**MINISTRY OF HEALTH OF UKRAINE**
**ODESA NATIONAL MEDICAL UNIVERSITY**

Faculty of Medicine No. 1

Department of simulation medical technologies

**CONFIRMED by**
Vice-rector for scientific and pedagogical work

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# METHODICAL RECOMENDATION FOR PRACTICE

# «SIMULATION MEDICINE»

Faculty, course: International, 6 year

Educational Discipline: Simulation medicine

**Approved:**

The methodical recommendation was approved at the meeting of the department of simulation medical technologies

Protocol No. 1 of 28.08.2023

Head of the department _____ Oleksandr ROGACHEVSKYI

*(signature)*

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## PRACTICAL TRAINING

*Practical lessons No. 8 — 10*

**Topic:** Emergency conditions in children of different ages. Simulation training .

**Purpose:** To form, master and practice professional skills in providing emergency care for the most common emergency conditions in children of all ages .

To learn the ability to independently use knowledge and skills in the diagnosis and treatment of the most common emergency conditions in children of various ages .

C to form a clear idea of the sequence of actions in the algorithm of providing emergency aid for the most common emergency conditions in children of various ages .

To form the competence of professional communication in the team when providing emergency aid to children of different ages .

**Basic concepts:** Diagnosis and assistance in emergency situations: Convulsions, Anaphylaxis, Diabetic coma, asthma attack.

**Equipment:** Pediatric HAL S3005 , oxygen cylinder, computer with monitor and software from a mannequin, mixing console, single-channel wireless system receiver, headset microphone, gooseneck microphone, single-channel wireless system receiver, broadcast video camera, projector (large screen), Ambu bag, pulse oximeter, functional bed, tripod, glucometer, equipment for neurological examination, peak flow meter, latex gloves, medical masks, syringes, solutions for injections.

**Plan:**

1 Organizational activities (greetings, verification of those present, announcement of the topic, purpose of the lesson, motivation of higher education seekers to study the topic).

2 Control of the reference level of knowledge (frontal survey) :

requirements for students' theoretical readiness to perform practical classes (know nosology and their treatment protocols);

questions (clinical situations) to check basic knowledge on the subject of the lesson:

For the following patient conditions (meningococcal infection, enterovirus infection, resuscitation of newborns ), know:

1. Differential diagnosis.
2. Examination.
3. Making a preliminary diagnosis.
4. Providing emergency care according to protocols.
5. Adherence to the algorithm of actions.

3 Formation of professional skills and abilities:

mastering skills:

1. Quick recognition of an emergency in a patient.
2. Be able to quickly give and receive commands to medical personnel depending on the critical situation (teamwork).
3. To be able to quickly carry out a differential diagnosis of an emergency.
4. Examination (physical methods, measurement of blood pressure, blood pressure, heart rate, SpO2, thermometry, capnometry-graphy, glucometry, peak flowmetry, plan laboratory and instrumental studies).
5. Determination of the treatment scheme based on theoretical knowledge of protocols obtained at previous departments.

6 Assistance (introduction of IV injections and a catheter, oxygen supply, use of a functional bed, ECG recording and interpretation).
7 Communication skills with staff and relatives in an emergency patient situation.

task content:

For each topic nosology:

1 Briefing.
2 Conducting a clinical simulation scenario.
3 Debriefing.

recommendations (instructions) for performing tasks:

1 It is mandatory to have theoretical knowledge on the topic obtained while attending classes at previous departments.
2 Acquaintance with the methodical recommendations of the department before the class.
3 Completion of the elective course of the department of simulation medical technologies "Professional communication skills in extreme situations".

requirements for work results and control materials for the final stage of the lesson:
Passing a clinical scenario with a positive result for a simulated patient.

Meningococcal infection is an acute infectious disease caused by meningococcus, transmitted by airborne droplets and characterized by nasopharyngitis, meningococcemia, purulent meningitis, which occur separately, sequentially or simultaneously, meningococcal infection often leads to bacteremia. Meningococcal infection is widespread worldwide. According to the WHO, it is registered in 200 countries.

The causative agent of the disease is Neisseria meningitidis , there are nine different serotypes of meningococcus (A, B, C, X, etc.). It is a gram-negative diplococci, has the shape of a coffee bean, and in typical cases is placed in pairs inside neutrophilic granulocytes. The causative agent is not resistant to environmental factors, quickly dies when cooled, therefore the material for research should be delivered in a water bath at a temperature of 37° C. The source of infection is patients and carriers of bacteria. The mechanism of transmission of meningococcal disease is airborne. Susceptibility of people to meningococcus is quite significant. Meningococcal disease is characterized by a winter-spring seasonality.

The nasopharynx is the site of primary localization and reproduction of meningococci. In most cases, the presence of the pathogen on the mucous membrane of the nasopharynx is not accompanied by noticeable changes and ends with a healthy carrier or nasopharyngitis. In cases of spread of meningococcus in the body (the main route is hematogenous), generalized forms of the disease develop. Bacteremia is accompanied by massive decay of meningococci - toxemia, which leads to damage to the endothelium of vessels - meningococcemia. Meningococcemia also affects the vascular membrane of the eyes, the synovial membrane of the joints, possible damage to the adrenal glands, kidneys, and endocardium. In cases of penetration of the causative agent through the blood-brain barrier, there is irritation of the receptors of the soft brain membrane and CSF-forming systems by toxic factors with the development of the inflammatory process and increased secretion of cerebrospinal fluid, swelling and swelling of the brain, and over-irritation of the membranes of the brain and the roots of cranial and spinal nerves. In the pathogenesis of particularly severe forms of meningococcemia, the leading role is played by infectious-toxic shock, which manifests itself as acute vascular collapse against the background of severe intoxication. Toxemia leads to hemodynamic disorders and disruption of microcirculation in organs and tissues, to disseminated intravascular coagulation (DVZ - syndrome), significant disruption of metabolic processes, water-electrolyte balance, function of endocrine glands. Progressive infectious-toxic shock can cause severe damage to the adrenal glands and kidneys (kidney shock) with the subsequent development

of their acute insufficiency. Vascular and degenerative changes in parenchymal organs are also possible - septic spleen, glomerulonephritis, endocarditis, signs of damage to the brain and spinal cord and their membranes may not be present.

**Clinic.** The incubation period lasts 1-10 days, most often 5-7. According to the accepted classification by V.I. Pokrovsky, the following are distinguished: 1) localized forms (meningococcal disease and acute nasopharyngitis), 2) generalized forms (meningitis, typical and fulminant meningococcemia, meningoencephalitis, mixed form), 3) rare forms (arthritis, endocarditis, pneumonia, iridocyclitis, etc.).

