A.A. Baranov[1], S.F. Bagnenko[2], L.S. Namazova-Baranova[1, 2, 3], Y.S. Alexandrovich[5], K.V. Pshenisnov[5], E.A. Alexeyeva[1], L.R. Selimzyanova[1, 2]
[1] Scientific Center of Children's Health, Moscow, Russian Federation
[2] Pavlov First Saint Petersburg State Medical University, Saint Petersburg, Russian Federation
[3] Sechenov First Moscow State Medical University, Moscow, Russian Federation
[4] Pirogov Russian National Medical Research University, Moscow, Russian Federation
[5] Saint Petersburg State Pediatric Medical University, Saint Petersburg, Russian Federation

Federal Clinical Recommendations on Emergency Medical Care Rendering to Children with Acute Intoxication

Author affiliation:
Liliya Robertovna Selimzyanova, PhD in Medicine, senior research fellow at the department of standardization and clinical pharmacology of the Scientific Center of Children's Health, assistant at the department of pediatrics and pediatric rheumatology at the Sechenov First Moscow State Medical University
Address: 2/1 Lomonosovskiy Av., Moscow 119991; tel.: +7 (495) 967-14-65; e-mail: selimzyanova@nczd.ru

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The article is dedicated to the issue of intoxication in children. Acute accidental intoxication appears to be especially relevant for pediatric practice. Drugs, various chemicals frequently used in everyday life and in farming, as well as animal poisons, including snake poisons, may have a toxic effect on children. Specialists of professional associations of physicians "Russian Society of Emergency Medicine" and pediatricians "Union of Pediatricians of Russia" formulated and briefly described the main causes of acute intoxication in children, clinical manifestations and the most significant laboratory indicators of toxic manifestations for various substances, as well as therapy principles and algorithms for such conditions in compliance with principles of the evidence-based medicine. The article presents pathognomonic symptoms and peculiarities of drug intoxication, provides a description of mediator symptoms of intoxication with various substances, as well as the symptoms that may indicate toxic effect. The article contains a description of principles of correction of vital body functions, measures for removing toxic substances from the body and information on the main antidotes. Special attention is given to the most frequent types of intoxication (with organic acids, lye, naphazoline, paracetamol, snake poisons [viper bite]). The article lists stage of medical care rendering to children suffering from acute intoxication and presents prognosis and further management of pediatric patients suffering from such conditions.
Keywords: *intoxication, toxic substances, poisons, children, emergency medicine.*

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METHODOLOGY

Methods used for collection/selection of evidence: search in electronic databases.

Description of the methods used for collection/selection of evidence:
the evidence base for the recommendations are publications from the Cochrane library, EMBASE and MEDLINE databases.

The methods used for the evaluation of quality and strength of evidence:

- consensus of specialists
- evaluation of science merit in accordance with the rating scheme (table 1):

Table 1.
Rating scheme for evaluation of the quality of evidence.

Levels of evidence	**Description**
1++	Meta-analyses of high quality, regular reviews of randomized controlled studies (RCS), or RCS with a very low risk of systematic errors
1+	Well conducted regular meta-analyses or RCS with a low risk of systematic errors
1-	Meta-analyses, systematic or RCS with a high risk of systematic errors
2++	High-quality systematic reviews of case-control or follow-up studies. High-quality reviews of case-control or follow-up studies with a very low risk of mixing effects and systematic errors and an average probability of a causal relationship.
2+	Well-conducted case-control or follow-up studies with an average risk of mixing effects and systematic errors and an average probability of a causal relationship.
2-	Case-control or follow-up studies with a high risk of mixing effects and systematic errors and an average probability of a causal relationship.
3	Non-analytical studies (for example: Descriptions of cases, series of cases)
4	Expert opinion

Methods used for analysis of evidence:

- Reviews of published meta-analyses;
- Systematic reviews with tables of evidence.

Description of the methods used for analysis of evidence:
During the selection of publications as potential sources of evidence, the methodology used in each study is reviewed to make sure it is valid. The result of review affects the level of evidence assigned to the publication, which in its turn affects the strength of recommendations.
In order to minimize potential errors, each study was assessed independently. Any differences in assessments were discussed by the complete group of authors. If they failed to achieve a consensus, an independent expert was involved.

Tables of evidence: filled by the authors of clinical recommendations.

Methods used for formulation of the recommendations: expert consensus.

Good Practice Points – GPPs

The recommended good practice is based on the clinical experience of the authors of recommendations.

Economic analysis

Cost analysis was not conducted nor were the publications on pharmacoeconomics analyzed.
Recommendation validation method

- External expert assessment
- Internal expert assessment

Description of recommendation validation method

The draft version of the present recommendations was reviewed by independent experts. Their primary task was to comment on how easily understandable is the interpretation of evidence on which the recommendations are based.
Any comments of the experts were thoroughly systematized and discussed by the members of the work group (the authors of recommendations). Every point was discussed separately.

Consultation and expert evaluation

The recommendation draft was reviewed by independent experts. Their primary task was to comment on the clarity and accuracy of the interpretation of evidence base underlying the recommendations.

The workgroup

For the final revision and quality control, the recommendations were again analyzed by the members of the work group who came to the conclusion that all the observations and comments of the experts were taken into consideration and the risk of systematic errors in the formulation of recommendations was minimized.

