastrointestinal tract. The tongue is covered with a brown coating,dry. Nausea, anorexia, pronounced flatulence. Vomiting sometimes with "coffee grounds" Abdominal pains are generalized, sometimes symptoms of an acute abdomen.

Pathogenesis of symptoms - smooth muscle paresis due to hypokalemia with stomach enlargement and intestinal paresis; sphincter spasms caused by acidosis; acute ketonemic ulcers of the gastric mucosa with gastrointestinal bleeding.

Kidney damage. Polyuria alternating with oliguria and anuria.

Skin changes. The face is pale, facial features are sharpened. The skin is dry, flabby, inelastic, with reduced turgor. Skin temperature is reduced. Mucous membranes are dry.

Eyeballs. The tone of the eyeballs is reduced, the pupils are narrowed; strabismus, converging or diverging.

Muscle damage. Hypotonia, muscles are lethargic, relaxed (a manifestation of hypokalemia); pain in the limbs - neuromyalgia.

Clinical signs of hypokalemia: pallor, muscle and general weakness, inattention, apathy, lethargy, hyporeflexia, atony of smooth muscles of the stomach and intestines, accompanied by flatulence, vomiting, intestinal obstruction.

Electrocardiographic signs of hypokalemia: tachycardia, high flattened P wave, prolongation of the P-Q interval, decrease and flattening of the T wave, decrease of the S-T segment, pathological U wave.

Electrocardiographic signs of hyperkalemia: a decrease in the P wave, a high, pointed T wave, an increase in the S-T interval.

Clinical variants of ketoacidotic coma:

1) encephalopathic - clinical manifestations of impaired cerebral circulation dominate;

2) cardiovascular - collapse, heart failure;

3) gastrointestinal - acute stomach, cholera-like syndrome;

4) renal - dysuria, changes in urine sediment.

Laboratory diagnostics

Clinical blood analysis: increased level of hemoglobin, erythrocytes, hematocrit, leukocytosis, thrombocytosis (polycythemia caused by blood clotting).

Clinical analysis of urine: glucosuria, ketonuria, increased specific gravity of urine (4 g of glucose - 0.001 units of optical density), acidic reaction of urine, proteinuria, cylindruria, microhematuria.

The level of immunoreactive insulin in the blood is significantly reduced.

Hyperglycemia up to 16-50 mmol/l.

Ketosis - an increase in the content of ketone bodies in the blood (N 0.9-1.7 mmol/l); the renal threshold for ketone bodies is 2.5-3.5 mmol/l.

Acidosis - a decrease in pH (N 7.35-7.45), a sharp decrease in the alkaline reserve to 5% (N 55%-75%), reduction of standard bicarbonates (SB) (N 20-27 mmol/l).

Hyperosmolarity is moderately pronounced or absent.

Violation of lipid metabolism due to the activation of lipolysis: increased level of NEC, hypertriglyceridemia, hypercholesterolemia.

Violation of protein metabolism due to acceleration of proteolysis: increase in creatinine, increase in the level of urea, increase in the level of residual nitrogen.

Violation of electrolyte metabolism: a possible decrease in the level of Na and Cl. The level of K before the start of insulin therapy is normal or increased due to the migration of K from the cells. After the start of insulin therapy (after 4-6 hours), late hypokalemia develops.

Fibrinolytic activity of blood is increased.

Complications of ketoacidotic coma

1. Hypoglycemic coma.
2. Edema of the brain.
3. Hypokalemic crisis.

Differential diagnosis of ketoacidosis

Causes of non-diabetic ketoacidosis:

1) alcohol intoxication;
2) massive corticosteroid therapy;
3) incessant vomiting;
4) A-transferase coenzyme deficiency.

Causes of non-diabetic acetonuria:

1) fever;
2) intoxication;
3) incessant vomiting;
4) prolonged starvation.

Treatment of ketoacidotic coma

1. Filling the insulin deficiency.

Insulin therapy is carried out only with short-acting drugs. A regime of small doses of insulin is used - a gradual decrease in the level of glycemia prevents the development of cerebral edema, relative hypoglycemia, and vascular catastrophes.

Insulin is administered hourly intravenously as a drip, sometimes as a stream. In exceptional cases, when intravenous administration is impossible, intramuscular injections are possible. To eliminate the adsorption of insulin by the elements of the system, add 7 ml of 10% albumin solution or several milliliters of the patient's blood for every 500 ml of physiological solution.

The calculation of the insulin dose is carried out every hour, based on the patient's weight - 0.1 units/kg. Insulin dose (U) = patient's weight (kg) x 0.1(U/kg). If after 2 hours there is no decrease in the level of glycemia, the dose of insulin is increased by 2 times - the dose calculation coefficient is 0.2 units/kg.

After reaching a glycemia of less than 14 mmol/l, the dose of insulin administered per hour is reduced in 2 times (dose calculation coefficient 0.05 units/kg). The infusion medium is not saline, but 5% glucose. It is possible to increase the intervals between insulin injections - every 2 hours.

When blood sugar is less than 11 mmol/L, switch to fractional insulin injections once every 3-4 hours subcutaneously or intramuscularly. Fractional administration of short-acting insulins is continued for 1-2 weeks after the patient is discharged from a ketoacidotic coma - this circumstance is connected with the fact that the daily need for insulin may decrease by 2 times, and daily correction of the dose is necessary.

2. Rehydration.

If the blood osmolarity is normal, physiological solution or Ringer's solution is administered, if the blood osmolarity is increased (more than 320 mosmol/l), a hypotonic solution of 0.45% NaCl is used as an infusion medium until the osmolarity indicator is normalized, then switched to physiological solution.

It is important to ensure the adequate severity of dehydration and restore the lost fluid - during the first 6 hours, 0.5-1.0 l/hour is administered until the signs of dehydration decrease, then the amount of fluid administered is reduced to 0.2-0.3 l/hour. The total number of infusions is about 6-10 l/day. In the presence of signs of decompensation of the cardiovascular system, the infusion dose should not exceed 2-3 l/day.

3. Correction of acid-base status.

When the pH is less than 7, a sodium bicarbonate solution of 100 mmol/hour (1 g of NaHCO3 = 12 mmol) is administered intravenously until the pH is 7 (340 ml/hour of a 2.5% solution or 200 ml of 4% solution). For every 100 meq of NaHSO3, 13-20 meq of KCl are additionally introduced - 10-15 ml of 10% solution.

An overdose of sodium bicarbonate can cause cerebral edema, severe hypokalemia and hypernatremia, alkalosis.

