

**CLINICAL CHARACTERISTICS AND MANAGEMENT OF
EMERGENCY CONDITIONS IN PEDIATRIC DENTISTRY**

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Angioedema

Schwinke's edema is an immediate allergic reaction, manifested by angioedema spreading to the skin, subcutaneous tissue, and mucous membranes.

Clinical diagnosis

It is characterized by the sudden appearance of limited edema in places with loose subcutaneous tissue, thickets of the lips, auricles, neck, hands, feet. Edema can often reach significant sizes and deform the affected area. The immediate danger of this reaction is the frequent development of mechanical asphyxia due to edema of the upper respiratory tract. With laryngeal edema, the child has a barking cough, hoarseness of voice, difficulty inhaling and, possibly, exhaling due to the bronchospasm joining. If the tongue swells, speech becomes difficult, and the processes of chewing and swallowing are disrupted.

Emergency care

- Stop manipulating, provide convenient access to the child;
- reassure the child and parents;
- the administrator should call the NSR team;
- Stop ingesting the allergen immediately.

Administer antihistamines intramuscularly or intravenously:

- 2.5% pipolfen solution 0.1-0.15 ml / year of life or
- 2% suprastin solution 0.1-0.15 ml /year of life.

Enter a 3% prednisone solution at a dose of 1-2 mg / kg i.m. or iv.

According to indications, with increasing laryngeal edema with obstructive respiratory failure, intubation or tracheostomy is performed.

Hospitalization in the somatic department.

Anaphylactic shock

Anaphylactic shock is an acutely developing, life-threatening pathological process caused by an immediate allergic reaction when an allergen is introduced into the body,

characterized by severe circulatory, respiratory, and central nervous system disorders.

There are two variants of the rapid course of anaphylactic shock, depending on the leading clinical syndrome: acute respiratory failure and acute vascular insufficiency.

In case of anaphylactic shock with a leading respiratory syndrome

If the child is deficient, weakness suddenly appears and increases, a feeling of compression in the chest with a feeling of lack of air, an excruciating cough, a throbbing headache, pain in the heart area, and fear. There is a sharp pallor of the skin with cyanosis, foaming at the mouth, difficulty wheezing with dry wheezing on exhalation. Angioedema of the face and other parts of the body may develop. Subsequently, with the progression of respiratory insufficiency and the addition of symptoms of acute adrenal insufficiency, death may occur.

Anaphylactic shock with the development of acute vascular insufficiency is also characterized by a sudden onset with the appearance of weakness, tinnitus, and profuse perspiration. There is an increasing pallor of the skin, acrocyanosis, a progressive drop in blood pressure, a thready pulse, and heart tones are sharply weakened. Loss of consciousness and seizures are possible after a few minutes. Death occurs with an increase in the phenomena of cardiovascular insufficiency.

Less often, anaphylactic shock occurs with a gradual development of clinical symptoms.

Emergency therapy

-Stop the administration of the substance and/or other manipulations

-Inform the administrator about the need to call the NSR team

-Lay the child horizontally, fix the tongue, turn the head to the side

-Put a piece of ice on the injection site (asphyxia is possible with injections in the oral cavity!)

Hemodynamic and respiratory monitoring

-Injection of 0.3-0.5 ml of 0.1% epinephrine subcutaneously into any part of the body,

-1-3 mg / kg of prednisone weight intravenously or intramuscularly,

-To stabilize blood pressure: 1% mezaton 0.05-0.1 ml / year of life

-After stabilization of blood pressure, 2.5% pipolfen solution 0.1 ml / year of life / m or 2% suprastin

solution 2-4 mg / m in severe IV,

-For bronchospasm: 4 mg per kg of body weight 2.4% euphyllin IV,

-For tachycardia: 0.06% corglycone with 10 ml of saline solution IV.

If necessary, CPR!

Emergency hospitalization!!

Fainting

Sudden short-term loss of consciousness with loss of muscle tone due to transient

disorders of cerebral circulation

reasons

I-neurological diseases: epilepsy, VSD, hysteria

II- therapeutic diseases: drug-induced orthostatic hypotension, hypoglycemia, acute posthemorrhagic anemia, situational syncope, hypoparathyroidism, aortic stenosis, hyperventilation, reflex, cough, etc.

Clinical diagnosis

Syncope may be preceded by a presyncopal condition (lipothymia): feeling of discomfort, nausea, yawning, sweating, weakness in the legs, darkening in the eyes, flashing "flies" in front of the eyes, increasing dizziness, noise or ringing in the ears, numbness of the extremities. If the child manages to sit down or lie down, the attack does not develop completely, but is limited to a state of deafness, yawning, and nausea.