Meningococcal disease is not accompanied by clinical manifestations and is detected during contact examination.

Acute nasopharyngitis. The most common form. It is characterized by headache, nasal congestion, sore throat, mucous or purulent-mucous discharge from the nose, low-grade fever, bright hyperemia and swelling of the back wall of the pharynx. Inflammatory changes are observed for 5-7 days. In 30-50% of patients, nasopharyngitis precedes the development of a generalized form of the disease.

Meningitis can develop after nasopharyngitis or the first signs of the disease appear suddenly, in the midst of complete health. The clinical picture is characterized by a pathognomonic triad of initial symptoms: fever, headache, vomiting. In the presence of such signs, the doctor should always check for meningeal symptoms. The body temperature rises suddenly with chills, it can reach 40-41°C in a few hours. Headache without certain localization, severe, aggravated by bright light, sharp sounds. Vomiting occurs suddenly, without nausea, in a fountain. General hyperesthesia is often observed. Tonic, clonic or mixed convulsions are possible, in young children the appearance of convulsions may be the first sign of meningitis. During an objective examination, meningeal symptoms are revealed, which appear on the first day of the disease and progress rapidly. The most constant are the stiffness of the muscles of the back of the head, symptoms of Kernig and Brudzinski (upper, middle, lower). Infants have a parietal triad: bulging, tension of the parietal, lack of its normal pulsation. The characteristic posture of the patient is that he lies on his side with his head thrown back and his legs pulled up to his stomach (the pose of a folded chicken or a lying dog). The severity of the meningeal syndrome may not correspond to the severity of the disease. Sometimes, especially in young children, there is a dissociated meningeal syndrome, when some leading symptoms are absent. With inflammation of the meninges, so-called local symptoms are possible, which are caused by the spread of the inflammatory process at the root of the cranial and spinal nerves, the substance of the brain. In some patients, pathological reflexes of Babinski, Oppenheim, Rossolimo, etc., decrease or disappearance of spinal reflexes, strabismus, ptosis, paresis of facial muscles, paresis and paralysis of limbs, etc. can be detected. The autonomic nervous system is often affected (persistent red dermographism). There can also be disorders of consciousness: sopor, which turns into a deep coma from the first hours of the disease, is an unfavorable prognostic sign. Herpes simplex is activated, a characteristic rash appears on the wings of the nose, the mucous membrane of the lips. During the blood test - neutrophilic leukocytosis, increased ESR.

Meningococcemia is characterized by a violent onset. The temperature rises to 39-41°C and is kept at a high level for 2-3 days. At the same time as the fever, a headache, sudden general weakness, pallor or cyanosis of the face and limbs appear. Blood pressure rises at the beginning, in case of shock it can decrease sharply (to collapse). A constant sign of meningococcemia is exanthema. A skin rash appears after 5 — 15 hours, sometimes - on the 2nd day after the onset of the disease. The most typical rash is a hemorrhagic, irregularly shaped, star-shaped rash with clear contours. The elements have different sizes - from speckled petechiae to significant hemorrhages. As a result of dusting, a rash of various colors and brightness, often with a typical, barely noticeable grayish (steel) shade, necrotization of deep and large hemorrhages, the formation of defects that do not heal for a long time, with subsequent scarring. Cases of necrosis of the auricles, the tip of the nose, and the terminal phalanges of the fingers are possible. The most typical locations of the rash are the buttocks, back surfaces of the thighs, eyelids, sclera, and auricles. Hemorrhagic rash is often combined with roseolous, papular, roseolous-petechial elements. Histologically, the exanthema is a

leukocyte-fibrinous thromboembolism (clots or emboli contain meningococcus). Skin rash with meningococcemia is a metastatic foci of infection. Joints (arthritis and polyarthritis), as well as the choroid of the eye (iritis, iridocyclitis, uveitis) occupy the second place in the localization of metastases. This sometimes leads to the development of a characteristic triad - hemorrhagic rash, arthritis, iritis. A blood test reveals moderate or significant neutrophilic leukocytosis with a shift of the leukocyte formula to the left towards young and myelocytes, an increase in ESR, often thrombocytopenia.

Among the complications of meningococcal meningitis, swelling and edema of the brain and infectious-toxic shock are the main ones. Clinically, cerebral edema is characterized by symptoms of rapidly increasing intracranial hypertension: intense headache, vomiting fountain, weakening of vision. The color of the face is crimson-bluish. Convulsions, excitement, and causeless hiccups are often observed. Acute shortness of breath and breathing disorders according to the Cheyne-Stokes type. Brain swelling usually develops on the first day of illness.

The main symptoms of clinical diagnosis in meningococcal meningitis are acute onset, fever, headache, vomiting, the presence of meningeal syndrome (general hypertension, stiffness of the muscles of the back of the head, symptoms of Brudzinski, Kernig, etc.), in infants - bulging of the parietal lobe , neutrophilic pleocytosis in cerebrospinal fluid. With meningococcemia - violent onset, fever, typical hemorrhagic-necrotic stellate rash, sometimes - damage to joints and choroid eye

**Clinical diagnostic criteria for meningococcemia:**

– sudden, acute onset with an increase in body temperature to 38-40°C;

– pronounced intoxication syndrome: general weakness, headache, muscle pain, pale skin;

– in most patients, after a few hours, a spotty-papular rash appears on the skin without a certain localization. After a few more hours, hemorrhagic elements of the rash, ranging in size from 1-2 mm to several centimeters, form on the skin of the buttocks, thighs, lower legs, and the lower part of the body. Over time, necrosis forms in the center of the largest elements of the rash;

– hemorrhages in the sclera, mucous membranes of the oropharynx, nose, and stomach bleeding may be observed;

– in lightning forms - manifestations of infectious-toxic shock rapidly increase, hypostatic bluish spots are formed on the body.

The rapid spread of the rash, its localization on the face and mucous membranes, significant hyperpyrexia (above 40°C), the absence of meningeal symptoms, the rapid progression of consciousness disorders, the presence of hypostases, hemorrhagic syndrome, and shock are considered to be clinical signs of an unfavorable prognosis in case of meningococcal infection. The absence of leukocytosis (the number of leukocytes in the blood below 10 x 10 9/l) or leukopenia, thrombocytopenia (below 100x106/l) and a normal or reduced erythrocyte sedimentation rate (below 10 mm/h) are considered hematological signs of an unfavorable prognosis in MI. A low level of C-reactive protein is considered a modern laboratory criterion for an unfavorable prognosis in MI. An instrumental criterion for an adverse course of MI is a low left ventricular PV (30-40% or less).

