Ministry of Health of Belarus
Gomel State Medical University

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

Head of the department, Y. M. Cherniakova

TOPIC: "Especially of intensive care on different poisoning "

Educational and methodical development for students
4th year medical faculty

Author:

Assistant L A Alekseeva

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

1. Background.
2. The purpose of training (skills and knowledge).
3. Basic sections.
4. Suggested Reading.
5. Questions for self-preparation.
6. Topics UIRS.
7. Teaching tools for organization of independent work of students
8. Training Material
9. Self-study.
10. Clinical problems and test control.

Relevance of the topic

In practice, doctors often have to deal with acute domestic poisoning, which often occur as a result of accidental and sometimes intentional taking chemicals with sufficiently high activity. In addition, the possibility of poisoning people at work in the production and use of toxic substances, as well as the use of plants for food, the bite of poisonous animals. On how quickly, effectively and consistently will be given medical aid to the victim, usually depends on the outcome of poisoning. Today's actions in most cases provide a life, even a few were poisoned with lethal doses, late as irrational and ineffective assistance, in less severe cases can develop serious complications. At the same time, the effectiveness of urgent measures in case of poisoning, in turn, depends on how quickly the doctor determines the nature of the toxic agent and the pathogenesis of major disorders, apply a specific antidote, and allocate syndromes that are most important pathogenetic significance, allowing to choose the means of pathogenetic therapy.

Purpose of the lesson

Development of clinical symptoms and the principles of intensive care, using non-specific methods and products - antidotes, acute and chronic poisoning with organophosphorus and organochlorine means alcohol and its surrogates, hypnotics and sedatives, cauterizing liquids, carbon monoxide, poisons of biological origin.

The student should know:

- features of the pathogenesis of acute and chronic intoxication with poisons of different origin;
- Clinical features of poisoning by various poisons;
- methods of diagnosis in different poisoning;
- the use of means and methods of non-specific therapy, and medications - antidotes for poisoning by various poisons;
- pharmacokinetic and pharmacodynamic characteristics of drugs specific treatment of poisonings.

The student should be able to:

- diagnose acute alcohol poisoning and its surrogates, hypnotics and sedatives, phosphororganic compounds, chlorinated hydrocarbons, acetic acid, cauterizing liquids, carbon monoxide, mushrooms;
- to evaluate the severity of poisoning by various poisons;
- justify the tactics at different poisoning according to their circumstances;

• provide emergency assistance in the event of vital signs;
• Conduct a full range of therapeutic measures in acute poisoning by various poisons, poisonous animals bite.

Sections studied before and needed for the session

• basic pharmacology of drugs
• biochemistry of carbohydrate, protein and lipid metabolism
• pathophysiology and physiology of the central nervous system, cardiovascular system, respiratory and excretory systems
• clinic, diagnosis, treatment of respiratory, cardiovascular, renal, hepatic failure

Recommended Reading

Textbooks pathophysiology, pharmacology, biochemistry, internal medicine for medical students.

Suggested Reading on lessons

Main Reading

1. Dale, OA Anaesthesia and Intensive Care. / O. Valley. - M., 1998. - S. 508-574.

Further Reading

1. Epiphany, VF Clinical diagnosis and emergency treatment of acute poisoning. / VF Epiphany, IF Epiphany. - M., 2002. - S. 3-29.
2. Malyshev, V. Intensive care. Resuscitation. First aid. / VD Malyshev. - M., 2000. - S. 437-444.
3. Kattsung, Bertram G. Basic and Clinical Pharmacology: in 2 vols 2. / Bertram G. Kattsung. - M., 2000. - S. 487-528.
4. Luzhniki, EA Acute poisoning. / EA Luzhniki. - M., 1999. - S. 5-241.
5. Sumin, SA Emergency conditions. / SA Sumin. - M., 2004. - S. 428-436.
6. Intensive therapy threatening conditions / VA Koryachkin [and others] Ed. Ed. VA Koryachkina - Saint-Petersburg, Medical Publishing, 2002. - 283 p.
7. Petrov, VI Drugs and Poisons: poisonous animals and plants / VI Petrov, T. Revyako - M: Literature, 1997. - 275 p.
8. Internet resources.

Questions for self-control. Questions on basic knowledge

1. Classification of poisons. Types of poisoning.
2. Toxic damage of the CNS, cardiovascular, respiratory and other systems.
3. Pharmacology of sedatives and hypnotics.
4. Using the WCF and organochlorines in the economy and as a means of warfare (ERD).
5. Cellular respiration, aerobic and anaerobic metabolism, oxygen tension in arterial blood. Transport of respiratory gases in the blood, the oxyhemoglobin dissociation curve.
6. Poisons of animal and vegetable origin, found in the Republic of Belarus

Questions about the topic studied

1. Alcohol poisoning and its surrogates. Pathophysiology, clinical features, diagnosis.

2. Intensive management of alcohol poisoning and its surrogates.
3. Pathophysiology, clinical manifestations, diagnosis and intensive treatment of poisoning hypnotics and sedatives.
4. Pathophysiology, clinical features, diagnosis of poisoning by organophosphorus compounds (OPC) and chlorinated hydrocarbons, intensive care.
5. Pathophysiology, clinical manifestations, diagnosis and intensive treatment of poisoning cauterizing liquids.
6. Pathophysiology, clinical manifestations, diagnosis and intensive treatment of carbon monoxide poisoning.
7. Pathophysiology, clinical manifestations, diagnosis and intensive therapy of mushroom poisoning.
8. Pathogenesis and clinical features of bites of poisonous snakes and insects. Emergency and Intensive Care.

Topics UIRS

1. Dioxins: sources of origin, pathogenesis, clinic.
2. The course and intensive care in cases of poisoning with strychnine, brilliant green, nitrites and nitrates, aniline dyes, sulfonamides, potassium permanganate, TNT, TNT.
3. Poisoning poisonous plant
4. The history of the use of biological toxins (from Ancient China and India to 21c.).