The basic recommendations

The strength of recommendations based on the relevant levels of evidence is cited in the text of recommendations (table 2).

Table 2.
Rating scheme for evaluation of the strength of recommendations.

Strength	Description
A	At least one meta-analysis, a systematic overview or RCS rated 1++, directly applicable to the target population and demonstrating sustainability of results. or an evidence group comprised by results of studies rated 1+ directly applicable to the target population and demonstrating overall sustainability of results
B	An evidence group comprised by results of studies rated 2+ directly applicable to the target population and demonstrating overall sustainability of results or Extrapolated evidence from studies rated 1++ or 1+
C	An evidence group comprised by results of studies rated 2+ directly applicable to the target population and demonstrating overall sustainability of results; or Extrapolated evidence from studies rated 2++
D	Evidence of level 3 or 4; or extrapolated evidence from studies rated 2++

DEFINITION

Poisoning (intoxication) is acute or chronic life-threatening condition evolving as a consequence of interaction of the human body and the poison. The poisoning can develop as a result of poison

coming from the external environment (exogenous poisons) or saturation with toxins secreted during dysfunction of organs and systems (endogenous poisons).
Poison is defined as substances of biological (animal or plant) and anthropogenic origin that when affecting living organisms including humans can cause intoxication: death or various impairments of biochemical, physiological, genetic, psychic and other processes and functions.

ICD-10 codes

T36 Poisoning by systemic antibiotics
T37 Poisoning by other systemic anti-infectives and antiparasitics
T38 Poisoning by hormones and their synthetic substitutes and antagonists, not elsewhere classified
T39 Poisoning by nonopioid analgesics, antipyretics and antirheumatics
T40 Poisoning by narcotics and psychodysleptics [hallucinogens]
T41 Poisoning by anesthetics and therapeutic gases
T42 Poisoning by antiepileptic, sedative- hypnotic and antiparkinsonism drugs
T43 Poisoning by psychotropic drugs, not elsewhere classified
T44 Poisoning by drugs primarily affecting the autonomic nervous system
T45 Poisoning by primarily systemic and hematological agents, not elsewhere classified
T46 Poisoning by agents primarily affecting the cardiovascular system
T47 Poisoning by agents primarily affecting the gastrointestinal system
T48 Poisoning by agents primarily acting on smooth and skeletal muscles and the respiratory system
T49 Poisoning by topical agents primarily affecting skin and mucous membrane and by ophthalmological, otorhinorlaryngological and dental drugs
T50 Poisoning by diuretics and other and unspecified drugs, medicaments and biological substances

CLASSIFICATION

The classification of poisonings is presented in table 3.

Table 3.
Classification of poisonings

With respect to the action period of the toxin	**With respect to the cause and place of emergence**	**With respect to severity**
1. Acute 2. Chronic 3. Subacute	1. Accidental 2. Deliberate (usually suicidal) 3. Industrial 4. Home	1. Mild 2. Medium 3. Severe 4. Very severe 5. Fatal

Acute accidental poisonings are the most frequent in pediatric practice. Chronic and subacute intoxications are very rare.
Acute poisonings are diseases of the chemical etiology caused by one-time exposure to a toxic dose of chemical substances capable of provoking disturbances of vital functions and development of life-threatening or critical conditions.
Drug poisoning in children is usually caused by the following drugs:
■ narcotic analgesics;
■ tricyclic antidepressants;
■ antihypertensive agents;
■ psychotropic agents (benzodiazepines);

■ digoxin;
■ iron preparations;
■ paracetamol;
■ potassium preparations;
■ anti-arrhythmic drugs (quinine, quinidine).

Usually drug poisonings are found in children from 1 to 6 years old and teenagers (14-18 years old).

FIRST MEDICAL AID DELIVERED BY THE AMBULANCE TEAM IN THE PREHOSPITAL STAGE

DIAGNOSTICS

Prehospital diagnostics of acute poisonings in children is based on four key points.
1. The anamnesis (the reliably established fact of exposure to a toxic substance or the absence of parents at the moment of deterioration of the child's condition).
2. Examination of the emergency scene.
3. Physical examination of the child.
4. Detection of specific symptoms and syndromes.

The peculiarities of clinical signs of drug poisoning are presented in table 4.

Table 4.
Clinical picture of drug poisoning

Drugs	**Pathognomonic symptoms and peculiarities of poisoning**
Indirect anticoagulants (warfarin, dicoumarol, rodenticide)	1. Maximum effect is observed 12-72 hours later. 2. Hemorrhagic diatheses. 3. Bleedings of different localization.
β-adrenergic blocking agents	1. Pronounced arterial hypotension, bradycardia, asystole. 2. β-blockers can promote asthma attacks in patients with spasmodic asthma. 3. CNS depression, cramps, hallucinations.
Barbiturates	1. Depression of consciousness (the degree of consciousness depression depends on the dose of the drug up to a deep coma with impairment of vital functions of the organism). 2. Bradycardia. 3. Arterial hypotension. 4. Hypersalivation, bronchorrhea. 5. Miosis (presence of photoreaction depends on the depth of coma).
Benzodiazepine	1. Maximum effect is observed 2-4 hours after exposure. 2. Ataxia, dysarthria, muscular hypotension. 3. Depression of consciousness (stun, sopor). 4. Changes in pupillary diameter or respiratory or cardiovascular dysfunction are not typical.
Digoxin (heart glycoside)	1. Arythmogenic syndrome: life-threatening heart rhythms, most often ventricular arrhythmia is observed. 2. Dyspeptic syndrome: Anorexia and vomiting of central origin, sometimes diarrhea. 3. Vision disorders: contraction of the pupils, color perception disorders (multicolored circles, yellow color).