Laboratory diagnosis of meningococcal infection is the detection of meningococcus in cerebrospinal fluid, nasopharyngeal mucus and blood. The bacterioscopic method of detecting meningococci in the sediment of cerebrospinal fluid and a thick drop of blood is of auxiliary importance. The general clinical method (pressure, transparency, circles, cellular composition), bacterioscopic and bacteriological methods and biochemical research (the amount of protein and sugar) are used for the study of cerebrospinal fluid. With meningococcal meningitis, the pressure of the cerebrospinal fluid is increased, it is cloudy and purulent. There are thousands of cells in 1 μl, of which 70-80% are neutrophil granulocytes. The amount of protein is from 1 to 7 g/l, the Pandey globulin reaction is positive.

Meningeal syndrome can be observed with other meningitis of viral and bacterial etiology. The main criteria for meningococcal and secondary purulent meningitis, which includes pneumococcal, streptococcal, and staphylococcal, are listed in Table 1.

Table 1. Diagnostic criteria of meningococcal and secondary purulent meningitis

| Criterion | Meningococcal meningitis | Secondary purulent |
|---|---|---|
| History of life | | Skull injury, otitis, exacerbation of rhinitis, etc. |
| Medical history | Nasopharyngitis | Exacerbation of otitis, sinusitis, pneumonia, febrile illness before meningitis, etc. |
| The beginning of the disease | Acute | Acute or gradual |
| Chills | Frequent | Rarely with otogenic meningitis |
| Fever | Permanent | Septic or permanent |
| Herpetic rash | Often (85-87%) | Rarely (2 - 4%) |
| Rash on the skin | Often hemorrhagic (40%) | There is none |
| Clinical data | There are no concomitant diseases, in 5 | Otitis, sinusitis, pneumonia, |
| | - 10% arthritis | other purulent foci |
| Peripheral paresis | Rarely | Relatively often |
| nerves | | |
| Nature | Cloudy, whitish or | Cloudy, sometimes with yellow or |
| cerebrospinal | yellowish | greenish tint |
| liquid | | |
| Bacteriological | Meningococcus | Pneumococcus, staphylococcus, |
| research | | hemophilic bacilli, other pathogens |

**Treatment.** Therapeutic tactics depend on the clinical form, the severity of the course and the presence of complications.

Hospitalization of a patient with MI in a specialized infectious hospital (city, regional children's infectious hospital/department) is optimal.

All patients with MI must be examined by an anesthesiologist and an infectious disease specialist upon admission to the hospital for the first stage of medical care. Patients with mild forms of MI who do not have signs of shock and increased intracranial pressure are hospitalized in the infectious department. Patients with moderate, severe and fulminant forms of MI are hospitalized in the department of anesthesiology and intensive care or, in its absence, in the intensive care ward.

Antibacterial therapy:

The drugs of choice for severe forms of meningococcal infection are cefotaxime or ceftriaxone, administered intravenously as an isotonic sodium chloride solution. Cefotaxime should be the first-line antibiotic for meningococcemia in cases where the use of solutions containing calcium (Ringer's solution, etc.) is envisaged at the hospital stage. However, ceftriaxone can be considered as a drug to continue the therapy of meningococcemia after the acute phase, when the introduction of calcium solutions is no longer necessary.

The need for protection against nosocomial infection and one's own opportunistic flora in critical conditions and with aggressive supportive therapy (catheterization of central veins and urinary bladder, ventilation) dictates the need to prescribe a second antibiotic. It is more appropriate

to use aminoglycosides (amikacin 15 mg/kg/day, netilmicin - 7.5-9 mg/kg for children under 1 year, 6-7.5 mg/kg for children older than 1 year). All drugs are administered intravenously.

Antibacterial therapy should be started on the condition that intravenous infusions are started in a volume sufficient to maintain adequate central hemodynamics.

In moderately severe forms of meningococcemia, antibiotics are started intravenously. Benzylpenicillin can be prescribed for mild forms of meningococcemia. Reserve antibiotics are ampicillin, ceftriaxone, cefotaxime or chloramphenicol succinate.

Doses of the main antibacterial drugs, route, frequency and method of their administration in meningococcemia

| Antibiotic | Optimal route of administration | Daily dose | Number of entries |
|---|---|---|---|
| Ceftriaxone | Bolus, slow IV infusion | 100 mg/kg | 2 |
| Benzylpenicillin | Intravenous bolus | 300-500 thousand units/kg | 6 - 8 |
| Levomycetin succinate | Intravenous bolus | 100 mg/kg | 2 - 4 |
| Cefotaxime | Bolus administration, slow IV infusion | 150 mg/kg | 2 - 4 |
| Ampicillin | Bolus intravenous administration | 300 mg/kg | 4 - 6 |

The duration of antibacterial therapy for MI is 7-10 days.

**Enterovirus infection is** a group of acute infectious diseases caused by intestinal viruses (enteroviruses), characterized by fever and polymorphism of clinical symptoms caused by damage to the central nervous system, cardiovascular system, gastrointestinal tract, muscular system, lungs, liver, kidneys and other bodies.

One of the main features of these infections is a healthy viral load, which constantly determines the occurrence of sporadic forms and mass diseases, which, like morbidity, is observed not only among younger and older children, but also among adults. It was established that the duration of stay of enteroviruses in the intestines does not exceed 5 months. Proteas, the main importance in maintaining the circulation of enteroviruses among the population, apparently, has two factors - the presence of susceptible contingents and a significant duration of virus-carrying. The last feature allows the virus after infection of non-immune persons, creating a highly immune layer, to wait for new susceptible contingents.

The modern classification of enteroviruses was developed in 2000 on the basis of data accumulated so far on the genetic structure and phylogenetic relationships of various representatives of the genus Enterovirus. This genus includes the Picornoviridae family, which, in turn, includes 5 types of non-polio enteroviruses, namely Enterovirus A, B, C, D, E. According to this classification, polioviruses are a separate species within the genus Enterovirus. Type A includes Coxsackie viruses A2-8 10 12 14 16 and enterovirus 71. Type Enterovirus B is the most numerous and includes all Coxsackie B viruses and ECNO, with the exception of ECNO 1, as well as Coxsackie virus A9 and enteroviruses of types 69 73 77 78 . Enterovirus C type unites representatives of Coxsackie A viruses, including types 1 11 13 15 17-22 24. Types of Enterovirus D and E are relatively few and include 2 (Enterovirus 68 and 70) and 1 (A2 plaque virus) representatives, respectively. In addition, the genus includes a significant number of unclassified enteroviruses. Thus, the genus Enterovirus includes more than 100 viruses dangerous to humans. They are widespread and highly resistant to the effects of physical and chemical factors.