Teaching tools for organization of independent work of students

1. Computer database.
2. Objectives, test control.
3. Thematic sick.
4. Patient records.
5. Bank jobs for self-study.

Training Material

Alcohol poisoning.

Ethanol is part of all alcoholic beverages. The lethal dose of ethanol is 96 ° from 4g/kg to 12g/kg body weight (approximately 700-1000ml of vodka with no tolerance). Alcoholic coma occurs when the blood alcohol 3 g / l and above, the death of 5-6g / L and above. But it is impossible to judge the degree of severity only by the concentration of alcohol in the blood. The severity of acute alcohol poisoning depends on the amount of alcohol taken, indie ¬ vidual response, age of the victim, psychogenic and physical factors of the environment, etc. Poisoning usually have the character of everyday, casual, with the purpose of intoxication.

Pathogenesis. The most important is knowledge of the following provisions.

1. Ethanol has a psychotropic and narcotic effects on the central nervous system (CNS), and the products of its life (acetaldehyde and acetic acid, formed under the influence of the enzyme alcohol dehydrogenase (ADH), have a toxic effect on the entire body.
2. The rate of ethanol oxidation in the liver to 6-hour 7r

(ADH Ethanol → → → Acetaldehyde Acetic acid → $H_2O + CO_2$)
3. 90% ethanol was adopted subject to oxidation in the liver, 10% excreted unchanged in the urine and through the lungs.
4. About 2% ethanol oxidation in muscle enzyme catalase.
This information leads to an important practical conclusion that the paramount importance of the functional state of the liver in cases of poisoning with ethanol.
Clinic. The degree of acute alcohol intoxication varies widely - from mild to severe, the most formidable of which is the manifestation of alcoholic coma with impaired respiratory function and development of the collapse. Admission to hospital patients to be only in cases of severe and fatal poisoning. The clinical picture of alcoholic coma are three successively developing stage: above someone who moderate and deep coma.
For surface coma characteristic disturbance of the cortical-subcortical function with preservation of tendon reflexes, pain sensitivity. Severe disorders of respiration and circulation are observed.
At moderate coma consciousness is absent, sharply depressed tendon, corneal, pupillary, pharyngeal, and cough reflexes, eliminated pain sensitivity. Breathing shallow, weakened, auscultation listened wet large bubbling rale. Possible as a result of asphyxia the tongue, bronhorei, aspiration of mucus and vomit. Revealed tachycardia, sometimes a moderate increase in blood pressure.
With a deep coma has been a sharp narrowing of the pupils, their lack of reaction to light. Corneal, pharyngeal, tendon reflexes are absent, there may be pathological reflexes of muscle weakness. Pale skin, cyanotic color with marked acrocyanosis. The body temperature is lowered. Often have severe disorders of respiratory function, frequent different aspiration-obstructive complications. In some rare cases a surface (up to 6-10 per minute) breathing, may be in breach of its rhythm by type Cheina - Stokes complete airway. A marked tachycardia, voiceless heart tones, weak filling pulse, hypotension up to the collapse. Hypoxia infarction, acute cardiovascular failure, sudden respiratory depression, can stop the heart.
Coma for alcohol poisoning has some specific features:
1. On examination, attention is drawn to an odor, skin often covered with sticky sweat.
2. Face often hyperemic, although in some cases it is possible pallor and acrocyanosis, hypersalivation noted.
3. Respiratory disorders often are obstructive in nature.
4. More common superficial coma and coma moderate.
5. Be sure there is a high level of ethanol in the blood.
Complications of alcoholic coma: acute respiratory failure by aspiration-obstructive type apnea of central origin, a long position compression syndrome, acute cardiovascular failure, etc.
Frequent intoxication syndrome complicating chronic alcoholism, is an acute encephalopathy Gaye-Wernicke-Korsakoff. Characteristic rapid onset: psychomotor agitation, impaired consciousness, tachypnea, nystagmus, ptosis of eyelids, inhibition of the reaction of pupils to light, decerebrate rigidity, a symptom of forced grasping, bilateral pathological reflexes Babinski.
First aid and treatment of acute alcohol poisoning.
• correction of life-threatening respiratory and circulatory disorders, if necessary treat seizures.
• Gastric lavage through a tube (in a coma - after tracheal intubation) water. Gastric lavage is performed, if after drinking alcohol was not more than 2-3h. Gastroenterosorbtsiya - activated carbon

(sorbent) 0.5-1 g / kg every 8 hours for 1-3 days. Stimulation of intestinal saline laxative, enema.
• intravenous vitamin B1 5% -3-5 ml, then glucose, 60 ml of a 40% solution, in order to avoid the development of encephalopathy Gaye-Wernicke-Korsakoff (glucose administration should always precede the administration of vitamin B1) Also: 3-5 ml 5% solution of vitamin B6, 5.3 ml of a 1% solution of nicotinic acid, 5-10ml of 5% solution of ascorbic acid, 1% -200 ml glutamic acid intravenously for normalization of metabolic processes in tissues.
• Speed up the oxidation of alcohol injected 500 ml of 20% glucose solution with insulin. Relief of metabolic acidosis - alkalization of blood 4% sodium bicarbonate solution (100-200 ml);
• alkylation of plasma and urine;
• forced diuresis. Due to the fact that ethanol inhibits the production of antidiuretic hormone, the need for diuretics is rare. At extremely high levels of ethanol in the blood (> 7r / L) is an urgent need to perform hemodialysis (HD).
• prevention and treatment of cerebral edema, toxic encephalopathy, delirium tremens, aspiration pneumonia, hypothermia, positional compression syndrome, acute renal failure (ARF).