The necessary dose of bicarbonate is calculated according to the formula: the normal level of sodium bicarbonate in the blood (20 mmol/l or 20 mEq/l) minus the available level of bicarbonate multiplied by the volume of extracellular fluid (15-20 l).

Another formula: body weight in kg, multiplied by 0.3 and multiplied by VE (alkaline deficit). Use a factor of 0.15 and enter half the calculated dose. Administration of sodium bicarbonate can be repeated 2-3 times a day with an interval of 2 hours.

Sometimes this solution is administered in an enema, the stomach is washed or given to drink. Glutamic acid (1.5-3.0 g per day) is also used to combat acidosis and dehydration.

4. Correction of hypokalemia.

It is carried out using potassium chloride, since equivalent doses of panangin will cause hypermagnesemia (1 g of KSl contains 17 mmol).

With hypokalemia less than 3 mmol/Lv, 39 mmol/hour of KCl (3 g of KCl per hour), about 30 ml of 10% solution.

With normokalemia, prevention of late hypokalemia is carried out.

With potassium 3-4 mmol/l, 26 mmol/hour of KCl (2 g of KCl per hour), about 20 ml of a 10% solution, are administered.

At potassium level of 5-6 mmol/l, 13 mmol/hour of KCl (1 g of KCl per hour) is administered, about 10 ml 10% solution.

In case of hyperkalemia of more than 6 mmol/labo, the development of anuria stops the introduction of potassium preparations.

5. Prevention of DIC syndrome.

Heparin 5000 units intramuscularly 4 times a day or a higher dose under the control of the prothrombin index - kept at the level of 70%.

6. To improve oxidative processes, 100 mg of cocarboxylase, 5 ml of a 5% solution of ascorbic acid, 200 μg of vitamin B_{12}, 1 ml of a 5% solution of vitamin B_6 are administered intravenously.

7. In case of uncontrollable vomiting, 200-300 ml of plasma is administered 4-6 hours after the start of treatment to fill the protein deficiency and combat starvation. To avoid a hypochloremic state, 10-20 ml of 10% sodium chloride solution is administered intravenously.

8. To prevent cardiovascular insufficiency, 2 ml of cordiamine or 1-2 ml of a 20% caffeine solution are injected subcutaneously every 3-4 hours. With low blood pressure, intravenous plasma, dextran, whole blood, intramuscularly 1-2 ml of 0.5% DOXA solution are prescribed. With severe tachycardia, 0.25-0.5 ml of 0.05% strophanthin solution or 1 ml of 0.06% corglycon solution in isotonic sodium chloride solution is administered intravenously.

9. Oxygen therapy is carried out by introducing humidified oxygen through a nasal catheter at a rate of no more than 5-8 l/min.

10. If oliguria or anuria develops, diuretics are prescribed, if ineffective - hemodialysis.

11. The development of signs of cerebral edema is an indication for intravenous administration of hydrocortisone 400 mg.

12. Antibiotics and indications.

13. To restore metabolic disorders, add easily digestible carbohydrates to the diet, prescribe a low-fat diet for 10 days, drink plenty of fluids (up to 1.5-3.0 liters per day), alkaline mineral water. From the 2nd-3rd day, the diet is expanded.

Hyperosmolar (non-acidotic) coma

Hyperosmolar coma is an acute complication of diabetes caused by blood hyperosmolarity with severe intracellular dehydration in the absence of ketosis.

Hyperosmolar coma is rare - 0.23% of patients with diabetes. This complication most often develops in patients older than 50 years, with accompanying obesity or in children (adolescents). Mortality is high - 50%.

Precipitating factors:

1) sudden dehydration of the body - vomiting, diarrhea, blood loss, increased diuresis,

burns, frostbite;

2) excessive introduction of glucose solutions and saline solutions;

3) intercurrent diseases, infections;

4) surgical interventions;

5) long-term treatment with diuretics, massive doses of glucocorticosteroids, immunosuppressants.

6) hemodialysis, peritoneal dialysis.

Scheme of the pathogenesis of hyperosmolar coma Acute hyperglycemia blocks ketogenesis. A decrease in the excretory function of the kidneys with a decrease in the excretion of Na, Cl and urea in the urine: hypernatremia, hyperchloremia, hyperazotemia.

Pronounced hyperosmolarity of the blood. Dehydration is intracellular, intercellular. Hypovolemia.

Blood clotting. Thrombosis, thromboembolism

Dehydration of body tissues

Clinical manifestations of hyperosmolar coma. Hyperosmolar coma develops gradually, over several days, rarely – during days Coma precursors are polyuria, polydipsia, sometimes polyphagia. Then asthenia, signs of dehydration, drowsiness, confused consciousness join.

Blood hyperosmolarity is accompanied by a pronounced tendency to clot formation, severe microcirculation disorders in various tissues - primarily in the brain and kidneys. Clinical manifestations are due to impaired cerebral blood circulation - characteristic early and pronounced functional neurological symptoms. Thrombosis of renal vessels is the cause of acute renal failure.

The signs of tissue dehydration are especially pronounced - dryness and decrease in skin turgor, muscle hypotonia, decrease in tone of the eyeballs.

Neurological disorders. Bilateral spontaneous nystagmus, hemianopsia. Aphasia Convulsions, epileptoid attacks. Hemiparesis, paralysis. Tendon reflexes are absent, pathological reflexes appear (Babinsky, etc.). Muscle hypertonus or muscle hypotonia. Vestibular disorders. Hallucinations are possible. Disturbance of consciousness - drowsiness, stupor, sopor, coma. Hyperthermia of central genesis.

Kidney damage: early, frequent oliguria, anuria.

Changes in peripheral tissues. Sharp dryness of the skin, mucous membranes; decrease in skin turgor.

Changes in the eyes: a decrease in the tone of the eyeballs; the pupils are narrowed, react sluggishly to light.

Changes in the respiratory system: shallow, rapid breathing; no Kussmaul breath, no acetone smell.

Damage to the cardiovascular system. Tachycardia, arrhythmia. Hypotension, collapse, shock. Thrombosis of peripheral arteries and veins. Swelling of the lower limbs and scrotum is possible.

The condition of the gastrointestinal tract: vomiting, flatulence, abdominal pain; intestinal obstruction in late hypokalemia.

Laboratory diagnostics

In the clinical blood analysis, there are signs of thickening: increased hemoglobin, hematocrit,

leukocytosis

In the clinical analysis of urine: glucosuria, hyponatriuria, proteinuria, cylinduria, hematuria.

Hyperglycemia 50-200 mmol/l.