Enterovirus infections belong to the group of anthroponoses. The existence of enteroviruses in nature is due to the presence of two main reservoirs - humans, in which the virus reproduces and accumulates, and the external environment (water, soil, food products), in which they can survive due to high resistance. The risk of outbreaks increases significantly when massive enterovirus contamination is "thrown" into the human population, which can most often be realized through water and food transmission. The vertical route of transmission of enterovirus infections is

described. A high risk of congenital enterovirus infection, as a rule, is determined not by an acute enterovirus disease suffered by the mother during pregnancy, but by the presence of a persistent form of enterovirus infection in a woman. Sudden infant death syndrome is associated with congenital enterovirus infection.

The source of infection is a sick person or a virus carrier. The transmission mechanism is airborne or fecal-oral. Children and young people get sick more often. Characteristic summer-autumn seasonality. Immunity after the disease is quite long (up to several years).

Entrance gate infections - mucous membranes of the upper respiratory tract or digestive tract, where the virus multiplies, accumulates and causes a local inflammatory reaction, manifested by symptoms of herpetic sore throat, acute respiratory infections, pharyngitis or intestinal dysfunction. As a result of subsequent viremia, viruses are hematogenously spread throughout the body and settle in various organs and tissues. The tropism of enteroviruses to nervous tissue, muscles, and epithelial cells determines the variety of clinical forms of infection. When the virus penetrates the central nervous system, its defeat is possible with the development of aseptic meningitis, meningoencephalitis or paralytic poliomyelitis-like forms.
EKHO viruses usually do not disseminate from the places of primary penetration, only sometimes they are hematogenously introduced into other organs.

The wide pantropy of enteroviruses is the basis of the wide variety of clinical forms of infection caused by them, which affect almost all organs and tissues of the human body: nervous, cardiovascular, gastrointestinal, respiratory tract, as well as kidneys, eyes, muscles, skin, mucous oral cavity, liver, endocrine organs. Enterovirus infections pose a particular danger in immunocompromised individuals.
Most cases of enterovirus infections are asymptomatic.

Conditionally, two groups of diseases caused by enteroviruses can be distinguished:
I. Potentially severe: - Serous meningitis; - Encephalitis; - Acute paralysis; - Neonatal septic-like diseases; - Myo-(peri-) carditis; - Hepatitis; - Chronic infections of immunodeficient persons. II. Less dangerous: - Three-day fever with or without a rash; - Herpangina;
- Pleurodynia;
- vesicular pharyngitis; - Conjunctivitis; - Uveitis; - Gastroenteritis.

1. Herpetic angina . On the first day of the disease, red papules appear, which are located on the moderately hyperemic mucous membrane of the palatal arches, tongue, soft and hard palate, quickly turn into vesicles 1-2 mm in size, numbering from 3-5 to 15-18, they do not merge between by myself After 1-2 days, the blisters open with the formation of erosions or dissolve without a trace by the 3-6th day of the disease. Pain when swallowing is absent or insignificant, sometimes drooling appears. The increase in cervical and submandibular lymph nodes is small, but their palpation is painful.
2. Epidemic myalgia (Bornholm's disease, "devil's dance", pleurodynia). It is characterized by acute pain localized in the muscles of the anterior abdominal wall, lower chest, back, and limbs. The pains are paroxysmal in nature, lasting from 30-40 seconds to 15-20 minutes, repeat over several days, may be recurrent, but with less intensity and duration.
3. The meningeal syndrome lasts from 2-3 days to 7-10 days, the cerebrospinal fluid is cleared in the 2nd - 3rd week. Possible residual phenomena in the form of asthenic and hypertensive syndromes. Among other neurological symptoms in meningitis of enterovirus etiology, there may be disorders of consciousness, increased tendon reflexes, absence of abdominal reflexes, nystagmus, clonus of the feet, and short-term oculomotor disorders.
4. Paralytic forms of enterovirus infection differ in polymorphism: spinal, bulbospinal, pontine, polyradiculoneurotic forms can develop. The spinal form is more common than others, which is characterized by the development of acute flaccid paralysis of one or both legs, less often - hands with a pronounced pain syndrome of a muscular nature. The course of these forms is easy, does not leave persistent paresis and paralysis.

5. Enterovirus fever (minor illness, 3-day fever). This is the most common form of enterovirus infection, but it is difficult to diagnose with sporadic disease. It is characterized by a short-term fever without pronounced symptoms of local lesions. It proceeds with moderate general infectious symptoms, the well-being is disturbed little, there is no toxicosis, the temperature persists for 2-4 days. Clinically, it can be diagnosed when there is an outbreak in the team, when other forms of enterovirus infection are also found.
6. Enterovirus exanthema ("Boston fever"). It is characterized by the appearance from the 1st - 2nd day of the disease on the face, trunk, limbs of rashes of pink color, spotted or spotted-papular in nature, sometimes there may be hemorrhagic elements. The rash lasts 1-2 days, less often - longer and disappears without a trace.
7. Intestinal (gastroenteric) form . It occurs with watery diarrhea up to 5-10 times a day, abdominal pain, flatulence, infrequent vomiting. Symptoms of intoxication are moderate. In children under 2 years of age, the intestinal syndrome is often combined with catarrhal phenomena from the nasopharynx. The duration of the disease in young children is 1-2 weeks, in older children 1-3 days.
8. The respiratory (catarrhal) form is manifested by weakly expressed catarrhal phenomena in the form of nasal congestion, rhinitis, dry rare cough. During the examination, hyperemia of the mucous membrane of the oropharynx, soft palate and back wall of the pharynx is revealed. Mild dyspeptic disorders may be noted. Recovery occurs in 1-15 weeks.
9. Myocarditis, encephalomyocarditis of newborns, hepatitis, damage to the kidneys, eyes (uveitis) - these forms of enterovirus infection are rare in children. Their clinical diagnosis is possible only in the presence of manifest forms of enterovirus infection or epidemic outbreaks of the disease. Most often, they are diagnosed during virological and serological studies. The leading place among children's neuroinfections is still occupied by meningitis, which accounts for 70-80% of the total number of infectious lesions of the central nervous system. An increase in the incidence of enterovirus meningitis is noted annually in the summer-autumn period. Mostly children of preschool and school age are sick. Clinically, aseptic serous meningitis, caused by different types of polioviruses, ESNO viruses, Coxsackie viruses A and B, is practically impossible to distinguish. Changes in cerebrospinal fluid are also not different.