POISONING alcohol substitutes

All alcohol substitutes can be divided into 2 groups: containing ethyl alcohol and do not contain it.
The first group (the true surrogates) include meth, hydrolysis and sulfite alcohol, glue BF, lotions, colognes and so on the basis of these substances is industrial alcohol, with a high content of fusel oils and the addition of a large number of specific components (essential oils, acetone, etc.). True surrogate alcohol poisoning are characterized by a deep and prolonged coma with respiratory and hemodynamic parameters. Emergency care is similar to treatment of alcohol poisoning.
The second group includes - methanol and ethylene glycol. Poisoning by these substances are more dangerous to the life of the victim.

Methyl Alcohol Poisoning

Penetrating into the body through the digestive tract, methanol is oxidized to form highly toxic products - formic acid and formaldehyde, which cause delayed very severe consequences, leading to a marked metabolic acidosis. Sooner develops deep coma. Derivatives of methanol selective effect on the retina, causing vision loss. Minimal lethal dose of methanol - 100 ml.
Clinic. Methyl alcohol poisoning can be mild, moderate and severe.
Mild form is accompanied by headaches, nausea, persistent vomiting, pains in the stomach, dizziness and visual disturbances (haze, blackout) and lasts from 2 to 7 days.
With the average shape - eye - the same phenomenon more pronounced and after 1-2 days of the first symptoms of poisoning occur blindness. The defeat of view, usually asymmetrical, recovery of vision after treatment is possible only in 10-12% of cases. Bad prognostic sign for recovery of a persistent pupillary dilation.
Severe form of poisoning characterized by bright symptoms, joining seizures (generalized or lockjaw chewing muscles and neck muscles). Skin "marble" cold breath atsidoznoe (like Kussmaul), hypotension, tonic seizures, and neck stiffness against psychomotor agitation or coma. On the third day developed acute renal failure. Death occurs as a result of central respiratory paralysis, brain edema and pulmonary vascular collapse or acute renal failure.
Methanol poisoning should be differentiated from a traumatic brain injury, subarachnoid hemorrhage,

stroke, diabetic ketoacidosis, uremic coma, poisoning by salicylates.
Emergency treatment of acute methanol poisoning - patients to be hospitalized in intensive care units or toxicological equipped with the artificial kidney. You want to explore and hospitalize all who drank with the victim.
• correction of life-threatening respiratory and circulatory disorders, treat seizures and excitement: Intravenous seduksen 0.5% - 2-4ml (10-20 mg) or other benzodiazepines, and 10 ml of 25% solution of magnesium sulfate, sodium thiopental to effect
• urgent application of different methods of detoxification - cleansing the gastrointestinal tract, forced diuresis with alkalinization of plasma
• Early hemodialysis, plasmapheresis, peritoneal dialysis. Hemodialysis (HD) hold for 6-7h 2-3 times a day throughout toxicogenic stage. On stage somatogenic DG is used in combination with hemosorption by indications in connection with acute renal failure.
• specific antidote therapy - 30% solution of ethyl alcohol into 50 ml after 3h or 5% solution intravenously (dose of pure alcohol 1.2 g / kg / day). Ethanol prevents oxidation of methanol to formic acid and formaldehyde, accelerates the removal from the body;
• If the view - supraorbital atropine, hydrocortisone
• correction of metabolic acidosis
• Early treatment of protectors hypoxia (Cerebrolysin, stuterona, Cavinton, Actovegin, ynstenona, nootropics, etc.
• B vitamins, to speed up the metabolism of formic acid, folic acid is introduced to 50-100mg 4-6 times a day.
• treatment of cerebral edema, toxic encephalopathy: brain hypothermia, glucocorticoids (up to 300 mg methylprednisolone) osmodiuretics, symptomatic therapy.

Ethylene Glycol Poisoning

Ethylene glycol is a part of anti-freeze and brake fluid. The minimum lethal dose - 100 ml. The poison is rapidly absorbed from the gastrointestinal tract is subject to oxidation to oxalic acid, glycol aldehyde, glyoxal, etc. Oxalic acid combines with calcium ion to form insoluble calcium oxalate (calcium oxalate), the crystals which fall in the tissues of various organs. Is kidney and liver, with the development of ballooning degeneration of the epithelium of renal tubules and hepatocytes CNS damage cell structures and the subsequent development of brain edema, heart disease, blood vessels and lungs. In addition, developing hypocalcemia, can lead to tetany, seizures.
The clinical picture. When ethylene glycol poisoning characterized by a certain periodicity in the development of symptoms, and the development of certain symptoms in each period.
In the first period, which develops after ingestion of ethylene glycol and extends to 12-14 hours, develop a condition similar to alcohol intoxication - nausea, vomiting, abdominal pain, agitation, unsteadiness of gait, characteristic dysarthria, diplopia, mydriasis, impaired memory. progressive disorder of consciousness, even coma, seizures, brain edema and the manifestation of hypocalcemia developing metabolic acidosis, possibly fatal.
In the second period, which begins in about 12-14 hours and lasts 1-3 days, joining phenomena arising in lesions of the cardiovascular and respiratory systems, and compounded the growing metabolic acidosis: tachycardia, moderate hypertension, dyspnea, cyanosis of the total development, in severe cases, the development of acute cardiovascular failure and pulmonary edema. In the third period, which begins

after a day or more, to the fore the phenomenon of acute renal, hepatic or renal insufficiency: oligo or anuria with increasing azotemia, hepatic parameters.
Emergency care and treatment of ethylene glycol poisoning are similar to the treatment of poisoning with methyl alcohol. Add to treatment: gastric lavage with 1% solution of calcium chloride or gluconate and intravenous 10-20 ml of 10% solution of calcium gluconate.