Hyperosmolarity up to 500 mosmol/l with a norm of 285-295 mosmol/l. Blood osmolality is determined by the formula: osmolality (mosmol/l)= glycemia (mmol/l)+urea (mmol/l)+2(K+Na)(mmol/l)+((protein g/l x 0.243): 8) Hyperchloremia.

Hypernatremia.

Hyperkalemia before the start of insulin treatment or normokalemia. Hypokalemia after the start of insulin therapy.

Hyperproteinemia. Hyperazotemia.

pH, level of bicarbonates, ketone bodies is normal.

Treatment of hyperosmolar coma. 1. Rehydration.

A hypotonic solution of NaCl 0.45% is injected at 2 l/hour during the first 2 hours, then 1 l/hour until normalization of blood osmolality and venous pressure. When glycemia reaches 14 mmol/l, they switch to infusion of 2.5% glucose solution. 8-12 liters of fluid are administered per day.

2. Correction of hypokalemia.

The introduction of potassium preparations is carried out only when blood glucose is reduced to 14 mmol/l. If the level of potassium in the blood is less than 3.5 mmol/l and there is no anuria, 10-20 mmol of KCl (7.5-15 ml of 10% KCl) per 1 liter of NaCl solution per hour.

3. Insulin therapy.

Insulin therapy is carried out only by intravenous drip administration of 8-12-16 units of insulin every hour until blood sugar drops below 14 mmol/l.

When glycemia reaches 14 mmol/l (250 mg%), switch to fractional insulin administration of 6-8 units every 3 hours.

4. To prevent cerebral edema and improve its metabolism, 50 ml of 1% glutamic acid is administered intravenously.

5. Oxygen therapy.

6. Prevention of thrombosis - heparin 5000-6000 IU 4 times a day intramuscularly.

7. Symptomatic therapy.

With heart failure - cordiamine, strophantin or corglukon.

In case of hypotension - 1-2 ml of 0.5% DOXA or other mineralocorticoids, plasma-substituting solutions (hemodesis, albumin, reopoliglyukin).

Antibiotics as indicated.

Hyperlactacidemic coma. Hyperlactacidemic coma is an acute complication of diabetes that develops due to the accumulation of lactic acid in the body and the occurrence of metabolic acidosis. It is rare, has a high lethality - 50%.

Etiology. 1. Old age (proneness to hypoxia).

2. Concomitant diseases with hypoxia (heart, lung damage, chronic alcoholism).

3. Acute hypoxia (traumatic, cardiogenic, toxic shock, acute infectious or inflammatory diseases, bleeding, collapse, myocardial infarction).

4. Endogenous intoxications (liver or kidney failure, leukemia, severe infections).

5. Exogenous intoxications (methanol, ethanol).

6. Pharmacogenic lactic acidosis when using biguanides, fructose, polyhydric alcohols, salicylates, sodium lactate.

Precipitating factors:

1) excessive physical exertion (lactic acid is synthesized in working muscles);

2) hypovitaminosis, especially vitamin B1;

3) magnesium deficiency;

4) acute intoxication.

Scheme of the pathogenesis of hyperlactacidemic coma Hypoxia.

Hypersecretion of catecholamines, STG, glucocorticoids.

Suppression of aerobic glycolysis. Stimulation of anaerobic glycolysis. increased production of lactic acid.

decrease in the activity of pyruvate dehydrogenase, which converts pyruvic acid (PVA) into acetyl-CoA - blockade of PVA oxidation. Accumulation of PVC with its reduction to lactic acid.

Blockade of H+ secretion by the kidneys.

Violation of the ratio of oxidized NADF and reduced NADF H2.

Violation of PVC transport in mitochondria.

Clinical manifestations of hyperlactacidemic coma

Coma can develop quickly, within several hours.

Prodromal periodinody is accompanied by dyspeptic phenomena (anorexia, nausea, vomiting), rapid breathing, angina pectoris and muscle pains (lactic acid is a strong "muscle poison"), disturbances in the psycho-emotional sphere. Lethality is high

- 50-80%, since even when the level of lactic acid rises to 7 mmol/l, irreversible changes in the tissues develop.

Damage to the central nervous system: apathy, drowsiness. Excitement, delirium, insomnia, motor restlessness are possible. Coma gradually develops.

Damage to the cardiovascular system: blockade of adrenergic receptors of peripheral vessels - severe and persistent hypotension, collapse, hypothermia. Blockade of adrenergic receptors of the myocardium - persistent bradycardia, decrease in contractility of the myocardium, development of progressive heart failure resistant to therapy. Intravascular thrombosis.

Kidney damage: oliguria, then anuria.

Damage to the gastrointestinal tract: dyspepsia, nausea, vomiting.

Muscle changes: muscle pain, muscle hypotonia.

Respiratory organs: rapid shallow breathing, then Kussmaul breathing, due to acidosis.

Laboratory diagnostics

Hyperglycemia or normoglycemia.

An increase in the level of lactic acid (normally 0.6-1.2 mmol/l; 2 mmol/l - renal

A decrease in the level of pyruvic acid (normally 0.06-0.12 mmol/l).

A sharp increase in the lactic acid/pyruvic acid index (normally 10:1).

Acidosis - a decrease in pH with a normal pH of 7.35-7.45.

A decrease in the level of standard bicarbonates (SB) at a norm of 20-27 mmol/l.

A decrease in the reserve alkalinity level at a norm of 55-75%.

Hyperkalemia. Hyperazotemia.

Treatment of hyperlactacidemic coma

1. Correction of acidosis.

When the pH is less than 7, 1-2 liters of 2.5% sodium bicarbonate solution are administered intravenously 100 mmol/hour (340 ml of 2.5% solution per hour). Administration is stopped when pH is reached

2. Stimulation of the transition of lactic acid into pyruvic acid using 1% methylene blue in the amount of 50-100 ml, 2.5 mg per 1 kg of the patient's weight.

3. Insulin therapy.

It is carried out even in the presence of normoglycemia - 6-8 units of short-acting insulin in 500 ml of 5% glucose are administered intravenously.