Isoniazid (tuberculosis drug)	1. Symptoms of CNS affliction: psychosis, memory loss, ataxia, depression of consciousness, cramps, optic neuritis.
Clonidine	1. Maximum blood plasma concentration is observed 1.5-2.5 hours after exposure. 1. Depression of consciousness (the degree of consciousness depression depends on the dose of the drug). 2. Bradycardia. 3. Arterial hypotension. 5. Increase in the duration of QRS complex. 6. Atrioventricular blockades, early repolarization syndrome. 7. Miosis.
Xanthines (theophylline, aminophylline, teobromin, caffeine, teofedrin)	1. Different doses – from 17 to 300 mg/kg – can turn out to be lethal. 2. Teofedrin (teophylline + ephedrine) are especially dangerous. 3. Tachyarrhythmias (supraventricular tachycardia). 4. Consciousness depression, cramps, coma. 5. Hyperglycemia, hypokalemia, metabolic acidosis.
Methyl alcohol	1. Dyspeptic syndrome (nausea, vomiting). 2. Vision disorders (floaters, diplopia, sharp decrease in visual acuity, blindness 2-3 days after exposure). 3. Symptoms of alcohol poisoning (hyperemia of mucosae, thirst, muscle weakness etc.) 4. Mydriasis, weakened photoreaction. 5. Pronounced metabolic acidosis.
Methemoglobin formers (potassium permanganate, aniline dyes, nitrites, nitrobenzene)	1. Skin hyperemia is observed in initial stages. 2. Central skin cyanosis is observed when the concentration of methemoglobin exceeds 15 g/l. 3. Normal SpO2 readings. 4. Chocolate tint of blood. 5. Signs of respiratory failure depends on the degree of manifestation of methemoglobinemia (>20 g/l). 6. With methemoglobin concentration over 50%, depression of consciousness, heart rhythm disorders, pronounced signs of respiratory failure are observed.
Narcotic analgesics;	1. Atony coma. 2. Apnea. 3. Miosis 4. Extraocular paralysis.
Paracetamol	1. In paracetamol overdose, symptoms of hepatobiliary system lesion are observed in the first place. 2. It is not possible to estimate the severity of paracetamol poisoning based on the initial symptoms! 3. In the first 12-24 hours, nausea, vomitins, profuse sweating are observed. 3. 24-36 hours after exposure, enlargement and soreness of the liver, jaundice, hyperbilirubinemia, hyperammonemia, extension of prothrombin time are observed. 4. Activity of aminotransferases in serum peaks on the 3-4th day after the poisoning and, unless there is liver failure, returns to the normal condition within a week.

Iron preparations	1. Any iron preparations are highly toxic! 2. Signs of poisoning are observed 30 minutes to 2 hours after ingestion of iron-containing drugs. 3. The main symptoms are signs of gastrointestinal lesion: Sharp abdomen pains, nausea, hematemesis, diarrhea, fecal blood. 4. 6-24 hours later fever, metabolic acidosis, and acute liver failure can develop. 5. In some bad cases, pronounced disorders of hemodynamics (shock) are observed, acute cerebral failure (anxiety, convulsions, coma) can develop.
Piperazine drugs (used for enterobiasis and ascariasis treatment)	1. All the piperazine drugs are low toxic. 2. Two poisoning options are described: with a CNS depression and skin lesion, possibly a combination of symptoms CNS depression: anxiety, fear, headache, nausea, sleepiness, motor dysfunction, vomiting, meningeal symptoms. In some bad cases, vision disorders and consciousness depression, up to coma, ore observed. Muscular hypotension is replaced by generalized clonicotonic cramps. Disorders of hemodynamics (arterial hypotension, tachycardia) can be observed. Skin lesion: urticarial rash (typical).
Salicylates	1. Signs of poisoning are observed 2-6 hours after exposure. 2. Depression of consciousness. 3. Hyperthermia. 4. Dehydration. 5. In initial stages of a poisoning tachypnea (respiratory alkalosis) is observed. 6. Metabolic acidosis, especially in younger children. 7. Hemorrhagic syndrome. 8. Hyperglycemia developing into hypoglycemia.
Tricyclic antidepressants	1. Symptoms of a poisoning develop within 4 hours after exposure. 2. Antichoclinergic syndrome: Mydriasis, dryness of the skin and mucosae, tachycardia, enteroplegia, urine retention. Life-threatening heart rhythm disturbances: Sinus tachycardia or brachycardia, atrioventricular blockade, ventricular fibrillation. 4. Depression of consciousness, sometimes seizure-type clonicotonic cramps.
Phenothiazines (amineazine, tisercin, triftazine, thioridazine)	1. Depression of consciousness. 2. Disorders of hemodynamics and respiration.
Organophosphorus compounds, scetylcholinesterase inhibitors	1. Manifestations of cholinergic syndrome: Miosis, pronounced humidity of the skin, hypersalivation, bronchorrhea, bradycardia, involuntary urination and defecation. 2. Severe poisonings can cause CNS depression, convulsive syndrome.
Quinine	1. Sharp reduction of hearing and visual acuity. 2. Fever, delirium. 3. Hemolysis of erythrocytes and aleukia.
Quinidine (anti-arrhythmic drug)	Life-threatening heart rhythm disorders (ventricular extrasystoles, ventricular fibrillation) up to a complete cardiac arrest.