Poisoning of psychotropic drugs

Barbiturate poisoning

Long-acting barbiturates (8-12) - phenobarbital (Luminal), medium-acting (6-8 hours) - barbital (veronal), barbital sodium (Medinai), sodium amytal (Barbamyl), short-acting (4-6 h) - etaminal-sodium (Nembutal). Drugs containing barbiturates: Tardy, Bellaspon, powders Sereyskogo, verodon, bromital, Andipal, dipasalin, kamfotal, tepafilin etc. Psychotropic (narcotic, sedative) effect. Lethal dose - about 10 therapeutic dose (concentration in the blood - 0.1 g / L) with large individual differences.
Absorption in the stomach and small intestine, sometimes at patients unconscious drugs are found intact in the stomach for 2-3 days after administration. Short-acting barbiturates almost entirely (90%) is metabolized in the liver, 50-60% bound to proteins. Long-acting barbiturates bind to proteins (8-10%), 90-95% is not metabolized, excreted in the urine.
Clinic. Observed four clinical stages of intoxication.
Stage 1 - sleep: drowsiness, lethargy, possible contact with a patient, mild cramps with live reaction to light, bradycardia in superficial sleep, hypersalivation.
Stage 2 - superficial coma (a - uncomplicated, b - complicated): complete loss of consciousness, the stored response to painful stimuli, weakening pupillary and corneal reflexes. Unstable neurological symptoms: decreased or increased reflexes, hypotonia, or hypertension, abnormal reflexes, Babinski, Rossolimo wearing rolling nature. Impaired breathing due to hypersalivation, the tongue, of aspiration. Marked hemodynamic disorders not.
Stage 3 - deep coma (a - uncomplicated, b - complicated): the absence or drastic reduction of ocular and tendon reflexes, lack of response to painful stimuli. Pupils narrow. Breathing rare, superficial, weak pulse, cyanosis. Urine output is reduced. In the case of prolonged coma (12 hours) may develop pneumonia, collapse, deep bedsores and septic complications. Liver and kidneys.
Stage 4 – long-term effects period: unstable neurological symptoms (ptosis, staggering gait, etc.), emotional lability, depression, subsequently appear complications (pneumonia, tracheobronchitis, mental disorder, thromboembolic complications).
Emergency care and treatment.
• correction of life-threatening respiratory disorders (airway management, oxygen therapy, in severe cases - mechanical ventilation) and circulation
• the introduction of 0.1% atropine solution (1 ml) to reduce salivation
• Gastric lavage (in a deep coma - after intubation) through a tube, followed by a saline laxative and sorbent, repeated washing. When barbiturate poisoning gastric lavage is performed in the first 2-3 hours, then the tone of smooth muscles is reduced, possibly opening the cardiac sphincter and regurgitation, so in the future it is better to produce only aspiration of gastric contents
• intravenous solution of 5% sodium bicarbonate alkalinity to the blood. Forced diuresis, enterosorbtion.
• hemodialysis, hemodiafiltration, plasmapheresis - in cases of poisoning with phenobarbital, because it binds to plasma proteins is only 8-10%. Hemosorbtion, peritoneal dialysis - barbiturate poisoning, and

the average duration of a short-acting

• Vitamins, liver protecting, antibiotics and other symptomatic agents.

POISONING tranquilizers

Benzodiazepines: clonazepam, diazepam (sibazon, Seduxen, Relanium, Rohypnol, apaurin) Medazepam (rudotel), temazepam (signopam, prazepam) nozepam (tazepam, oxazepam), Elenium (napoton, hloriazepoksid) and others, as well as trioxazine , tofizopan, skutamil. As a result of the antagonistic interaction with benzodiazepine GABA receptors blocked its inhibitory effect on different transmitter systems (serotonin, adrenergic and cholinergic).

Rapidly absorbed from the gastrointestinal tract, 80% bound to belkamisyvorotki blood biotransformed in the liver and the kidneys slowly. The half-life in the case of high-dose extended to 3-4 days.

Tranquilizers are less toxic than barbiturates. Dose resulting in death after oral poisoning, individual. The threat of life in monootravleniyah occurs only under the influence of dose, 100 times the therapeutic (1-2d). However, the combined toxicity is more severe.

The clinical picture is similar to barbiturate poisoning. In mild cases of poisoning appear muscle weakness, lethargy, drowsiness, ataxia, and in more severe - stunning even coma, hypotonia, tremor of the limbs and sometimes the head, tonic-clonic seizures, tachycardia, arrhythmia, lower blood pressure up to the collapse. Unlike poisoning barbituric hypnotics, tranquilizers poisoning respiratory disorder preceded the collapse. Following the collapse appear cyanosis, metabolic acidosis. Often detected lung injury: edema, inflammation, atelectasis.

Emergency care and treatment are the same as for barbiturate poisoning, except for a small nuance: forced diuresis held without alkalinization of urine.

In cases of poisoning by benzodiazepines can be interrupted sedation with intravenous antidote - flumazenil at a dose of 0.2-2 mg, or 0.5 mg / h Its introduction is allowed only on the background of sufficient oxygenation and a good airway. Sometimes a dose of up to 10 mg. Flumazenil should not be prescribed in the case of combined benzodiazepines and lithium poisoning, cocaine, methylxanthines, isoniazid, cyclosporine, MAO inhibitors, tricyclic antidepressants, or if the patient has an arrhythmia, hypotension, seizures or miofibrillyatsiya. Not stopped them sedation induced with midazolam.

Opioids POISONING

Opioids (morphine, opium, heroin, methadone, codeine, etc.) are rapidly absorbed from the gastrointestinal tract as well as the possibility of poisoning when administered parenterally. Detoxification in the liver by conjugation with glucuronic acid (90%), 75% is excreted in the urine within the first day in the form of conjugates.

Toxic dose of morphine when administered 60 mg, lethal - from 100 to 1000 mg. Lethal blood concentration 0.1-4 mg / l. . Lethal dose of opium - 2-3g. When parenteral administration of morphine is more toxic. Duration toxicogenic stage - up to 2 days. All preparations are especially toxic to young children.