4. Hypotension correction - plasma substitutes and hydrocortisone 250-500 mg each.

5. Oxygen therapy.

6. Hemodialysis with indications for anuria.

CONTROL QUESTIONS

1. What is the main sign of the need to start resuscitation measures:
A. Absence of independent breathing
B. Change in skin color
C. Lack of consciousness
D. Wide pupils
E. Absence of heart sounds

2. In what situation is the Heimlich technique used:
A. Electric shock
B. Strangulation by a foreign body
C. Laryngospasm
D. Acute myocardial infarction
E. Hydrogen sulfide spraying

3. What pressure rhythm should be followed during external heart massage in adults:
A. 100 in 1 min
B. 70 in 1 min
C. 60 in 1 min
D. 40 in 1 min
E. 120 in 1 min

4. If it is impossible to administer intravenous drugs during resuscitation, adrenaline hydrochloride is most often administered:
A. In the trachea
B. In the tongue
C. Intramuscularly
D. Intrapulmonary
E. Subcutaneous

5. To restore the patency of the respiratory tract in case of apnea in the absence of injuries, you should:
A. Perform conictomy immediately
B. Press on the chest
C. Turn the patient on his side
D. Throw back the head, bring out the lower jaw, open the mouth
E. Stick out the tongue

6. In the park, a passer-by (who turned out to be a medical worker) found a 30-35-year-old man. He established that the patient has no pulse on the carotid arteries, no respiratory movements, moderately dilated pupils, and does not respond to a shout or a needle prick. What should a medical worker do first:
A. Perform cardiopulmonary resuscitation

B. Call an ambulance
C. Take the patient to an emergency hospital
D. Catheterize the subclavian vein
E. Incubate the trachea

7. A casual passer-by (medical worker) found a 30-35- year-old patient on the sidewalk. During the examination: the patient is unconscious, the pulse on the peripheral arteries is not determined, on the carotid arteries the pulse is frequent, with a weak filling, breathing is independent, 20 in 1 minute. What actions of a passerby are correct:

A. Perform cardiopulmonary resuscitation and call an ambulance
B. Call an ambulance without wasting time
C. Put the patient in a side position face down and call an ambulance
D. Conduct only indirect heart massage
E. Self-transport the patient to the medical facility

8. A 55-year-old man with a diagnosis of coronary artery disease: unstable angina fell while walking down the corridor. The general practitioner established the absence of consciousness and pulsation on a. Carotis, tones of the heart; constricted pupils and shallow breathing. The likely diagnosis?

A. Fainting
B. Asphyxia
C. Sudden stoppage of blood circulation
D. Collapse

9. The most effective urgent method of artificial lung ventilation is:
A. Rhythmic compression of the chest
B. Word-of-mouth method
C. Sylvester's method
D. The Holger-Nielsen method
E. Mishkom Ambu

10. After primary cardiac arrest, consciousness disappears due to:
A. 10-15 p
B. 2 min
C. 15-20 p
D. 1 min
E. 30 p

11. The depth of bending of the sternum to the spine in newborns during resuscitation should be:
A.2-3 cm
B. 1-2 cm
C. 3-4 cm
D. 4-5 cm
E. 5-6 cm

12. The depth of bending of the sternum to the spine of teenagers during resuscitation should be:

A.2-3 cm
B. 1-2 cm
C. 3-4 cm
D. 4-5 cm
E. 5-6 cm

13. Focal epileptic seizures include all of the following listed, except:

A. Jacksonovskii are sensitive
B. Jacksonian movements
C. Secondary generalized convulsions with aura
D. Kozhevnikov's epilepsy
E. Absences

14. What diseases can you think about if a convulsive attack occurred against the background of an increase in body temperature?

A. Epilepsy
B. Acute inflammatory diseases of the brain
C. Alcoholism
D. Acute hypertensive encephalopathy
E. Brain infarction

15. Emergency care for a generalized tonic-clonic attack:

A. Prevent further injury
B. Prevent tongue biting
C. Ensure patency of the respiratory tract
D. Sibazon 0.5% - 2 ml (fine to 6 ml) after 10 minutes before the seizure stops
E. All of the above

16. Emergency care for febrile convulsions:

A. Physical cooling methods for hyperthermia
B. Cleansing enema
C. Antipyretics - nurofen (ibuprofen) 5-10 mg/kg orally (for older 3 months), paracetamol 10-15 mg/kg, analgin 50% - 0.1 ml per year of life intravenously, but no more than 1 ml
D. Magnesium sulfate 25% in/m 0.2 ml for 1 year of life, but no more than 10 ml, sibazon 0.55 - 0.3 mg per 1 kg of weight
E. All of the above

17. While staying in a stuffy room, the patient developed nausea, blurred vision, ringing in the ears, pale face, and loss of consciousness for a minute. Previous diagnosis?

A. Fainting
B. Absence
C. Muting
D. Sopor
E. Nonconvulsive seizure

18. The patient, 20 years old, became acutely ill during classes in the sports hall. She felt a sharp "blow" to the head, quickly followed by an intense headache, nausea, repeated vomiting, and later loss of consciousness. In the neurological status: somnolent, tendon reflexes S=D, bilateral pathological reflex of Babinski, paresis in the Bare test is not determined. Pronounced symptoms: stiffness of the occipital muscles, Kernig's on both sides, Brudzinsky's. Previous diagnosis?

A. Subarachnoid hemorrhage
B. Parenchymal hemorrhage
C. Intracerebral hemorrhage
D. Migrainous stroke
E. Thromboembolic ischemic stroke

19. In a patient, 60 years old, suffering from a malignant course of arterial hypertension against the background of high blood pressure of 210/130 mm Hg. diffuse increasing headache, nausea, vomiting, impaired consciousness, generalized epileptic seizure occurred. In the neurological status: focal neurological symptoms are not determined, pronounced meningeal symptoms are determined. On the fundus: bilateral swelling of optic nerve discs. Against the background of blood pressure correction and cerebral edema, the above-described symptoms regressed after 72 hours. Previous diagnosis?

A. Acute hypertensive encephalopathy
B. Subarachnoid hemorrhage
C. Intraventricular hemorrhage
D. Epilepsy. Generalized convulsive attack
E. Cardioembolic ischemic stroke

20. A 55-year-old patient, against the background of arterial hypertension and emotional stress, developed: sudden headache, vomiting, facial hyperemia, psychomotor agitation. Within 10 minutes, consciousness disorders, central plegia of the right limbs joined. Meningeal symptom joined after 3 hours. Previous diagnosis?

A. Intracerebral hemorrhage
B. Subarachnoid hemorrhage
C. Ventricular hemorrhage
D. Ischemic cardioembolic stroke
E. Acute hypertensive encephalopathy

21. A patient who previously suffered a myocardial infarction, after emotional overstrain suddenly developed: disorders of consciousness - coma. Violation of vital functions, drop in geodynamics and breathing disorders. Objectively: the pupils are narrow, the reaction to light is weakened, tendon and pathological reflexes are not determined. Previous diagnosis?