Diagnosis of enterovirus infection includes 4 main methods:

1) serological; 2) immunohistochemical; 3) molecular and biological; 4) cultural. Serological methods are aimed at detecting markers of enterovirus infections in the blood serum of patients. Early markers of infection include IgM and IgA. Immunofluorescence and enzyme-linked immunosorbent assay methods are used to indicate IgM. In patients with acute symptoms of the disease, EV-specific IgM is determined 1-7 days after the onset of infection. After 6 months, IgM usually disappears.

Virological methods of research are aimed at isolating enteroviruses from clinical material (blood, feces, cerebrospinal fluid) on sensitive cell cultures.
The main goal of immunohistochemical methods is the detection of enterovirus antigens in situ. Immunofluorescence and immunoperoxidase analyzes are among the most available methods of immunohistochemistry. Molecular biological research methods are aimed at identifying the genetic material of enteroviruses. Polymerase chain reaction with a reverse transcription stage is used to diagnose enterovirus infections , which has a number of advantages over the above-mentioned methods: high specificity, sensitivity and speed of execution.

Treatment of enterovirus infection:

An increase in the level of endogenous interferon in the cerebrospinal fluid in children with acute epidemic enteroviral meningitis was shown, which plays a major role in getting rid of the infection. Interferons are formed at the very beginning of a viral infection. They increase the resistance of cells to damage by viruses. Interferons are characterized by a wide antiviral spectrum (they do not have specificity of action against individual viruses). Viruses do not develop resistance to interferons. Currently, alpha-interferon preparations (alpha-2a, alpha-2b), both natural and recombinant, are mainly used as antiviral agents. Interferons are used locally and parenterally. The

second group of drugs used to treat enterovirus infections is immunoglobulins. Intravenous administration of the drug was the most effective, it is widely used in the treatment of immunodeficient patients with acute and chronic meningoencephalitis caused by enteroviruses. However, the experience of using immunoglobulins in this situation has not been sufficiently studied. There are data on the successful treatment of meningoencephalitis with intraventricular administration of gamma globulin.

Specific prevention . Not developed.

Non-specific prevention . Contact children can be instilled with 5 caps of leukocyte interferon in the center of the infection. in the nasal passages 3-4 times a day for 7 days. Immunoglobulin in a dose of 02 ml/kg, in/m provides a protective effect. Ventilation and disinfection of premises, compliance with the rules for removal and disinfection of impurities, providing the population with epidemiologically safe products.

Resuscitation of newborns. APGAR scale.

The level of mortality and disability of asphyxiated newborns depends on the timeliness and quality of resuscitation measures in the delivery room. In carrying out resuscitation measures, the participation of two, and in some cases, three specialists at the same time is necessary.

A neonatologist on round-the-clock duty must be present in the delivery room during all deliveries before the birth of the child. It is necessary to select, mount and check the functioning of the resuscitation equipment before each delivery, since the birth of a baby in a state of asphyxia can be unpredictable.

*Equipment preparation:*

1. Turn on the source of radiant heat to warm the resuscitation table (the temperature on it should be 36-37 $^0$C);

2. Check the oxygen supply system: availability of oxygen, pressure, availability of connecting hoses;

3. Roll up the roller from the diaper under the shoulders;

4. Prepare equipment for suctioning contents from the upper respiratory tract. A rubber balloon is used. Electromechanical suction devices must create a vacuum of no more than 100 cm of the water column;

5. Prepare a gastric tube, plaster, scissors;

6. Mount a set for artificial lung ventilation (Ambu type). The size of the mask is selected depending on whether the birth of a full-term or premature child is expected. The resuscitation bag can be anesthetic and self-inflating. In the latter case, an oxygen tank is attached to the bag to provide ventilation with 90-100% oxygen. After attaching the mask and the oxygen source to the bag, clamp the mask with the palm of your hand and squeeze the bag. Check the feeling of the pressure of the breathing mixture on the palm and the functioning of the safety valve;

7. Prepare a set for intubation: with interchangeable blades, attach blade N0 for a premature baby or N1 for a full-term baby; check the functioning of the lighting system; choose an intubation tube depending on the estimated body weight and gestational age of the child.

**Measures after the birth of the child (depends on the absence or presence of meconium particles in the amniotic fluid):**

*1. There are no meconium particles in the amniotic fluid:*

- place the newborn under a source of radiant heat;
- dry the skin with quick wetting movements through the diaper;
- throw away the wet diaper;
- ensure maximum airway patency: position on the back with the head slightly thrown back. This position is better fixed with a roller placed under the shoulders;
- suck the contents first from the mouth, then from the nasal passages;
- if spontaneous breathing has appeared, conduct tactile stimulation (one of the three methods is performed, which is repeated no more than twice: irritation of the sole, light blows on the heel,

irritation of the skin along the spine. Repeatedly repeating them is impractical, as it does not give success , but it leads to the loss of precious time. It is forbidden to: irrigate the child with cold or hot water, give a jet of oxygen to the face, squeeze the chest, hit the buttocks).

*2. There are particles of meconium in the amniotic fluid, that is, meconial aspiration takes place:*

- the newborn is placed under a source of radiant heat without wiping;
- without wasting time on drying, they position themselves on the back with the head slightly thrown back and a roller under the shoulders;
- tracheal intubation is performed;
- re-suction the contents of the upper respiratory tract;
- the contents of the tracheobronchial tree are sucked directly through the intubation tube (if meconium remains in the intubation tube after suctioning , intubation and suctioning are repeated. Tracheobronchial tree lavage is not performed in order not to wash away the surfactant).

All preparatory measures must be completed no later than in 20 seconds. After that, the first assessment of the child's condition is made. The condition of the child according to the Apgar scale is not used to determine the amount of resuscitation measures, because the first assessment is made too late, at the end of the first minute of life. The assessment on the Apgar scale at the 1st and 5th minutes serves to determine the effectiveness of resuscitation measures.

**Assessment of breathing:**

*Spontaneous breathing is present* - heart rate is assessed.