Selective toxic effects: psychotropic, neurotoxic (inhibition of thalamic brain regions), decreased excitability of respiratory and cough center. Surrogate narcotic drugs, home-made poppy often have hepato-and nephrotoxicity.

Clinic. Relatively quickly after the intake of these substances are observed flushing, dizziness, nausea, possible daydreaming. Then develop a stunning, pale skin, cramps with weakening photoreaction pupils, bradycardia, muscle hypertonicity, sometimes clonic-tonic convulsions. In severe poisoning - acute

respiratory failure (ARF) (bradypnea, irregular, such as Cheyne-Stokes respiration), cyanosis of the skin and mucous membranes, mydriasis, collapse, bradycardia, hypothermia. Atony of the stomach, intestines, bladder, respiratory depression up to the stop. The degree of respiratory depression prevails over oppression consciousness. Death usually occurs from respiratory or central type, or from a massive aspiration of gastric contents.

If poisoning heroin sometimes observed transient deafness. At a time like heroin poisoning have a duration of about 3-5 hours, in case of poisoning with methadone clinic can occur up to 72 hours or more. If poisoning codeine possible respiratory depression, convulsions when saving consciousness, hypotonia.

After helping opioid poisoning may experience repeated waves deteriorating state of health due to enterohepatic circulation.

Complications including cerebral edema, pulmonary edema, pneumonia.

Emergency care and treatment.

• normalization of breathing (possibly breathing a mixture of oxygen and carbon dioxide), if necessary, correction of hemodynamic disorders

• cleansing the gastrointestinal tract repeated gastric lavage through a tube and aspiration of its contents (even with a parenteral opiates), enterosorption

• specific antidote therapy - naloxone (Narcan) intravenously at a dose of 0.01 mg / kg, repeated every 2-3 minutes until the respiratory depression and recovery of reflexes. Contraindicated naloxone to eliminate ODN (intubation, oxygen therapy, respiratory support).

If, after the 10-15 mg naloxone improvements were observed, the diagnosis of opioid poisoning doubtful.

• atropine 0.5-1 ml of 0.1% solution intravenously or intramuscularly because of bradycardia.

• warm body

• forced diuresis with alkalinization of urine (4% solution of sodium bicarbonate at a dose of 3-4 ml / kg).

• hemosorbtion, peritoneal dialysis

• Fluid therapy crystalloid solutions and plasma substitutes, glucose 40% - 40ml intravenous

• Vitamins B1, B6, B12, C, E, etc., prevention and therapy of complications generally accepted principles, symptomatic therapy.

Impractical introduction of barbiturates, Bemegride, magnesium sulfate, and alcohol.

Organophosphate poisoning

Organophosphorus (OP) are widely used in agriculture and households. The penetration of the poison is through the skin, respiratory tract, gastrointestinal tract. The toxic effect of FOS is associated with exposure to the poison of cholinesterase, which destroys the neurotransmitter acetylcholine (a transmitter of motor impulses from the nerve to the muscle). As this would upset the exchange of acetylcholine. Its excess leads to the first excitation of the nervous system, muscle spasms of the eye, increased secretion glands, seizures. There overexcitation yards holinoreaktivnyh systems (pupillary constriction, bronchoconstriction, bronchial hyperactivity, salivary, sweat glands, cramps, nausea, vomiting, bradycardia). FOS also have a direct effect on the central nervous system cells, inactivate many enzyme systems of the body. Against the background of the WCF and the accumulation of acetylcholine, the tone of the parasympathetic nervous system.

The clinical picture of poisoning are the following steps:

Stage I - excitement. Characteristically patient anxiety, a feeling of fear, aggressiveness manifested. Patients complain of dizziness, headache, blurred vision, nausea, indicated moderate cramps, sweating, salivation. Sometimes joins bronhoreya. Appear vomiting and abdominal cramps. Defined moderate tachycardia and increased blood pressure.
Stage II - the emergence of hyperkinesis and spasms with a fully developed picture of poisoning. Gradually there lethargy soporous state, sometimes coma. Severe cramps, reaction of pupils to light is absent. Pronounced sweating, salivation and bronhoreya. Develop hyperkinesis - almost all the muscles of the body (miofibrillyatsii), periodically - general muscle hypertonicity, tonic convulsions. Appears bradycardia or tachycardia increases. Blood pressure rises to 250/160 mm Hg. Art., then comes the fall of cardiovascular activity. There have been painful tenesmus, involuntary diarrhea, frequent urination.
Stage III - the development of paralysis. Deep coma with a sharp weakening of reflexes or complete areflexia. Pronounced miosis, sweating. All muscles - in paralysis. Predominant form of central respiratory failure. A marked bradycardia (heart rate - up to 20 per minute) or tachycardia (heart rate over 120 per minute). Profuse salivation, bronhoreya, pulmonary edema, and profuse sweating. Growing phenomenon of collapse.

First aid and treatment for poisoning of the WCF:

• correction of life-threatening respiratory disorders (airway management, oxygen therapy, in severe cases - mechanical ventilation, prolonged respiratory support (in the 2-3rd stage)) and blood circulation
• gastric lavage, followed by a saline laxative, cleansing enemas, gastroenterosorbtsiya
• Early hemodialysis hemosorbtion
• antidote therapy (anticholinergics - atropine in all patients during the first hour of treatment until the appearance of the characteristic signs of atropine patients (dryness of the skin and mucous membranes, mild tachycardia, mydriasis).

Introduction of 0.1% solution of atropine

Stage	in the 1st hour mg	per day, mg
I	2-3	4-6 Maximum
II	20-25	to 30-60
III	30-50	to 100

Atropine administered drip. In the 1-hour of high doses must be entered, then support - to create a counter-blockade M holinoreceptor body systems against the action of acetylcholine on the period required for the removal or destruction of the poison (2 to 5 days).
Reactivator of cholinesterase - 15% solution dipiroksima - administered intravenously in 1 ml 3-4 times in the first day, no more than 1.5 g / day. Izonitrozin 40% - 2 ml (no more than 3-4 g), intramuscularly. Again (only the first day)
• forced diuresis, infusion therapy
• symptomatic therapy: relief of pulmonary edema, cerebral edema, prevention of toxic hepatopathy, nephropathy, myocardiodystrophy.