A. Hemodynamic stroke in the brain stem
B. Cardioembolic stroke in the brain stem
C. Intracerebral hemorrhage
D. Repeated myocardial infarction
E. Cardiogenic fainting

22. A 45-year-old patient, after physical overexertion and drinking alcohol, quickly developed a disturbance of consciousness leading to a coma. Objectively: Pale skin, hyperhidrosis. Mydriasis. Blood pressure 100/70 mm Hg. Body temperature is 36.70C. Clonic convulsions, increased tendon reflexes. Determine the nature of the comma:

A. After an epileptic attack
B. Diabetic
C. Hypoglycemic
D. Stroke
E. Alcoholic

23. A 34-year-old patient, suffering from thrombophlebitis of the lower extremities, suddenly developed nausea, cough, dizziness, nausea, a feeling of distension and pain in the right subcostal area, swollen neck veins. On the electrocardiogram recorded at that moment, P-pulmonale was registered, which was not present before the attack. What is the most likely diagnosis:

A. Neurocirculatory dystonia
B. Bronchial asthma
C. Thromboembolism of the pulmonary artery
D. Myocardial infarction
E. Acute cholecystitis

24. The most effective method of quickly removing a patient from a hypoglycemic coma is the intravenous injection of 40-60 ml of 40% glucose (for 3-5 minutes) followed by a maintenance infusion of a 5-10% glucose solution (the blood plasma glucose level must be above 8.25 mmol/l, or 150mg%). In some cases, it is impossible to quickly introduce glucose to the patient (coma outside the hospital, there is no venous access). What preparation for internal, ulcer administration should be used in the following cases:

A. Glucagon
B. Adrenaline hydrochloride
C. Prednisolone
D. Estradiol
E. Nicotinamide

25. The patient, 20 years old, on the second day after the appendectomy, had a high body temperature, psychomotor agitation with transition to sopor and coma. The family doctor, who examined the patient, drew attention to pronounced tachyarrhythmia. The ECG revealed a pulsatile arrhythmia with a frequency of 220 per 1 min. For which coma is this characteristic:

A. Thyrotoxic
B. Uremychnya
C. Pechinkova
D. Hypoglycemic
E. Ketoacidotic

26. A patient, 50 years old, who is in a comatose state, in addition to other symptoms, was found to have severe hypothermia (body temperature 340C). For which coma is this characteristic?

A. Teriotoxic
B. Uremychnya
C. Hypothyroidism
D. Hypermolar
E. Ketoacidotic

27. The patient, 45 years old, suffering from ischemic heart disease, suddenly fell and fainted. At the same time, the pulse and blood pressure were not determined, there was no breathing. An electrocardiogram showed heart rhythm disturbances - ventricular fibrillation. Which of the treatment methods should be used in this situation:

A. Electric defibrillation
B. Electrical cardioversion
C. Electrical cardiac stimulation
D. Intravenous administration of strophanthin
E. Intracardiac injection of adrenaline hydrochloride

28. A 35-year-old patient developed anaphylactic shock in response to the administration of penicillin. Which of the listed drugs should be administered first:

A. Adrenaline hydrochloride
B. Prednisolone
C. Diphenhydramine
D. Famotidine
E. Saline solution

29. The depth of deflection of the sternum to the spine of adults during resuscitation should be:

A. 2-3 cm
B. 1-2 cm
C. 3-4 cm
D. 4-5 cm
E. 5-6 cm

30. What medicines can be injected into the trachea during resuscitation:

A. Adrenaline hydrochloride, calcium chloride, sodium bicarbonate
B. Adrenaline hydrochloride, lidocaine
C. Adrenaline hydrochloride, glucose, atropine sulfate
D. Adrenaline hydrochloride, calcium chloride, novocainemid
E. Adrenaline hydrochloride, lidocaine, sodium bicarbonate

31. Electric defibrillation in case of cessation of blood circulation:

A. It is used as a first measure in case of an electrocardiogram asystole
B. Should not be used without ECG control
C. Ineffective in the case of a decrease in diuresis of less than 30 ml per hour
D. It is used in case of ineffective resuscitation even in the absence ECG monitoring
E. Not to be used unless the patient is grounded

32. According to the latest recommendations of the European Council of Reanimators (ERC), the frequency ratio of external heart massage and artificial lung ventilation during resuscitation of an adult victim should be:

A.15:2
B. 18:1
C. 15:1
D. 30:2
E. 5:1

32. According to the latest recommendations of the European Council of Reanimators (ERC), the ratio of the frequency of external heart massage and artificial lung ventilation during resuscitation of children by one person should be:

A.15:2
B. 18:1
C. 15:1
D. 30:2
E. 5:1

33. According to the latest recommendations of the European Council of Reanimators (ERC), the ratio of the frequency of external heart massage and artificial lung ventilation during resuscitation of children by two people should be:

A. 15:2
B. 18:1
C. 15:1
D.30:2
E. 5:1

34. The signs listed below testify to the effectiveness of external heart massage, with the exception of:

A. Narrowing of the pupils
B. The presence of a pulse on the carotid artery
C. Regurgitation
D. Occurrences of the glossopharyngeal reflex
E. Changes in skin color

34. In the case of asphyxiation due to the ingress of a foreign body and the impossibility of removing it at the scene, it is necessary to:

A. Throw the head of the victim, bring the lower jaw up
B. Insert the duct
C. Perform vest resuscitation
D. Use a laryngeal mask
E. Perform a conictomy

35. Sudden death is most often caused by:

A. Fibrillation of the ventricles of the heart
B. Ventricular tachycardia
C. Electromechanical dissociation
D. Asystole

E. Atrioventricular block

36. The least threatening heart rhythm disorder:
A. Polytopic extrasystole
B. Supraventricular single extrasystole
C. Frequent ventricular extrasystole
D. Group extrasystole
E. Extrasystoles of type R on T

37. The patient suddenly fainted on the way home. The skin is bluish in color, the pulse is not determined, blood pressure is 95/70 mm Hg, the heart sounds are weakened. Breathing is vesicular. There are no swellings. On the electrocardiogram - complete dissociation of P waves and QRS complexes, heart rate - 30 in 1 min. What is the next strategy for the patient:
A. Hospitalization to the cardiology department
B. Outpatient treatment
C. Hospitalization to the intensive care unit
D. Treatment in a day hospital
E. Hospitalization to pools of the monologic department

38. The patient suddenly fainted, convulsions appeared. The skin is bluish, pulse and blood pressure are not determined. There is no breathing, heart sounds are not heard. On the electrocardiogram: a large number of waves that resemble the ventricular complex, different in shape and amplitude; the RR interval is almost absent. What emergency measures should be taken as a matter of priority:
A. Artificial pacemaker
B. Electrocardiostimulation
C. Electropulse therapy
D. Cardiac defibrillation
E. Introduction of cordarone