*There is no spontaneous breathing* - start artificial ventilation of the lungs with 90-100% oxygen through a bag and a mask. The effectiveness of ventilation is determined by chest movement and auscultation data. The first 2-3 breaths are performed with a pressure of 20-40 cm of the water column, after which ventilation is performed with an inspiratory pressure of 15-20 cm of the water column and a frequency of 20-40 per 1 min.

Carrying out artificial ventilation for more than 2 minutes. requires the introduction of an orogastric tube to prevent distension of the stomach with gas and prevent regurgitation. The probe is inserted to a depth equal to the distance from the bridge of the nose to the earlobe and from the earlobe to the epigastric area. After inserting the probe, gas is sucked from the stomach with a syringe, the probe is left open and fixed with an adhesive plaster to the cheek. Artificial ventilation is continued over the probe.

After 15-30 sec. artificial ventilation give another assessment of the child's condition, determine the heart rate (HR). Heart rate is counted for 6 seconds. and multiplied by 10. Ventilation is stopped during counting.

**Heart rate estimate (in 1 minute):**

- more than 100;
- from 60 to 100 and the frequency increases;
- from 60 to 100 and the frequency does not increase;
- less than 60.

**1. HR more than 100:**

- in the presence of spontaneous breathing, stop ventilation and evaluate the color of the skin,
- in the absence of spontaneous breathing, artificial ventilation is continued until it appears,
- with heart rate less than 100, artificial ventilation is always carried out, regardless of the presence of spontaneous breathing.

**2. Frequency of SS from 60 to 100 and increasing:**

- continue artificial ventilation of the lungs.

**3. Frequency of SS from 60 to 100 and does not increase:**

- continue artificial ventilation of the lungs and start a closed cardiac massage with heart rate less than 80.

**4. HR less than 60:**

- artificial lung ventilation and closed heart massage.

Heart rate control is carried out after 10-15 seconds, until the heart rate is more than 100 and spontaneous breathing is established. In this situation, the last assessment of the condition is carried out, the color of the skin is evaluated. With the effectiveness of ventilation and blood circulation, the skin color is pink, the child needs observation. Acrocyanosis, characteristic in the first hours after birth, develops as a vascular reaction to a change in the temperature of the external environment and does not indicate hypoxia. A sign of hypoxia will be general cyanosis. The child needs an increased concentration of oxygen in the mixture for inhalation, it is provided by supplying a free jet from an oxygen hose. When the end of the hose is located at a distance of 1-1.5 cm from the nasal passages, the oxygen content in the inhaled air will be approximately 80%. The disappearance of cyanosis indicates the elimination of hypoxia. The hose is gradually removed from the nasal passages. Preservation of the pink color of the skin when the hose is removed by 5 cm indicates that there is no need for an increased concentration of oxygen.

**Closed heart massage:**

Indications for closed heart massage — after 15-30 seconds. artificial lung ventilation, heart rate less than 60 per minute.

A closed heart massage is performed, pressing on the lower third of the sternum. It is located below the conventional line drawn between the nipples. It is important not to press on the xiphoid process to prevent tearing the liver.

They press with two thumbs, the other 4 fingers of both hands support the back, or with the tips of two fingers of one hand: II and III, or III and IV, and the other hand supports the back. The depth of pressing is 1-1.5 cm, the frequency is 120 in 1 minute. Simultaneous compression of the sternum and inhalation during artificial ventilation through a bag and mask will lead to gas entering the stomach. This is prevented by synchronizing the procedure: after 1 breath, 3 pressures on the sternum are applied.

**Tracheal intubation:**

Indications are the need for long-term artificial lung ventilation, meconium aspiration, diaphragmatic hernia, unsuccessful ventilation through a bag and mask.

Equipment preparation: installation of the laryngoscope and inspection of lighting, selection of the tube, the tube is shortened to 13 cm, the conductor is inserted. Procedure - the end of the blade is inserted into the epiglottis, the entrance to the larynx is visualized, the intubation tube is inserted, the laryngoscope and guide are removed, preliminary control of the position of the intubation tube is carried out based on the movement of the chest, anterior abdominal wall and auscultation data, on symmetrical areas of the chest and epigastric region, fix the intubation tube.

**Use of medications:**

The following medications are used in the delivery room: adrenaline, agents that normalize BCC, sodium bicarbonate, anesthetic antagonists.

The use of the previously recommended drugs (calcium gluconate solution, cocarboxylase, euphilin, hormones, etc.) is considered inappropriate, and certain drugs are also contraindicated.

**Adrenalin.** Indications for use: after 15-30 seconds. artificial ventilation of the lungs with 100% oxygen and closed heart massage, the heart rate remains less than 60 in 1 min., absence of heart contractions. In this case, artificial ventilation, closed heart massage, and adrenaline injection are started at the same time. The simultaneous participation of three specialists is necessary. The concentration of the solution is 1:10000. Prepare 1 ml: 0.1 ml of adrenaline solution and 0.9 ml of isotonic sodium chloride solution. The dose is 0.1-0.3 ml/kg of a 1:10000 solution. The route of administration is intravenous or endotracheal. With endotracheal administration, the dose is 0.3-0.5 ml/kg. If there is no effect, repeat every 5 minutes, but no more than 30 minutes.

**Means that normalize BCC:**

Preparations: 0.9% sodium chloride solution. Indications: hypovolemia.

Symptoms of hypovolemia: pallor, weak pulse with sufficient heart rate, decrease in blood pressure, history of childbirth. It is recommended to assume that all children who require

resuscitation have hypovolemia. Prepare 40 ml of solution, dose - 10 ml/kg. The route of administration is intravenous.

**Narcotics antagonists:**

Drugs: Naloxone. Indications: narcotic depression. The means do not stimulate the respiratory center and are ineffective in the case of respiratory depression of another nature. Naloxone: a dose of 0.01 mg/kg, can be repeated after 5 minutes. Route of administration: intravenous and endotracheal - preferred, intramuscular or under the skin - acceptable. Speed - type fast.

After carrying out resuscitation measures, the newborn should be transferred to the intensive care unit (ward) for further treatment.