POISONING organochlorine compounds

This group of compounds has its prototype is widely known substance - DDT (dichlorodiphenyl-trichloroethane). This includes hexachloran, dichloroethane, carbon tetrachloride, trichlorethylene, chloroform, etc. They feature - accumulation (accumulation) of toxic substances in the body. It is necessary to note the exceptional durability of these substances in the environment, with the result that

they are distributed to the air and water over long distances and remain in the soil for a long time, from moving into the plant and animal organisms. Found that DDT is stored in the soil 8-10 years. That is why the use of DDT for agricultural processing plants have long been prohibited by law.
Currently, a huge amount of synthesized compounds containing chlorine, which are mainly due to their activity is that element. Used as fumigants and insecticides. Of fumigants (dichloroethane, chloropicrin and paradihlorbenzol) are especially toxic different chloropicrin, during the First World War was the representative of the CWA and suffocating lachrymatory. Dichloroethane is widely used in the home as a means for bonding plastics, the universal solvent, is part of the glue. Lethal dose - more than 10-15 ml. Hospital mortality for poisoning these substances - 30-80%%, with the development exotoxic shock - up to 98%. Carbon tetrachloride is widely used in the home as a solvent stain remover.
Toxicity of organochlorine compounds from the group of fumigants and insecticides quite different. Of the physical properties especially relevant volatility substances and their solubility. Volatile substances used as fumigants are hazardous if inhaled air containing impurity dichloroethane, chlorobenzene and chloropicrin. Solubility in fats and oils at resorption through the digestive tract causes lipoidotropnoe effect in the body, reflected, above all, damage to the nervous system. Chemical properties of the group are determined by the presence and amount of chlorine in a given compound.
The toxicity of ethylene dichloride determined by processes "lethal synthesis", ie products of its biotransformation in the body (monochloroacetic acid, chloroethanol) is much more toxic than most dichloroethane.
Significant place in the mechanism of toxicity as dichloroethane, carbon tetrachloride, and other chlorinated hydrocarbons, played by free radicals, and the processes of free-radical oxidation of the transition in lipid peroxidation, and these processes are irreversible damage to hepatocytes grow as a "snowball" and did not depend the presence of fluorine in the body.
Chlorinated sensitize the myocardium to endogenous and exogenous catecholamines, resulting in can develop ventricular arrhythmias, up to ventricular fibrillation.
Biotransformation products dichloroethane have preferential toxicity to the cell membrane and intracellular structures, causing cytolysis of cells. In the most affected vascular endothelium and hepatocytes. Increased vascular permeability, lesions of the gastrointestinal tract with symptoms of gastroenteritis cause expressed absolute hypovolemia, is the main pathogenic factor in the development exotoxic shock.
The natural way to detoxify the body of dichloroethane is to combine it with the reduced glutathione liver with low toxic mercapturic acids. With a decrease in endogenous glutathione reserves of up to 20% and less developed necrosis of the liver.
Clinic. Acute poisoning with organochlorine drugs are accompanied by headache, nausea, salivation, vomiting, diarrhea. Characterized by retrosternal pressing pain, pain in the epigastric region and in the right upper quadrant. In severe cases, these symptoms joins trembling fingers, limb spasms, sometimes there is jaundice.
The clinical picture of acute poisoning with dichloroethane depends on the dose. At a dose of poison to 50 ml for 30-60 minutes after taking the patient experiences symptoms of central nervous system (euphoria, headaches, dizziness), gastrointestinal (nausea, vomiting, loose stools), the phenomenon exotoxic shock (tachycardia, reduced arterial and central venous pressure). When taking doses greater than 50-100 ml is rapidly developing depression of consciousness up to coma and aggressive type

exotoxic shock. The event of failure or inadequate medical care exotoxic shock progresses rapidly, becomes decompensated character after 12-24 hours ends in death. If you are able to compensate for a shock, for 2-3 days develop signs of toxic hepatopathy, which later can cause death.
If poisoning by carbon tetrachloride is much less pronounced compared with poisoning dichloroethane exotoxic shock, but difficult the liver and kidneys.

Treatment:

• correction of life-threatening respiratory and circulatory disorders

• clarification of the gastrointestinal tract - ingestion: gastric lavage through a tube, enterosorbtion
- With the skin are removed first drop dry swab, and then cool off by 5% solution of sodium bicarbonate rum or 2% p-rum potassium permanganate.
- The eyes are washed with water and 2% p-rum sodium bicarbonate, followed by instillation of 2% solution of novocaine or other local anesthetics, similarly treated nasal mucosa.
- By inhalation - inhalation of aerosols: sodium bicarbonate, antibiotics, novocaine.
• artificial detoxification: hemosorbtion (maximum effect - up to 4-6 hours after taking the poison.) Dichloroethane at doses received more than 1 mL / kg of body weight - hemosorbtion combination with hemodialysis.
• forced diuresis, alkalinization with plasma (with acidosis)
• massive infusion therapy of crystalloids isotonic solutions, alkalizing therapy (sodium hydrogen trisamin, laktosol, etc.), an infusion of plasma and plasma substitutes, 5-20% p-ditch glucose
• drug therapy:
- Corticosteroids (10 mg / kg prednisone per day w / w);
- Antioxidants - vitamin. E up to 50 mg / kg body weight per day / m, vit. C, B1, B12, vitamin PP (nicotinic-ta) 1% - 3 ml / m
- Protect of liver (geptral, silibinin, methionine, lipoic acid, esentsiale)
- In patients with severe anxiety - benzodiazepines (no drug effect). If symptoms of parasympathetic arousal - atropine, of pain – sol. analgini or other non-opioid analgesics.
• Primary prevention of cardiac toxicity (treatment arrhythmias, conduction), brain edema, toxic encephalopathy. for suspected pneumonia may prescribe antibiotics before. Aspiration - Treatment aspiratsinnoy pneumonia. Treatment of toxic pulmonary edema, toxic hepatitis, toxic nephropathy, acute renal failure and other symptomatic therapy
Contraindicated. Milk, castor oil. Do not inject epinephrine, norepinephrine! Mezaton administered with extreme caution!