Chloralhydrate (analogue of ethanol)	Has a powerful sedative effect (sleep during 4-5 hours).
Cyanides	1. Central skin cyanosis. 2. Depression of consciousness, convulsive syndrome. 3. Progression of respiratory and cardiovascular failure.
Ethyl alcohol, alcohol surrogates	1. Depression of consciousness of various degrees. 2. Facial skin hyperemia. 3. Hypothermia. 4. Miosis, horizontal nystagmus. 5. Respiration and cardiovascular activity depression is observed with severe poisonings.
Ethylene glycol	1. Symptoms of alcoholic intoxication. 2. 5-8 hours later, expressed dyspeptic syndrome (stomachaches, vomiting, diarrhea) is observed. 3. Skin dryness and hyperemia. 4. Mydriasis. 3. Hyperthermia. 6. Metabolic acidosis. 7. Psychomotor agitation evolving into consciousness depression.

Given that the majority of drugs affect the mediator systems of an organism, the key objectives of the ambulance team are to reveal the primary mediator syndrome (tab. 5) underlying the clinical picture of poisoning and conduct targeted pathogenetic therapy.

Table 5.
Mediator syndrome characteristics in acute poisoning in children.

Syndrome	Drugs	Heart rate	Blood pressure	Pupil	Skin humidity	Peristalsis
Chronopositive syndromes						
Anticholinergic	Atropine, Diphenhydramine	↑	↑	↑↑	↓↓	↓↓
Adrenergic	An aminophylline, inhibitors of monoamineoxidase	↑↑	↑↑↑	↑↑↑	↑-	↓-
α-adrenolytic	Amineazine	↑↑	↓↓	↑↑	↑↓	↓↓
Chrononegative syndromes						
Chnolinergic	Cholinomimetics, heart glycosides, barbiturates	↓	↓	↓↓↓	↑↓	↑↑
B-adrenoblocking	β-blockers	↓↓	↓	-	↑	↑-
Sympatholytic	clonidine, verapamil, cordarone, heroin	↓↓↓	↓↓↓	↓	↓	↓-

In the stage of primary examination it is extremely important to evaluate the level of consciousness and effectiveness of unassisted breathing and hemodynamics, because curing vital disturbances is the key objective of the ambulance team. It is also necessary to check the condition oral cavity mucosa and skin around the mouth for stridor symptoms (a burn on the upper airways or larynx is possible). The paramedics should find out if any other children close to the poisoned child experience the same syndromes.
In order to determine the cause of poisoning, the child's clothes are to be thoroughly examined (vomit, its color and odor, powders or tablets that can be found in the pockets, odor of breath or

clothes in case of flavoring poisonings). The doctor called to the child with an assumption of poisoning shall collect vomit, if possible (not stomach washing water) and send for toxicological examination along with the assumed poisons (if available).
Physical examination data can also help in determining the poisoning etiology (table 6).
Differential diagnostics of acute poisoning in children is carried out in the first place with a neuroinfection (meningitis, encephalitis), hypo- and hyperglycemia, severe brain injury.

Table 6.
Pathognomonic symptoms of poisoning

Symptoms	**Typical cases**
Odor	Kerosene, arsenic, phosphorus, organophosphorus compounds (garlic odor), camphor, chloralhydrate, alcohol
Sweatiness	Increase: Paracetamol, organophosphorus compounds, cyanides (bitter almond), and salicylates. Reduction: Atropine
Fever	Salicylates, anticholinergic drugs, kerosene, camphor
Hypothermia	Opiates, barbiturates
Coma	Barbiturates, opiates, diazepam, salicylates, organophosphorus compounds, SO, kerosene, anticonvulsants, tricyclic antidepressants
Delirium	Salicylates, anti-histamine drugs, barbiturates
Ataxia	Piperazine, kerosene, anticholinergic drugs, phenothiazines, anti-histamine drugs, organochlorines
Abnormal motions	Phenothiazines
Cramps	Organophosphorus compounds, organochlorines, phenothiazines, phenol, camphor, amphetamine, atropine, kerosene, anti-histamine drugs, aminophylline, benzylbenzoate, benzylsalicylates, benzylstrychnine, lead
Pupils	Miosis: Opiates, organophosphorus compounds, chloralhydrate, an early stage of barbiturates poisoning. Mydriasis: Atropine, anti-histamine drugs, sympathomimetics
Mouth burns	Caustic substances, iodine
Cardiac arrhythmias	A digitalis, phenol, phenothiazines, theophylline, kerosene, SO, tricyclic antidepressants
Tachycardia	Atropine, theophylline
Bradycardia	Digitalis, β-blockers, quinidine
Gastrointestinal	Plant products (castor oil), iron, camphor, naphthalene, paracetamol, salicylates, food poisons; Haemorrhagic gastroenteritis – iron, salicylates, phenol, arsenic; Fluorescent vomit – phosphorus
Paralytic ileus	Opiates, anticholinergic drugs
Respiratory	Hyperventilation: Salicylates, atropine. Hypoventilation: Barbiturates, opiates. Distress: Kerosene. Lung edema and lipoid pneumonia: Petroleum products
Haematuria	Naphthalene, snake venom (bite)
Hypotension	Iron, barbiturates, anticholinergic drugs, phenothiazines, opiates, phosphorus
Anemia (paleness)	Iron, naphthalene, lead, snake venom (bite)
Jaundice	Arsenic, iron, naphthalene, phosphorus, paracetamol