Maintenance of adequate gas exchange after resuscitation measures in children with severe asphyxia is determined by the degree of respiratory insufficiency. Absence of independent breathing in the case of narcosis depression of the nervous system, the presence of severe respiratory failure (II-III degree) in newborns with aspiration syndrome, intrauterine pneumonia, developmental defects, immaturity of the lungs and airways serve as indicators for long-term (more than 2 hours) artificial ventilation of the lungs without transferring these children to independent breathing after birth. If after 5-10 min. after artificial lung ventilation, the child develops independent breathing, artificial ventilation is canceled and conditions are created to maintain adequate gas exchange:

1. ensure patency of the respiratory tract (individually selected position of the body, suction of sputum, stimulation of the cough reflex, massage, inhalations);

2. carry out inhalations of humidified and heated oxygen in a concentration that provides PaO2 in the range of 7.98-10.64 kPa, or 60-80 mm Hg. Art., but if oxygen therapy is used for a long time, the oxygen content in the mixture should not exceed 50% (FiO2=0.5), because as a result of prolonged use of oxygen in a concentration of more than 50%, toxic damage to the respiratory tract and lungs occurs;

3. provide correction of metabolic changes (hypo-, hyperglycemia, hypercalcemia, hypocalcemia, acidosis);

4. to eliminate pulmonary hypertension, use Talazolin (5-10 μg/kg for 1 min.) or Aminazine (0.25 mg/kg of body weight), or droperidol (under blood pressure control);

5. Protease inhibitors (contrical — 1000 units/kg per day) are administered, means of pharmacological correction of microcirculation — curantyl (0.05-0.1 mg of a 0.5% solution, trental (0.1 ml/kg of a 15% solution), antioxidants (50 mg/kg of tocopherol acetate per day);

6. after elimination of oxygen deficiency and stabilization of gas exchange, agents are introduced that normalize redox processes — 1-2 ml of 0.15% solution of essentiale, 3 mg/kg of cytochrome C (1.1 ml/kg of 0.25% solution), or 1 ml/kg cytomak, 0.1 ml/kg 0.5% lipoic acid solution, 0.1-0.2 ml/kg 5% unitiol solution, 8 mg/kg cocarboxylase.

Normalization of cardiovascular activity at the 2nd stage of treatment of asphyxia is mainly carried out by ensuring adequate cardiac output, blood pressure, correction of changes in microcirculation, rheological properties of blood, intravenous drip administration of dopamine at an individually selected rate (2.5-10 μg/kg in 1 min.), reodilutants (albumin, reopolyglukin), disaggregants (curantyl, trental, xanthine nicotinate, etc.).

Diagnosis and correction of metabolic changes that accompany severe asphyxia are important. Insufficient intake of food, impaired absorption of food products in the intestine, significant consumption of endogenous reserves of carbohydrates to maintain the energy balance in the body of newborns with a severe form of asphyxia lead to the development of hypoglycemia in them (a decrease in blood glucose to 2.2 mmol/l in full-term infants and to 1 .2 mmol/l in premature infants). Along with this, a decrease in glucose absorption, in particular in the case of centralization of blood circulation, tissue hypoxia, can cause an increased content of glucose in the blood (6-8 mmol/l), especially if it is administered intravenously. This may threaten the development of hyperglycemic hyperosmolar complications. To combat hypoglycemia, a 10% glucose solution is administered intravenously at the rate of 0.2 g/kg of body weight in 1 minute. (2 ml/kg/min) and subsequent transition to intravenous drip at an individually selected rate (the blood glucose level is determined every 2 hours until it stabilizes). If hyperglycemia with a blood glucose level of 8-12

mmol/l is detected in a newborn, then the rate of intravenous glucose administration should be reduced or this procedure should be stopped. Newborns with significant hyperglycemia (glucose content is 12 mmol/l) are given insulin at 0.1-0.2 units/kg every 6 hours until the glucose level normalizes. If the level of glucose in the blood is 20 mmol/l, then the dose of insulin (per 1 injection) can be increased to 0.5 units/kg. Correction of acidosis in case of adequate elimination of $CO_2$ is carried out when the concentration of hydrogen cations in the blood in the first hours of life is lower than 7.2, and VE is less than 10 mmol/l. Correction of hypocalcemia is carried out with the help of intravenous administration of 10% calcium gluconate solution when the content of ionized calcium in the blood decreases to 0.9 mmol/l.

Treatment of CNS injuries after sustained asphyxia should be differentiated depending on the time and depth of exposure to hypoxia, functional and morphological injuries, and the degree of maturity of the CNS. And in order to reduce postnatal damage to the central nervous system in a newborn child with asphyxia, it is necessary to ensure normal indicators of gas exchange, hemocardiodynamics, and metabolism from the first hours of his life.

In the intensive therapy of damage to the central nervous system during asphyxia, 2 main directions can be distinguished:

1) correction of shifts in systems that ensure the vitality of the body (gas exchange, hemocardiodynamics, hemostasis, metabolism, protein-energy deficiency);

2) therapy aimed at combating edema, normalization of blood circulation, fluid dynamics, brain metabolism, treatment of convulsive, hyperthermic, hydrocephalic syndromes.

To combat cerebral edema, which prevails in the first 3-5 days of severe asphyxia, it is important to maintain adequate blood hyperosmolarity, especially during infusion therapy. This is due to the fact that the 1st stage of acute asphyxia is accompanied by hyponatremia, hypoproteinemia, which lead to a decrease in blood osmolarity, which can further increase due to the intravenous administration of a large amount of glucose solutions with a predominant content of "free" water. It is dangerous to administer a bolus solution in a volume of more than 15 ml/kg of body weight to newborns with posthypoxic cerebral edema. Preference should be given to slow, dropwise administration of solutions, ensuring maintenance of blood osmolality within 290-300 mosm/l. This is mostly albumin, concentrated plasma, which cause an increase in blood osmolarity, and glucose solutions can be administered in a volume of 20-30 ml/kg of body weight per day. It is known that glucocorticoids improve indicators of systemic and cerebral hemodynamics, restore the function of the blood-brain barrier, cell membranes, and stabilize lysosomal enzymes. In newborns with posthypoxic cerebral edema, dexamethasone (0.5-1.5 mg/kg per day, 3-5 days) is preferred, while prednisolone at a dose of 3-5 mg/kg per day is less effective. Sometimes, to combat post-hypoxic brain edema and relieve convulsive syndrome in newborns, a 10% solution of phenobarbital is administered intravenously in a shock dose of 20 mg/kg per day on the 1st day of life and 3-4 mg/kg per day in the following days, with tongue control of its concentration in the blood. Drugs that stabilize cell membranes include lipostabilizing triplet (essential, tocopherols, lipoic acid).