39. The patient suddenly fainted, convulsions appeared. Skin covers bruises, pulse and blood pressure are not determined. There is no breathing, no heart sounds are listened to on the electrocardiogram: a large number of waves that resemble the ventricles complex, different in shape, amplitude; the RR interval is almost absent. Which one rhythm disturbance occurred in the patient:
A. Ventricular fibrillation
B. Paroxysmal ventricular tachycardia
C. Paroxysmal atrial fibrillation
D. Paroxysmal supraventricular tachycardia
E. Frederick's syndrome

40. The emergency medical team was called to a 7-year-old boy. Consciousness and breathing are absent, pulse on the carotid artery and blood pressure are not determined, the pupils are wide and do not react to light. After releasing the airways, carrying out artificial respiration and restoring blood circulation, it is necessary to introduce:
A. Calcium chloride

B. Atropine sulfate
C. Adrenaline hydrochloride
D. Sodium bicarbonate
E. Glucose solution

41. The emergency medical team was called in the summer to a 10-year-old girl who was taken out of the river in a state of unconsciousness. Objectively: the skin is pale, there is no spontaneous breathing, the peripheral pulse on the main vessels is not determined, the pupils are dilated. Which of the following resuscitation measures should be used as a priority:
A. Release of airways
B. Administration of calcium chloride
C. External heart massage
D. Cardiac defibrillation
E. Administration of adrenaline hydrochloride

42. An eleven-month-old boy suddenly fell, his breathing and heartbeat stopped, convulsive movements appeared, accompanied by urination and defecation. Objectively: lack of consciousness, breathing and heart rate, pupil reaction to light, corneal and conjunctival reflexes. What is the most likely diagnosis:
A. Meningoencephalitis
B. Epileptic condition
C. Sudden death syndrome
D. Diabetic coma
E. Cardiogenic shock

43. The ambulance team arrived 15 minutes after the call. Two paramedics perform cardiopulmonary resuscitation on the patient: artificial ventilation of the lungs and indirect heart massage. During the initial examination of the patient: the pupils are moderately dilated, the pulse on the carotid arteries is not determined, there are no respiratory movements; on the electrocardiogram - ventricular fibrillation. What measures should be carried out as a matter of priority:
A. Incubate the trachea
B. Administer adrenaline hydrochloride intravenously
C. Administer intravenous lidocaine
D. Administer β-blockers intravenously
E. Defibrillation

44. A 55-year-old man with a diagnosis of coronary heart disease: unstable angina fell while walking in the corridor. The general practitioner established the absence of consciousness and pulsation on a. Carotis, tones of the heart; constricted pupils and shallow breathing. What should resuscitation measures be started in this case?
A. From carrying out transesophageal cardiostimulation
B. Intravenous administration of epinephrine
C. Intracardiac administration of atropine
D. From inflicting a precardial shock

E. Carrying out artificial ventilation of the lungs

45. A condition in which thinking and speech are slowed down, insufficient perception and assessment of what is happening, reduced attention, sharp exhaustion, drowsiness:

A. Clear consciousness
B. Muting
C. Sopor
D. Coma
E. Syncope

46. A condition in which mental activity is sharply suppressed, upon repeated application, patients open their eyes, but contact with them is not possible:

A. Clear consciousness
B. Muting
C. Sopor
D. Coma
E. Syncope

47. A condition in which there is a complete loss of consciousness, there are no reactions to various external stimuli:

A. Clear consciousness
B. Muting
C. Sopor
D. Coma
E. Syncope

48. Short-term loss of consciousness, which is accompanied by a loss of postural tone and is caused by a short-term decrease in blood supply to the brain:

A. Clear consciousness
B. Muting
C. Sopor
D. Coma
E. Fainting (Syncope)

49. One of the most important indicators of coma severity:

A. Complete loss of consciousness
B. Lack of reaction to external stimuli
C. Bilateral fixed mydriasis
D. Absence of reflexes
E. Decreased muscle tone

50. Types of syncopal states:

A. Neurogenic
B. Orthostatic
C. Cardiogenic
D. Cerebrovascular
E. All of the above

51. An attack that begins with turning the head, loss of consciousness, the

patient falling, followed by tonic and clonic phases and ending in coma, followed by sleep, or muting or psychomotor excitement:

A. Absence
B. Fainting
C. Generalized tonic-clonic seizure
D. Myoclonic seizure
E. Focal attack

52. A condition in which the patient suddenly loses consciousness for a short time for a few minutes and is accompanied by freezing of the patient:

A. Absence
B. Fainting
C. Atonic attack
D. Myoclonic seizure
E. Focal attack

53. A generalized convulsive attack occurs when:
A. Epilepsies
B. Alcohol abstinence
C. Fever, infectious diseases of the brain
D. Metabolic disorders
E. All of the above

54. In which metabolic disorders is a comatose state observed:
A. Uremia
B. Diabetes
C. Hypoglycemia
D. Hepatic coma
E. All of the above

55. The most frequent reasons for the development of asthmatic status are:
A. infections
B. sharp withdrawal of GCS and cromons
C. overdose of b2 -agonists of short action
D. significant contact with allergens
E. all of the above

56. Status asthmaticus is a severe attack of bronchial asthma that lasts longer than:
A. 20 hours
B. 24 hours
C. 10 hours
D. 15 hours
E. 15 minutes

57. Which of the listed drugs are used as basic therapy for BA?
A. Euphilin
B. Atropine
C. Propranolol

D. Seretide
E. Salbutamol

58. Which of the listed means for the treatment of bronchial asthma are classified as anti-inflammatory drugs:
A. Serevent
B. Teopek
C. Salbutamol
D. Fluticasone
E. Atrovent

59. What mechanisms of broncho-obstruction belong to the reverse:
A. Hypercrinia, Dyskrynia
B. CHD
C. Bronchospasm
D. Emphysema of the lungs
E. Sclerosis of the bronchial wall

60. Which mechanisms of bronchial obstruction are irreversible:
A. Hypercrinia
B. Dyscrinia
C. Bronchospasm
D. Emphysema of the lungs
E. Sclerosis of veins

61. The main cellular elements of inflammation in bronchial asthma are:
A. Eosinophils
B. Striated cells
C. T-lymphocytes
D. Macrophages
E. All answers are correct

62. 1. Factors capable of causing ketoacidotic coma do not include:
A. A sharp decrease in the need for insulin
B. A sharp increase in the need for insulin
C. Late diagnosis of diabetes
D. Inadequate treatment of diabetes
E. Fatty infiltration of the liver