POISONING cauterizing liquids.

Acid poisoning.

Widespread domestic concentrated acetic acid led to the fact that it for many years, remains the leading cause of serious injury, accompanied by high mortality and persistent disability affected. Acetic acid (ethanoic acid, CH 3 COOH) - monobasic organic acid. Applies to weakly dissociating acids. The main toxicological acetic acid - pronounced resorbtive effect. Easily penetrate through lipid membranes and tissue barriers, rapidly absorbed and broken down, engaging in natural metabolic pathways. At home acetic acid prevalent in the form of glacial acetic acid (96-98% solution), vinegar (40-80%), dilute acetic acid (30%), dining room (3-9%) and wine (3-4%) vinegar. The main route of acetic acid in the body

orally, but there is a possibility of getting through the respiratory tract, skin when applied bandages moistened with concentrated solutions. Toxicity depends on the concentration. The lethal dose of vinegar and about 50 ml.

The mineral acids are most common poisoning hydrochloric (HCl) and sulfuric acids. These acids are used in many industries. Inorganic acid poisoning accounts for about 7% of all poisonings cauterizing liquids. Mortality in this disease is as high as 30-40%. The lethal dose is 40 - 50 ml.

Pathogenesis of poisoning. The most common cause of poisoning of acetic acid is a suicide attempt or accidental admission to alcohol intoxication. Its severity depends on many factors: dose, concentration, adopted the substance, the age of the victim, filling the stomach motility, vomiting, timeliness of care, comorbidity. The pathogenesis of acetic acid poisoning is a combination of local and caustic general effects.

Locally damaging effect of acid on the route - the mucous membranes of the mouth, pharynx, esophagus, stomach, colon, larynx, trachea, bronchi, and lower respiratory tract up to the alveoli. After skin contact, contact with acid damage to the site. It develops chemical burn tissue with the formation of coagulation necrosis of varying length and depth, which results in large fluid losses, and often from damaged blood vessels, and the development of absolute hypovolemic or hemorrhagic shock.

Resorptive effect is due to: absorption of acetic acid and the development of metabolic acidosis, damaged red blood cells and their subsequent development of acute hemolysis and kidney's damage. In connection with development disorders microcirculation caused shock and hemolysis, there liver, toxic coagulopathy, renal damage is compounded.

Hypovolemia, acidosis, hypoventilation, the toxic effect of the products of tissue decay, the centralization of circulation, tissue hypoxia and impaired microcirculation vicious circle, leading to the development exotoxic shock - a condition which, without the timely assistance becomes irreversible and causes the death of more than 70% of victims. Decrease in the production of urine due to dehydration, hypovolemia, hypoxia, blood circulation due to the centralization of pain, acidosis and aciduria, blocked tubules of the nephron hemoglobin and its products of dehydration are the leading causes of acute renal failure in cases of poisoning with acetic acid.

A large area of surface area, the presence of necrotic masses and insufficient blood supply, together with the depletion of the immune system creates an ideal environment for the growth of microflora, which largely determines the flow rate of wound healing and healing of burns.

Burn disease mineral acid poisoning is caused mainly by a direct destructive action of these substances. Resorbtive effect of acids, its duration and intensity depending on the concentration of acid. Poisoning mineral acids cause more pronounced changes CBS blood poisoning than acetic essence is determined by a deep tissue destructive changes. Hemolysis can be observed only by the action of acids unconcentrated, but in intensity it never reaches the level that observed in the employment of vinegar. Liver and kidney damage in this condition due to the development exotoxic shock and acidosis.

The clinical picture.

If poisoning mild superficial burns occur oral mucosa, pharynx, esophagus, stomach, intravascular hemolysis mild or absent, may be subclinical nephropathy with transient proteinuria without affecting renal excretory function.

With an average of severity burns extend to the oral mucosa, pharynx, esophagus, stomach, with damage more often in areas of physiological restrictions in the middle and lower third of the esophagus,

gastric cardia, the pyloric part of the stomach, intravascular hemolysis is more pronounced. Nephropathy accompanied by a violation of renal excretory function symptomatic, but not requiring replacement therapy (hemodialysis, peritoneal dialysis).

Severe poisoning with acetic acid followed by deep chemical burns with lesions of the esophagus, stomach, intestines, severe hemolytic syndrome, severe nephrotoxicity and hepatopathy. In the first, the second day after the poisoning, the most dangerous are hypovolemia, dehydration, violations of ventilation due to exudation and mucosal edema respiratory acidosis, decreased urine output against hemolysis and hemoglobinuria, anticoagulation and associated bleeding. On the second or third day, signs of burn toxemia, infection with hyperthermia, symptoms tracheobronchitis, pneumonia, kidney damage up to complete anuria in severe cases. In the first two weeks, and with deep burns, especially in the face of uremia in acute renal failure, and later during the danger profuse bleeding. At the end of the third week in a fully manifested burn fatigue with weight loss, impaired protein and electrolyte balance, begins the formation of cicatricial stricture, although the first signs of stenosis may occur in a few years.

By the time of acetic acid poisoning complications are divided into early (1-2 days) and late (3-days). The early complications include: mechanical asphyxia, early primary and secondary hemorrhage, acute reactive pancreatitis, primary oliguria or anuria, intoxication delirium. Late complications consider: bleeding, tracheobronchitis and pneumonia, acute renal failure, hepatic, cicatricial narrowing of the esophagus and stomach, malignancy scar deformed walls of the esophagus and stomach.