Starting from the 5-7th day of treatment, calcium channel blockers (0.1-0.2 mg/kg nifedipine, 2 mg/kg cinnarizine), other means that improve cerebral blood circulation can be administered to normalize brain hemo-liquid dynamics according to the indications under the control of cerebrovascular dopplerography (2 mg/kg cavinton, 20 mg/kg complamin, 0.5 mg/kg sermion per day). Drugs that have a cerebroprotective effect include nootropics (piracetam, encephabol), which, under conditions of hypoxia, improve blood supply to ischemic areas of the brain, utilization of glucose in nerve cells, reduce the level of lactate, activate oxidative phosphorylation, increase the formation of ATP, creatine phosphate and thus improve utilization oxygen by brain cells. However, the dose of these drugs (20-100 mg/kg per day) should be selected individually, taking into account the patient's positive reaction to their administration. These drugs should be prescribed especially carefully to newborns with convulsive syndrome. It should be noted that, in addition to the above-mentioned drugs, sodium oxybutyrate (80-100 mg/kg of body weight IV or 100-150 mg/kg IV or orally), pantogam (100 mg/kg of body weight per day). As an anticonvulsant, phenobarbital is used,

starting with shock doses (20 mg/kg of body weight per day IV) and then moving to a maintenance dose (3.4 mg/kg of body weight per day IV or orally). There is a warning in the literature that the use of phenobarbital 5 mg/kg per day or more can cause its accumulation in the body, which is manifested by significant clinical intoxication (lethargy, impaired suction, breathing, etc.). Diazepam (seduxen) is also used at a dose of 0.1 mg/kg IV. Diazepam significantly increases the activity of endogenous aminobutyric acid, which increases its sedative and anticonvulsant effect. A smaller anticonvulsant effect is given by such drugs as sodium oxybutyrate (80-100 mg/kg), droperidol (0.3-0.5 mg/kg of body weight per day).

In newborns with intracranial hemorrhages, it is important to use angioprotectors that reduce the permeability of the walls of brain vessels (3-4 mg/kg of sodium etamsylate, 0.5 ml of 0.025% androxone solution), a protease inhibitor — Kontrical (1000 units/kg per day). For the prevention and treatment of posthemorrhagic hydrocephalus, lumbar punctures are used, as well as drugs that inhibit fluid secretion in the cisterns of the brain and spinal cord (diacarb, furosemide). It is more appropriate to prescribe furosemide for the treatment of severe hypoxia in newborns from the first days of life at 3 mg/kg per day (in/in, in/m), controlling the level of potassium in the blood, and use diacarb after the 7th day, because its effect is mainly determined suppression of carbonic anhydrase, the activity of which is insignificant in the first days of a child's life. In addition, the effect of diacarb is not realized in conditions of acidosis, which is observed mainly in this period. The dose of diacarb is selected individually under the control of neurosonography (30-80 mg/kg per day). It should be administered in the first half of the day simultaneously with potassium preparations (potassium orotate, panangin), and in the presence of acidosis, alkaline therapy should be carried out. The lack of effect from the use of the maximum dose (80-100 mg/kg of body weight per day) of diacarb according to neurosonographic, clinical indicators (reduction of parietal tension, limb tremor, cessation of eye movement disorder) determines the need for operative treatment — shunting to remove excess fluid from the cisterns of the brain.

4 Summary:

After completing the lesson on the topic " Emergency conditions in children of various ages. Simulation training ", students should:

Have formed and practiced professional skills in providing emergency care for the most common emergency conditions in children of different ages .

To learn the ability to independently use knowledge and skills in the diagnosis and treatment of the most common emergency conditions in children of various ages .

Have a well -formed and clear idea of the sequence of actions in the algorithm for providing emergency care for the most common emergency conditions in children of various ages .

To have the competence of professional communication in the team when providing emergency care to children of different ages .

5 List of recommended literature:

Main:

1. Neonatology: a textbook in 3 volumes / T. K. Znamenska, Yu.G. Antipkin, M.L. Aryaev and others; under the editorship T.K. Znamenskaya Lviv: T.V. Marchenko Publisher, 2020, T. 1. 407 p.; T. 2. 455 p.; T. 3. 379 p.
2. Pediatrics. Differential diagnosis. Emergency conditions. / under the editorship Aryaeva M.L., Kotova N.V. Odesa: ONMedU, 2017. 280 p.
3. Unified clinical protocol "Initial, resuscitation and post-resuscitation care for newborns in Ukraine" No. 225 dated March 28, 2014

Additional:

1. Pediatrics with a course of infectious diseases and the basics of immunoprophylaxis: a textbook (University I-III year) / S.K. Tkachenko, R.I. Potsyurko, L.V. Besh and others; under the editorship S.K. Tkachenko, R.I. Rat — 7th ed., ed. 2018
2. Order of the Ministry of Health of Ukraine dated September 14, 2021 No. 1945 "On approval of the Unified clinical protocol of primary medical care "Integrated management of childhood diseases".
3. Order of the Ministry of Health of Ukraine dated June 5, 2019 No. 1269 "Emergency medical care: new clinical protocol."
4. Order of the Ministry of Health of Ukraine dated March 20, 2008 No. 149 "On the approval of the Clinical Protocol for the medical care of a healthy child under 3 years of age."
5. Order of the Ministry of Health of Ukraine dated October 8, 2013 No. 868 "Unified clinical protocol of primary, secondary (specialized) medical care for bronchial asthma in children."
6. Order of the Ministry of Health of Ukraine dated June 8, 2015 No. 327 "Unified clinical protocol of primary medical care for cough in children aged six years and older."
7. Order of the Ministry of Health of Ukraine dated December 30, 2015 No. 916 "Unified clinical protocol of emergency, primary, secondary (specialized) and tertiary (highly specialized) medical care for drug allergy, including anaphylaxis."

Electronic information resources:

1 https://zakon.rada.gov.ua/laws/show/z0356-22#n42 - Order of the Ministry of Health of Ukraine No. 441 dated 09.03.2022 "On approval of procedures for providing pre-medical assistance to persons in emergency situations "
2 www.ama-assn.org – American Medical Association
3 www.who.int - World Health Organization
4 http://bma.org.uk - British Medical Association
5 w w w.gmc-uk.org – General Medical Council ( GMC )
6 www.bundesaerztekammer.de – German Medical Association
7 https :// emergencymanual . Stanford . edu / downloads / – Stanford Handbook of Emergency Medicine
8 https://www.futurelearn.com/courses/critical-care – University of Glasgow Handbook of Emergency Medicine