TREATMENT

In the prehospital stage treatment is administered in four main directions:
1. Adjustment of vital disorders
2. Elimination of unabsorbed poison
3. Elimination of absorbed poison
4. Administration of antidotes

1. Adjustment of vital disorders (A)

With consciousness depression leading to coma of any severity intubation of trachea and artificial lung ventilation are obligatory. If intubation of trachea is impossible, a larynx mask or an airway (as a last resort) is installed. If a larynx mask is not available, artificial lung ventilation is carried out by means of a mask and a self-inflating bag (like AMBU).
In case of arterial hypotension and shock anti-shock therapy is conducted including initial volemic load [0.9% sodium chloride solution in 20 ml/kg or colloids (gelatin drugs, hydroxyethyl starches) in the same dose; vasoconstrictors if necessary (dopamine, epinephrine)].
If effective independent breathing is impossible, artificial lung ventilation is obligatory.

2. Elimination of unabsorbed poison

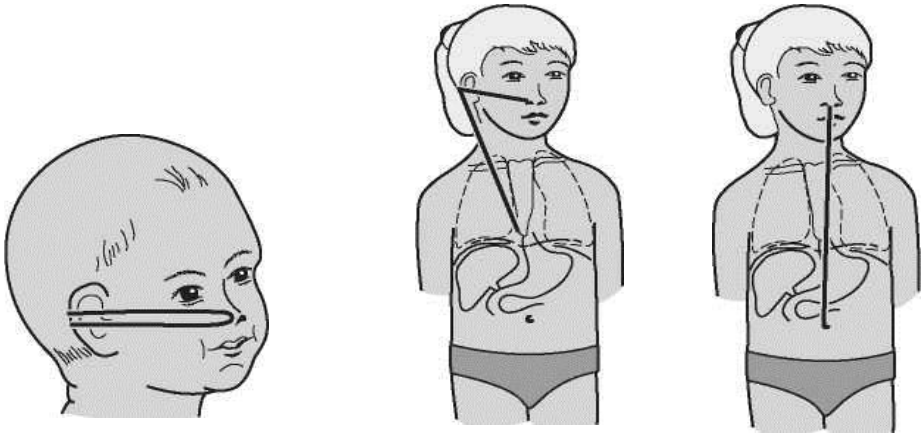
For elimination of unabsorbed poison, *stomach lavage, stimulation of vomiting, and prescription of sorbents* are used in nearly 100 per cent of cases.
Stimulation of vomiting (D). One is to remember that stimulation of vomiting by any means is most effective immediately after poisoning and ineffective after 1 hour, therefore vomiting as a way of eliminating the unabsorbed poison should be used immediately after the exposure of the child to a toxicogene.
Stimulation of vomiting is strongly counter-indicative in cases of consciousness depression and poisoning with substances with burning fumes [petroleum products, pesticides (soluble with gasoline), strong acids, alkalis (for example, laundry bleaches, accumulator acid, etc.)].
Vomiting can be stimulated by pushing on the root of the tongue or back of the throat with a spatula or a spoon.
Using vomiting as poisoning therapy one should remember that vomitives have a very high risk of side effects, the worst of which are:
■ uncontrollable vomiting;
■ aspiration pneumonia (even while preserving consciousness!);
■ deferred effect of drugs (delay of vomiting until loss of consciousness which can often lead to aspiration).

Stomach lavage (C). Both stomach lavage and administration of vomitives are only effective immediately after the poisoning and ineffective 1 hour after.
Stomach lavage is counter-indicative in case of consciousness depression (only possible after intubation of trachea) and poisoning with substances with burning fumes (petroleum products, strong acids, alkali). One-time volume of liquid for stomach lavage should be 10 ml of warm water per kg. The procedure is repeated a maximum of three times before until the lavage water is clear. The total volume of liquid for stomach lavage must not exceed 150 ml/kg. The volume of drained liquid should approximately correspond to the intake volume.
The tube diameter should be 24-28 Charriere (8-9 mm). The depth of insertion of lavage tubes in children with respect to their age is presented in table 7 and pic. 1.