Syncopal condition is characterized by loss of consciousness - the child does not make contact. Muscle tone is sharply reduced, the face is pale, pupils are dilated, pulse is weak, blood pressure is reduced, heart tones are muffled, heart rate and rhythm may be different, breathing is shallow. Deep fainting may (rarely) be accompanied by short-term tonic seizures. The restoration of consciousness occurs quickly in a horizontal position. In the postsyncopal period, children experience weakness, headache, pallor, and arterial hypotension.

Thus, the main signs of syncope are: suddenness of development; short duration (from a few seconds to 3-5 minutes); reversibility: rapid and complete restoration of consciousness - the child is guided by the environment, remembers the circumstances preceding the loss of consciousness.

Children with vegetative-vascular dystonia in prepubertal and pubertal age are more likely to faint. The most common are orthostatic and sinocarotid syncope (loss of consciousness). Vasovagal syncope is manifested in typical provocative situations (pain, fear of manipulation, the sight of blood, prolonged exposure to stuffy rooms, etc.); it is based on increased activity of the parasympathetic division of the autonomic nervous system with a sharp drop in peripheral vascular tone. Orthostatic syncope develops during the transition from a horizontal to a vertical position in children with insufficient compensatory mechanisms for regulating vascular tone. Sinocarotid syncope is provoked by sudden turns and tilts of the head, compression in the neck; it is based on increased sensitivity of the carotid sinuses and a reflex decrease in heart rate. In each case, it is necessary to exclude other causes of sudden loss of consciousness. Deep fainting attacks accompanied by seizures should be distinguished from epilepsy, which is characterized by loss of consciousness, hypersalivation, involuntary urination and/or defecation, amnesia, and paroxysms. Changes in heart rate, blood pressure, and pulse are not typical.

Emergency therapy

- Lay the child horizontally with raised legs (angle 30) without a headrest,
- Unbutton clothes and provide fresh air,
- spray (wipe) face and chest with water, pat on the face,
- inhale fumes of ammonia,
- if fainting continues, enter 25% r-r of cordiamine 0.1 ml / year of life in / m, 1% r-r of mezaton 0.1 ml / year of life (no more than 1 ml) in / m

Collapse

Collapse is a life-threatening acute vascular insufficiency characterized by a sharp decrease in vascular tone, a decrease in circulating blood volume, signs of brain hypoxia and suppression of vital functions.

The most common causes of collapse in children:

- Severe course of acute infectious pathology (intestinal infection, influenza, acute respiratory viral infections, pneumonia, pyelonephritis, sore throat, etc.).
- Acute adrenal insufficiency.
- Overdose of antihypertensive drugs.
- Acute blood loss.
- Severe injury.

Clinical diagnosis

The collapse clinic develops, as a rule, during the peak of the main disease and is characterized by a progressive deterioration of the patient's general condition. Depending on the clinical manifestations, three phases (variants) of collapse are conditionally distinguished: sympathotonic, vagotonic and paralytic.

Sympathotonic collapse is caused by a violation of peripheral blood circulation due to arteriole spasm and centralization of blood circulation, compensatory release of catecholamines. It is characterized by: agitation of the child, increased muscle tone, pallor and marbling of the skin, cold hands and feet, tachycardia, normal or elevated blood pressure. However, these symptoms are short-lived, and collapse is more often diagnosed in the following phases.

In vagotonic collapse, there is a significant expansion of arterioles and arteriovenous anastomoses, which is accompanied by blood deposition in the capillary bed. Clinically characteristic: lethargy, adynamia, decreased muscle tone, pronounced pallor of the skin with marbling, gray-cyanotic color, pronounced acrocyanosis, a sharp drop in blood pressure, a weak heart rate, often bradycardia, noisy and rapid breathing of the Kussmaul type, oliguria.

Paralytic collapse is caused by passive capillary expansion due to depletion of blood circulation regulation mechanisms. This condition is characterized by: lack of consciousness with suppression of skin and reflexes, the appearance of blue-purple spots on the skin of the trunk and limbs, bradycardia, bradypnea with a transition to periodic Cheyne-Stokes respiration, blood pressure drops to critical numbers, pulse thready,

anuria. In the absence of emergency care, death occurs.

Treatment measures should be initiated immediately! Emergency care:

-Lay the child horizontally on his back with his head slightly tilted back, cover with warm heating pads and provide fresh air.

-Ensure free passage of the upper respiratory tract: perform an inspection of the oral cavity, remove restrictive clothing.