63. 3. In blood serum with ketoacidotic coma, all the specified changes are noted, with the exception of:
A. Hyperglycemia up to 16-50 mmol/l
B. Ketosis
C. Acidosis
D. Violation of electrolyte metabolism
E. Alkalosis

64. 4. In the clinical analysis of urine in hyperglycemic coma, all the specified

changes are noted, except:

A. Glucosuria
B. Ketonuria
C. Decrease in urine density
D. Acid reaction of urine
E. Microhematuria

65. 6. The main clinical manifestations of hyperosmolar coma include all of the following, except:

A. Dry skin and mucous membranes
B. Epileptiform seizures
C. Kussmaul's breathing
D. Oliguria
E. Acute hypotension

66. 7. The treatment program for hyperosmolar coma includes the following measures, in addition to:

A. Insulin therapy
B. Rehydration
C. Prevention of thrombosis
D. Correction of hyperkalemia
E. Prevention of cerebral edema

67. 8. The treatment program for ketoacidotic coma includes the following measures, in addition to:

A. Intravenous infusion of small doses of insulin
B. Increased rehydration of the body
C. Restoration of normal acid-alkaline balance
D. Normalization of the activity of the cardiovascular system
E. Fights against hyperkalemia

68. 11. The most likely cause of persistent tachycardia in patients with type 1 diabetes is:

A. Combination of diabetes mellitus and thyrotoxicosis
V. Diabetic cardiomyopathy
C. Ischemic heart disease
D. Autonomic cardiac neuropathy
E. Hypokalemia

SITUATION TASKS

1. A 25 years old man developed an epileptic seizure during a doctor's appointment at the family clinic. Then, within 15 minutes, four generalized convulsive attacks were recorded, in the intervals between which consciousness did not return to the patient. What drug should be administered to the patient for emergency care?

Answer: sibazon.

2. The patient, 28 years old, has been suffering from epilepsy for a long time. As a result of the violation of the treatment regime, he had frequent convulsive attacks, in the intervals between which the patient was unconscious. It does not react to painful stimuli. The pupils are narrow, the reaction to light is weak. Muscle tone is reduced. With which drug should emergency care be started?

Answer: from sibazon.

3. A 19 years old patient has had seizures lasting up to 3 minutes since childhood. with loss of consciousness. For the past 2 years, he has been constantly using anticonvulsant drugs against which seizures have not been observed. She turned to a general practitioner about SARS, temp. body up to 38.50C. At the reception, she fainted with generalized convulsions lasting 2-3 minutes, after which she fell asleep. Define 1) the patient's psychopathological condition; 2) what trigger factors provoked the attack?

Answer: 1) epileptic attack;

2) febrile body temperature, intoxication.

4. A 30 years old woman with diabetes suddenly fainted. During the examination: the skin is moist, convulsions, the pupils are dilated, the pulse and blood pressure are normal. The level of glucose in the blood is 1.2 mmol/l. What immediate measures should be taken?

Answer: intravenous injection of 40% glucose solution.

5. A 54 years old woman, while in a crowded bus, felt short of breath, weakness in her lower limbs, and nausea. After 5 minutes, she fainted. Objectively: the skin is pale, covered with cold sweat. Blood pressure - 100/40 mm Hg. art., the pulse on the radial artery is rhythmic, 100 per 1 min. What is the most likely cause of this condition?

Answer: acute insufficiency of blood circulation of the brain.

6. A 65 years old man, being in a poorly ventilated room, felt a lack of air, heaviness during breathing, weakness in the lower limbs, and nausea. After 10 minutes, I fainted. About: the skin is pale, covered with cold sweat. Blood pressure - 100/40 mm Hg. art., the pulse on the radial artery is rhythmic, 100 per 1 min. What indicator can confirm a violation of cerebral blood flow?

Answer: reduction of oxygen transport to the brain.

7. A girl, 16 years old, fainted after a long stay in a vertical position in the sun during the ceremonial parade. Objectively: the skin is pale, the heart rate is 96 in 1 minute. Blood pressure - 70/50 mm Hg. art., heart tones are sonorous. Breathing is vesicular. Where should emergency care begin?

Answer: to ensure a horizontal position with raised lower limbs.

8. A 13 years old girl complained of dizziness after standing in the sun for a long time, fainted, fell, blood pressure - 80/40 mm Hg. st., pulse - 100 per 1 min, weak filling, shallow breathing, pale face. After 4 minutes, the symptoms regressed. What is the most likely diagnosis in this case?

Answer: simple neurogenic syncope.

9. A 13 years old girl with a history of septic conditions complained of dizziness, fainted, and fell during blood sampling. Blood pressure - 80/40 mm Hg. st., pulse - 100 per 1 min, weak filling, shallow breathing, pale face covered with sweat. What emergency measures should be taken?

Answer: put the patient on her back with her head down, ensure air access.

10. A girl, 18 years old, has been suffering from diabetes for 7 years. Receives insulin - 42 units per day. She suddenly fainted. Moist skin, spasms of the muscles of the upper and lower limbs. Pulse 86 per 1 min, rhythmic. Blood pressure – 105/60 mm Hg. Art. With which drug should emergency care be started?

Answer: 40% glucose solution.

11. A 33 years old patient was diagnosed with a paroxysm of supraventricular tachycardia. Immediately after the rapid intravenous administration of 10 ml of 10% solution of novocaine amide, the patient developed general weakness, darkening of the eyes, cold sweat, and she fainted. About: pale skin, blood pressure - 60/40 mm Hg. Art., pulse 120 per 1 min, weak filling, muffled heart sounds. What complication did the patient have?

Answer: collapse.

12. In a 10-month-old baby. psychomotor development corresponds to age. He has been sick for a day, the body temperature is 38.60C, there are manifestations of nasopharyngitis. With an increase in body temperature to 39.20C, motor restlessness, twitching of the facial muscles with subsequent transition to the muscles of the limbs appeared. Formulate a preliminary diagnosis.

Answer: acute respiratory viral infection, convulsions provoked by an increase in body temperature

13. In an 8 months old baby. the body temperature has risen to 38.90C, there is restlessness, repeated regurgitation unrelated to feeding. The large crown of the head is tense, the stiffness of the cervical and occipital muscles is noted. During the examination, clonic-tonic convulsions occurred. What is the most likely cause of seizures?

Answer: meningitis.