Table 7.
Lavage tube size

Child's age	Tube diameter, mm	Depth of insertion, cm
Newborn	3	20

Up to 3 months	4	25
Up to 1 year	6	28
2-5 years	10	30
Over 5 years	12	35
9-14 years	15	40-50



Pic. 1. Determination of the depth of insertion of a lavage tube in children of different age:
a - newborns, infants ("ear - nose tip - ear"); b, c - older children ("ear - nose tip - xypnoid"; "nose tip - navel").

Gastric intubation methodology

1. Patient position: lying or sitting (depending on the child's age and reaction to the manipulation).
2. The head need to be bent forward a bit irrespective of the position. Ask the child to breathe deeply.
3. Moisten the sterile tube with neutral liquid: water or tea. It is preferable not to use oil!
4. Insert the tube through nose or mouth quickly but cautiously. During the insertion, the patient is asked to swallow.
5. Insert the tube to the necessary depth (pic. 1).
6. Secure the tube and check its placement. If the tube is placed correctly, gastric secretions are coming out through the tube. For placement check, air introduction can also be used. Quickly introduce 5-10 ml of air into the tube and simultaneously conduct auscultation in the stomach area. If the tube is positioned into the stomach, at the moment of air introduction a loud noise can be heard. The mouth cavity has to be examined: the tube might roll up!
7. Permanently attach the tube with adhesive bandage. If the tube is inserted through the mouth, it is attached in the corner of the mouth. The plaster is attached to a cheek. If the tube is inserted through the nose, it is attached to the back of the nose.

Sorbents

In child poisonings, activated charcoal and drugs based on lignites (hydrolyzed lignin) are usually used.

Activated charcoal

Activated charcoal is universal sorbent, but it is, too, only effective immediately after the exposure and ineffective 1 hour after the poisoning. One is also to remember in some cases it is ineffective (table 8).

Table 8.
Activity of activated charcoal as a universal antidote

Effective	Ineffective
Chlorpropamide Cyclosporin Digoxin Methotrexate Phenobarbital Phenytoin Salicylates Theophylline Tricyclic antidepressants	Acids Alkali Boric acid Bromides Cyanides Ethanol Iron preparations Iodides Lithium carbonate Salts of heavy metals

The single starting dose of activated charcoal is 1-2 g/kg of body mass with the maintenance dose of 0.25-0.5 g/kg every 4-6-12-24 hours. It is prescribed before the stimulation of vomiting, stomach lavage, aspiration.
Activated charcoal is counter-indicated in case of gastrointestinal obstruction and oppression consciousness without intubation of the trachea, as well as in case of swallowing of caustic substances.
One is to note that prescription of activated charcoal is not obligatory in the prehospital stage (C, 2+).

3. ***Elimination of absorbed poison***

In the prehospital stage, the only therapeutic method for the elimination of absorbed poison is the infusion therapy administered at 10 ml/kg per hour (C). Balanced crystalloid solutions are used for the infusion. Meglumine sodium succinate (contraindicated in infants!) is the optimal solution for infusion in case of poisoning in children (D).

4***. Specific therapy of poisonings (antidotes) (B)***

The essential antidotes and their doses with respect to the age of the child are presented in table 9.

CHARACTERISTICS OF TREATMENT OF THE WIDESPREAD POISONINGS IN CHILDREN IN THE PREHOSPITAL STAGE

The most widespread poisoning substances for the children are organic acids (acetic, sorrel acid), caustic alkalis (household chemicals), potassium permanganate, naphazoline, and paracetamol. Snake (viper) bites are often encountered in the summer.
Table 9.
Essential antidotes used for treatment of acute poisonings in children

Toxic substance	Antidote	Method of administration
Narcotics	Naloxone	Administered intravenously Starting dose for teenagers: 0.4 mg (1 ml). For younger children: 0.01 mg/kg. If no effect is observed, 0.3 mg/kg more is administered after 2 minutes.

Warfarin, dicumarol, rodenticides	Menadione (vitamin K)	Administered intravenously at 2-5 mg/kg. Taking into consideration the intense metabolism of the drug, repeated injections are justified.
Organophosphorus compounds, acetylcholineesterase inhibitors	Atropine	Administered intravenously in the starting dose of 0.05 mg/kg (1-4mg). Every 2-5 minutes, the admission of up to 2 mg is repeated until effective.
Methemoglobin-producing agents	Methylene blue	Administered intravenously as 1 per cent solution in the dose of 1-2 mg per kg. Repeated in the same dose if necessary. In nursing infants, the dose should not exceed 4 mg/kg.
Paracetamol, toxic byproducts (lethal synthesis)	Acetylcysteine (not later than 36 hours after poisoning)	Patients with body mass below 20 kg are administered acetylcysteine intravenously with drop infusion. The starting dose is 150 mg/kg in 3 ml/kg of 5% of glucose solution during 15 minutes. Subsequent administration is in the dose of 50 ml/kg in 7 ml/kg of 5 per cent glucose solution during 40 minutes and then 100 mg/kg in 14 ml of 5 per cent dextrose solution over 16 hours. For larger children, the volume of dextrose solution can be increased. If there are signs of a toxic liver lesion, the infusion of acetylcysteine should be continued after 20 hours.
Clonidine	Metoclopramide	Loading dose: 0.5 mg/kg. Maintenance dose: 0.25 mg/kg over 4-5 hours
Hydrazine derivatives [isoniazid, Gyromitra mushrooms (false morel) containing girometrine], ethylene glycol	Pyridoxine (vitamin B6)	1gram of pyridoxine per 1 g of hydrazine (70-375 mg/kg). For ethylene glycol poisoning, the pyridoxine dose 50 mg every 6 hours until the acidosis is eliminated
Benzodiazepine	Flumazenil	Starting dose: 0.05-0.1 mg/kg Daily dose: 1-10 mg. N.B.! The effect of flumazenil develops 1-2 min after administration and continues for 2-5 hours
Iron preparations	Deferoxamine	Starting dose: 15 mg (per kg per hour) intravenously with subsequent reduction in 4-6 hours (maximum dose: 80 mg/kg per day, not more than 6 g/day). The infusion is stopped when the patient's condition is stabilized and the level of iron in blood serum does not exceed 60 mcM/l