-In case of symptoms of sympathotonic collapse, it is necessary to:

a) relieve peripheral vascular spasm by intramuscular administration of antispasmodics:

- 2% solution of papaverine 0.1 ml/year of life or

- 0.5% solution of dibazole 0.1 ml/year of life or

- solution of no-shpa 0.1 ml/year of life;

b) with neurotoxicosis, acute adrenal insufficiency, already in this phase, it is necessary to prescribe glucocorticoids intravenously or intramuscularly:

- Hydrocortisone (preferably!) in a single dose of 4 mg / kg or

prednisone in a dose of 1-2 mg / kg

. -In case of vagotonic and paralytic collapse:

a) provide access to the peripheral vein and begin infusion therapy with rheopolyglucine solution or crystalloids (0.9% sodium chloride solution or Ringer's solution) at the rate of 20 ml / kg for 20-30 minutes;

b) simultaneously administer glucocorticosteroids in a single dose:

- Hydrocortisone 10-20 mg / kg

- prednisone 5-10 mg / kg intravenously or intramuscularly, or into the muscles of the floor of the oral cavity, or

- dexamethasone 0.3-0.6 mg / kg (in 1 ml of a 0.4% solution - 4 mg) intravenously or intramuscularly;

c) with persistent arterial hypotension:

- re-inject 0.9% sodium chloride solution or Ringer's solution in a volume of 10 intravenously in combination with rheopolyglucine 10 solution under the control of heart rate, blood pressure and diuresis;

- prescribe a 1% solution of mezaton 0.1 ml / year of life in / in a jet slowly or

- 0.2% solution of norepinephrine 0.1 ml / year of life intravenously (in 50 ml of 5% glucose solution) at a rate of 10-20 drops

per minute (in very severe cases - 20-30 drops per minute) under blood pressure control.

Subcutaneous and intravenous administration of norepinephrine is not recommended due to the risk of necrosis at the injection site (only in exceptional cases when it is impossible to inject into a vein).

-In the absence of an effect from the measures taken, intravenous titration of

dopamine at a dose of 8-10 micrograms / kg per minute under the control of blood pressure and heart rate.

- According to the indications, primary cardiopulmonary resuscitation.

Hospitalization in the intensive care unit after emergency measures

Epileptic seizure

Epilepsy is a chronic progressive disease manifested by repeated paroxysmal disorders of consciousness and seizures, as well as increasing emotional and mental changes.

Clinical diagnosis

The main clinical forms are: major seizures and minor epileptic seizures. Major seizures include prodrome, tonic and clonic phases, and the post-seizure period. An attack classically begins in a child with a scream (initial scream), followed by loss of consciousness (often to coma) and convulsions. The tonic phase of seizures lasts 10-20 seconds and is characterized by tonic tension of the facial muscles, limb extensors, and trunk muscles, while the jaws are tightly compressed, and the eyeballs tilt upward and to the side. The complexion is pale at the beginning, later it becomes reddish-cyanotic. Pupils are wide and do not react to light. There is no breathing. The clonic phase lasts from 30 seconds to several minutes and is manifested by short flexor contractions of various muscle groups of the trunk. In both phases of the convulsive syndrome, biting of the tongue and lips may occur.

In the future, seizures gradually decrease, muscles relax, breathing is restored, the patient is constipated, motionless, reflexes are suppressed, and often involuntary discharge of urine and feces. After 15-30 minutes, sleep occurs or the child regains consciousness, completely amnesizing the seizure.

Epileptic status is a condition in which there are continuous recurrent seizures, and in the period between seizures there is no complete recovery of consciousness. It always presents an urgent condition and is characterized by an increase in the depth of impaired consciousness with the formation of cerebral edema and the appearance of respiratory and hemodynamic disorders. The development of an epileptic status provokes the cessation or irregularity of anticonvulsant treatment, a sharp decrease in dosages of antiepileptic drugs, as well as concomitant diseases, especially acute infections, intoxications, traumatic brain injuries, etc.

Emergency care:

-Lay the patient on a flat surface (on the floor) and place a pillow or roller under his head; turn his head to one side and provide fresh air.

-Restore airway patency: clear the mouth and pharynx of mucus, insert a mouth expander or a spatula wrapped in a soft cloth to prevent biting of the tongue, lips and teeth damage.

-If the seizures continue for more than 3-5 minutes, inject a 0.5% solution of

seduxene (relanium) at a dose of 0.05 ml / kg (0.3 mg / kg) i.m. or into the muscles of the floor of the oral cavity.

-Upon resumption of seizures and epileptic status, provide access to the vein and inject 0.5% seduxene solution at a dose of ml / kg (0.3 mg / kg).

-Enter a 25% solution of magnesium sulfate at the rate of 1.0 ml / year of life, and for children under one year - 0.2 ml / kg i.m. or a solution of lasix 0.1-0.2 ml / kg (1-2 mg / kg) i.v. or i.m.

-If there is no effect, inject a 20% solution of sodium oxybutyrate 0.5 ml / kg (100 mg / kg) in a 10% glucose solution intravenously slowly (!) to avoid respiratory arrest.

Hospitalization after emergency care in a hospital with a neurological department, in case of epileptic status - in the intensive care unit.

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