14. The patient, 35 years old, was brought to the outpatient clinic in serious condition with complaints of sharp pain behind the sternum, general weakness. The

persons accompanying the patient reported that he felt sick in the tram (the man was going to work). He turned pale, covered himself with a cold sweat, complained of a sharp pain in the area of the heart. At the stop, he was taken out of the tram and taken to the outpatient clinic. The doctor put the patient on the couch and began, taking his time, to count the pulse and measure blood pressure. The patient has a normal body weight. Before that, I was not sick with anything. Objectively: the skin is pale, moist, covered with droplets of sweat, pulse - 100 in 1 minute, weak filling and tension, blood pressure - 80/40 mm Hg. Art. The doctor had not yet finished the examination when the patient fainted. Pulse and blood pressure were not determined. The dilated pupils did not react to light. Individual respiratory movements were observed. Cyanosis rapidly increased. The registered ECG in the I standard lead had the appearance of a wavy line. The doctor and assistants started resuscitation measures: external heart massage, artificial respiration, intravenous injection of 6 ml of 2% lidocaine solution and 10 mg of panangin. The patient died without regaining consciousness. No pathology was detected during the solution, no focal changes were observed in the myocardium.

- Based on the clinical data and autopsy results, what do you think is the final diagnosis and immediate cause of death.

- Were mistakes made while providing assistance to the patient? If there were, then specify them.

- how should a doctor act in a similar situation?

Answer:

- CHD: acute coronary insufficiency, ventricular fibrillation.

- The doctor made 2 mistakes: a) did not initiate urgent measures to eliminate the pain syndrome; b) when ventricular fibrillation occurs began to administer drugs intravenously, which in the absence of hemodynamics has no therapeutic effect.

- In similar situations, the doctor should: a) urgently eliminate the pain, put the patient to sleep, give nitroglycerin, then, if there is no effect, introduce fentanyl with droperidol or promedol; b) after eliminating the pain attack, perform an ECG, complete the examination, urgently hospitalize the patient to the cardiology department; c) with the development of ventricular fibrillation, perform electrical defibrillation, after restoring the heart rhythm, intravenously inject sodium bicarbonate, lidocaine, potassium-glucose-insulin mixture; d) in the absence of an electric defibrillator, continue indirect heart massage and artificial respiration and at the same time intracardially inject 80-120 mg of lidocaine, 5-10 ml of panangin (or 5-10 ml of 10% novocainamide), with the restoration of cardiac activity, implement point b.

15. A 59 years old patient, a state farm worker, was brought to the outpatient clinic 4 hours after the occurrence of a transmural myocardial infarction in the area of the front wall of the left ventricle and the interventricular septum. At the time of the examination by the family doctor, there is no pain in the area of the heart. Cardiac activity is rhythmic, pulse 40 in 1 minute, heart sounds are dull, blood pressure - 120/60 mm Hg. The patient began to urgently attach electrodes for connection to the electrocardiograph, at which time he suddenly turned around and fainted. Pulse and blood pressure were not determined. Cyanosis of the face and neck rapidly increased.

Movement restlessness, convulsive contraction of the limbs, and convergence of the eyeballs were observed. Breathing stopped. A straight line on the ECG.

- What happened to the patient?
- What urgent measures are necessary to save the patient?
- What is the most effective way to prevent development the course of such a complication?

Answer:

- The patient has a complete atrioventricular block and a Morganhi-Adams-Stokes attack (hypokinetic form – asystole)
- Indirect heart massage, artificial respiration, with resumption of heart activity - intravenous administration of isadrine, atropine sulfate, prednisolone, cordiamine, sodium bicarbonate.
- Electrocardiostimulation (electrode into the right ventricle)

16. A 60 years old patient turned to the family doctor with complaints of sharp pain in the epigastric region. I fell ill 2 weeks ago, when I returned home after work and suddenly felt a sharp pain in the epigastrium, fainted. He regained consciousness, lying on the floor, the pain continued. Subsequently, the pain in the epigastric area occurred every day, but was much weaker and passed on its own after 30-40 minutes; 2-3 times during the week, the pain attacks had a protracted nature. Since the pain occurred almost every day, the patient sought help at the outpatient clinic. Smokes about 20 cigarettes a day, has been suffering from chronic bronchitis for many years. During the examination: there is no pain, there are pronounced signs of emphysema of the lungs. Heart sounds are muffled, 5-6 extrasystoles in 1 minute. The pulse is arrhythmic, 100 in 1 minute, intense. Blood pressure - 180/100 mm Hg. The abdomen is soft, painless during palpation, no deviations from the norm on the part of the organs of the abdominal cavity were detected. On the ECG: ST interval is arcuately raised, negative T wave in leads III, II, aVF.

- What is the most likely diagnosis in this case.
- What should be your tactics.
- Which of the following indicators are most likely to be changed during the examination: a) the number of leukocytes, b) ESR, c) the content of AsAT, AlAT, CFC, LDH?

Answer:

- CHD: infarction in the area of the back wall of the left ventricle in the scarring phase. Hypertensive disease, CHF. Chronic bronchitis of a smoker, pneumosclerosis, emphysema of the lungs. Chronic gastritis.
- Hospitalization to the cardiology department.
- All of them

17. A 30 years old patient complains of muscle weakness, paresthesias, unbearable headache, cramps in the muscles of the upper limbs, thirst (drinks up to 3 liters of water per day). He has been sick for 2 years. During the examination: the condition is satisfactory, the respiratory organs are without peculiarities. The borders of the heart are shifted to the left by 2 cm. Heart sounds are clear. Accent II tone over the aorta, over the top of the heart, a systolic murmur is heard. Pulse - 70 per 1 min,

BP-210/140 mm Hg. Art. The stomach is soft, not bloated. The liver and spleen are not palpable. The genitourinary system is unremarkable. ECG data: horizontal position of the heart axis, single (polytropic) ventricular extrasystoles, T wave in I and left thoracic leads is negative, unequal, widened. Examination of the fundus: the veins are dilated, the arteries are unevenly narrowed. X-ray of the skull: no pathological changes were detected. An overview picture of the urinary system: the right kidney is deformed due to the formation lying above it. Blood tests: Hb-120 g/l, leukocytes-6.0x109 in 1 l, eosinophils-2, rod-nuclear granulocytes-4, segmental granulocytes-78, lymphocytes-7, monocytes-9, ESR-12 mm/h, K+ blood plasma – 3 mmol/l, Ca+ - 3.5 mmol/l. Urine examination: 17-ketosteroids - 25.3 μmol per day, aldosterone - 140 nmol per day.

- What is the most likely diagnosis in this case?
- What additional diagnostic studies can confirm your diagnosis?
- What should be the medical tactics?

Answer:

- Aldesteroma of the right adrenal gland (Kohn's syndrome). Symptomatic endocrine arterial hypertension.
- Ultrasound and CT scan of the adrenal glands.
- Surgical treatment.

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