Organic acid poisonings

The clinical picture emerges from symptoms of a chemical burn of the oropharynx and the esophagus, in case of a pronounced edema of soft tissues of the oropharynx obstructive respiratory failure can develop. In some bad cases, gastric bleeding with elements of shock can be observed.

Symptoms:

1. Burn of the oral cavity and esophagus
2. Pronounced pain syndrome.
3. Hypersalivation.
4. Obstructive respiratory failure

First medical aid in the prehospital stage

1. Resuscitation for disturbance of vital functions (provision of secure airway, adequate respiration, and circulation of blood).
2. Provision of venous access.
3. Analgesia (in case of pronounced pain, narcotic analgesics in age-dependent doses).
4. Tube water lavage of the stomach (after the preliminary anesthesia):

■ tubeless lavage is strictly counter-indicated;
■ alkali solution lavage is strictly counter-indicated;
■ Presence of blood in lavage waters is not a contraindication to the continuation of the operation.

5. Prednisolone, 5 mg/kg, intravenous bolus administration.
6. Infusion of 0.9 per cent solution of sodium chloride in the dose of 10 ml/kg over an hour.
7. Monitoring of vital functions.

Caustic alkali poisoning

Like in organic acid poisonings, the clinical picture emerges from symptoms of a chemical burn of the oropharynx and the esophagus, in case of a pronounced edema of soft tissues of the oropharynx obstructive respiratory failure can develop. In some bad cases, gastric bleeding with elements of shock can be observed.

Symptoms:

1. Burn of the oral cavity and esophagus
2. Pronounced pain syndrome.
3. Hypersalivation.
4. Obstructive respiratory failure

First medical aid in the prehospital stage

1. Resuscitation for disturbance of vital functions (provision of secure airway, adequate respiration, and circulation of blood).
2. Provision of venous access.
3. Analgesia (in case of pronounced pain, narcotic analgesics in age-dependent doses).
4. Tube water lavage of the stomach (after the preliminary anesthesia):

■ tubeless lavage is strictly counter-indicated;
■ alkali solution lavage is strictly counter-indicated;
■ Presence of blood in lavage waters is not a contraindication to the continuation of the operation.

5. Prednisolone, 5 mg/kg, intravenous bolus administration.

6. Infusion of 0.9 per cent solution of sodium chloride in the dose of 10 ml/kg over an hour.
7. Monitoring of vital functions.

Potassium permanganate poisoning

Symptoms

1. Burn of the oral cavity and esophagus.
2. Pronounced pain syndrome.
3. Hypersalivation.
4. Obstructive respiratory failure.
5. Blood hypoxia.

First medical aid in the prehospital stage

1. Resuscitation for disturbance of vital functions (provision of secure airway, adequate respiration, and circulation of blood).
2. Elimination of the undissolved crystals.
3. Washing the oral cavity with a 5 per cent solution of ascorbic acid or with lemon juice.
4. Provision of venous access.
5. Adequate analgesia (in case of pronounced pain, narcotic analgesics in age-dependent doses).
6. Tube water lavage of the stomach (after the preliminary anesthesia).
7. Prednisolone, 5 mg/kg, intravenous bolus administration.
8. Prescription of antidotes:

■ Ascorbic acid – intravenous bolus administration in age-dependent doses (table 10). The preparation is diluted in glucose solution in advance. Should not be prescribed to infants younger than 6 months!

Table 10.
Age-dependent dose of ascorbic acid

Age	Dose, ml
7-12 months	0.75
1-3 years	1-2
4-6 years	2-3
7-14 years	3-6

Naphazoline poisoning

Symptoms:

1. Consciousness depression of varying degree of severity.
2. Pronounced paleness of the skin.
3. Hypothermia.
4. Sinus bradyarrhythmia.

First medical aid in the prehospital stage

1. Resuscitation for disturbance of vital functions (provision of secure airway, adequate respiration, and circulation of blood).
2. Provision of venous access.
3. Infusion of 0.9 per cent solution of sodium chloride in the dose of 10 ml/kg over an hour.
4. Monitoring of vital functions.

Paracetamol poisoning

Symptoms:

1. Toxic liver lesion.
2. In the first 12-24 hours, nausea, vomiting, profuse sweating are observed.
3. 24-36 hours after exposure, enlargement and soreness of the liver, jaundice, hyperbilirubinemia, hyperammonemia, extension of prothrombin time are observed. 4. Activity of aminotransferases in serum peaks on the 3-4th day after the poisoning and, unless there is liver failure, returns to the normal condition within a week. Paracetamol metabolites have hepatotoxic effects, too.
4. It is not possible to estimate the severity of paracetamol poisoning based on the initial symptoms. Liver disturbance is possible with a single exposure of a 2-3 year old child to 3g of paracetamol (about 150 mg/kg). The toxic dose in teenagers exceeds 8 g.

First medical aid in the prehospital stage

1. Resuscitation for disturbance of vital functions (provision of secure airway, adequate respiration, and circulation of blood).
2. Provision of venous access.
3. Tube water lavage of the stomach (after the preliminary anesthesia).
4. Administration of activated charcoal (in the first 4 hours after the poisoning!).
5. In case of severe paracetamol poisoning (for instance, when the amount of paracetamol taken is over 150 mg/kg and the concentration of paracetamol in blood serum remains toxic after 4 hours) an antidote, acetylcysteine is used (table 9). Inducers of enzyme synthesis (phenobarbital) are contraindicated.
6. Infusion of 0.9 per cent solution of sodium chloride in the dose of 10 ml/kg over an hour.
7. Monitoring of vital functions.

Snake venom poisoning (viper bite)

Symptoms:

1. Sharp pain in the place of the bite.
2. A dynamically growing pronounced edema of soft tissues, lymphostasis.
3. Imbibition of the skin and cellular subcutaneous adipose tissue in blood.
4. Circulatory failure manifestations.

First medical aid in the prehospital stage

1. Resuscitation for disturbance of vital functions (provision of secure airway, adequate respiration, and circulation of blood).
2. Antiseptic solution treatment of the bite.
3. Provision of venous access.
4. Adequate analgesia (in case of pronounced pain, narcotic analgesics in age-dependent doses).
5. Prednisolone, 5 mg/kg, intravenous bolus administration.
6. Infusion of 0.9 per cent solution of sodium chloride in the dose of 10 ml/kg over 30 minutes.
7. Prescription of anti-histamine drugs in age-dependent doses.
8. Prescription of intravenous bolus administration of 100 mg/kg of calcium gluconate.

9. Immobilization and elevated position of the limb.

Table 11.
Doses of anti-histamine drugs in children administered parenterally

Drug	Dose
Chloropyramine, 2% solution (**Suprastin**)	1 month to 1 year: 5 mg (0.25 ml). 1-6 years: 10 mg (0.5 ml) Older than 6 years: 10-20 mg (0.5-1.0 ml) The maximum daily dose is 2 mg/kg
Clemastine	0.0125 mg/kg

DONT'S

1. Using the E.A.Moshkin modification of lavage tube in children (strictly counter-indicated!).
2. Using a volume of lavage liquid larger than 150 ml/kg (very high risk of hypoosmotic hyperhydration and brain edema!).

FURTHER CASE MANAGEMENT, INDICATIONS FOR RESIDENTIAL TREATMENT

Indications for residential treatment in children with any acute poisonings.
1. Any drug poisonings.
2. Consciousness depression of any degree of severity.
3. Disorders of hemodynamics and respiration.
4. Suicide attempts.
5. Assumption of airway, esophagus, or stomach burn.

PROGNOSIS

The prognosis is favorable in most cases of acute poisonings in children.

FIRST MEDICAL AID IN THE HOSPITAL STAGE IN THE INPATIENT UNIT OF THE EMERGENCY DEPARTMENT

DIAGNOSTICS

In conditions of an inpatient unit of the emergency health care center, diagnostics of acute poisonings in children is based on the same principals as in the prehospital stage. The main difference is conducting laboratory and toxicological surveys (clinical and biochemical blood tests, coagulation profile, global urine analysis, toxicological urine and blood survey) allowing to reveal signs of lesion of internal organs and establish the actual cause of poisoning.

TREATMENT

The treatment is based on the correction of the observed disorders of vital functions (artificial ventilation of lungs, anti-shock therapy) and use of specific antidotes if available.
In cases of *ethanol poisoning* in the conditions of an inpatient unit of the emergency health care center the following operations are carried out.
1. Stomach lavage (if not yet performed in the prehospital stage).
2. Determination of blood glucose concentration.
3. In case of hypoglycemia, intravenous bolus administration of 0.5 g/kg of 40 per cent glucose solution.

4. Chemical toxicological survey of ethanol content in biological matrices.
5. Infusion therapy in physiologically required amount.
6. Infusion of 10 ml/kg of meglumine sodium succinate.
7. Monitoring of vital functions.

DONT'S

Release the patient from the hospital within one day of the poisoning, because the risk of late complications is very high.

FURTHER CASE MANAGEMENT

Indications for admission to the specialized department of a hospital, short-term unit of the inpatient department of the emergency healthcare service, or outpatient treatment in the place of residence.

- All the children with acute poisonings are admitted to toxicology units or, if there are no such units, to somatic pediatric units of hospitals.
- In life-threatening or critical cases, the patients are admitted to resuscitation and intensive care units.
- Only teenagers with acute ethanol poisonings without vital function disorders can be admitted to inpatient departments of the emergency healthcare service.

PROGNOSIS

The prognosis is favorable in most cases.

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Conflict of interest

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