The clinical picture of poisoning with mineral acids leading syndrome is burn the digestive tract. Corrosive mineral acids is more pronounced than cauterizing effect of vinegar. The vast majority of burn patients revealed moderate (40%) or severe (52%). With deep burns in 1-2% of cases develop severe perforation of the stomach wall and the phenomena of peritonitis. Significantly more than in poisoning acetic essence is expressed decrease pepsin and acid stomach function. More often (in 38% of cases) burns digestive tract completed scar deformation preferentially localized in the antrum of the stomach. Exotoxic shock accompanies common burns digestive tract and has the same features as the poisoning of acetic vinegar. Dates and causes of death in patients with essentially the same as that of acetic essence poisoning. Pathological features include more severe destructive changes of digestive tract wall, no manifestations hemoglobinuria's nephrosis and pigment cirrhosis of the liver.

Treatment of patients with acid poisoning involves a multitude of tasks, among which are:

1. Immediate action:
• Ensure adequate ventilation of the lungs;
• anesthesia;
• removal is not grown deep poison;
• replenishment of circulating blood volume, fluid loss, maintain hemodynamics;
• correction of acidosis;
• conducting forced diuresis with alkalinization of urine in intravascular hemolysis.
2. Prevention of stenosis of the esophagus.
3. Nutrition of patients, providing fluid needs, energy, essential ingredients.
4. Treatment of complications

1. Immediate action must begin at the pre-hospital and carried out by specialized emergency teams (resuscitation, toxicology team), having everything you need for endotracheal intubation, mechanical ventilation. Their tasks include - pain relief, gastric lavage and the beginning of infusion therapy.

Hospitalization is in hospital having intensive care, surgery and toxicology department.
To ensure adequate ventilation involves removing the contents of the oral cavity, trachea, postural drainage. Burns to the respiratory tract in violation of ventilation - an absolute indication for tracheostomy. As an emergency measure, a possible attempt intubation - preferably under local anesthesia. Long standing endotracheal tube against the swelling of the surrounding tissues can lead to necrosis, and the small diameter of its toilet difficult airway.
Anesthesia. In marked pain before gastric lavage, diagnostic procedures used narcotic analgesics. To achieve a rapid onset of action are administered intravenously (morphine 5-10mg, 5-20mg promedol 5-10 ml saline). With a lower volume of distribution against hypovolemia and hypoproteinemia in severe poisoning, the introduction should be done with caution, in divided doses, because of the risk of respiratory depression. Can serve as an alternative to synthetic drugs with selective action on opioid receptors (butorphanol, tramadol).
Removing free poison. Timely removal is not grown deep venom is a prerequisite for success of treatment. Crucial time factor. The rate of absorption of acetic acid is greater, the higher the concentration. If poisoning concentrated acetic acid bulk adopted poison is absorbed in the first 30 minutes after administration. Loss of 15-20 minutes for transportation can be crucial to the outcome of treatment. The first measure immediately after taking caustic substance - as soon as possible to dilute it by drinking 300-400 ml of water. Do not induce vomiting to avoid exposing the esophageal mucosa re-exposure. The method of choice is the tube gastric lavage. Danger resorbtive action is many times the risk of the probe. However, in cases of poisoning cauterizing poisons wash only possible for the first time since in the future, this procedure can lead to perforation of the gastrointestinal tract. Before washing the patient must be anesthetized to reduce the pathological reactions of the vagus nerve is introduced atropine. Locally: gargling, inside 0.5-2% solution of novocaine or lidocaine.
Restores blood volume, fluid loss, maintain hemodynamics. As a starting solution used crystalloids. Hypotension, oligo-anuria, intravascular hemolysis are indications for CVC insertion and monitoring of central venous pressure (CVP). In addition, the indication for CVC is the need prolonged, total parenteral nutrition with deep burns of the digestive tract. With hypotension or low CVP held infusion therapy with hemodynamically active rate (0.5-1 mL / kg per minute). When failure of these interventions were cardiac inotropic support means. An important measure of prevention of pulmonary edema is to maintain the colloid osmotic pressure of blood plasma. It is mainly determined by the concentration of albumin. Its reduction to 30 g / L is an indication for transfusion of protein drugs (albumin).
Correction of acidosis. Acid-base status (CBS) patients with acute poisoning with acetic acid at a given time is determined by a number of competing processes. This - Receipt of acetic acid from the gastrointestinal tract, its metabolism to carbon dioxide and water, the state of blood circulation, the presence of hypoxia, shock, the ventilation of the lungs. Therefore used alkalizing therapy. The method of choice - the introduction of sodium bicarbonate ($NaHCO_3$). Indispensable condition - adequate ventilation. Due to a number of side effects, the use of bicarbonate should be done with caution. The use of standard solutions are not without risk and hyperosmolarity hypersodiumemii. In addition, the rapid injection of concentrated solutions is accompanied by the release of large amounts of carbon dioxide. Against the background of hypovolemia, and respiratory disorders may result in a delay of carbon dioxide inside the cells and exacerbate tissue acidosis. Therefore it is better to use isotonic (1.26%), sodium bicarbonate, or spend the slow introduction of 4% solution through a separate dosing

system in parallel with the primary infusion, an evaluation of the clinical effect, conduct monitoring of indicators CBS reaction urine.

Conducting forced diuresis with alkalinization of urine. The main measure to prevent renal failure in intravascular hemolysis - conducting forced diuresis with alkalinization of urine. The maximum speed of diuresis (about 10 ml / kg per hour) depending on the state of blood circulation, the degree of hemolysis, time after poisoning. The duration of stimulation of diuresis - until no hemoglobin in the urine.

2. Prevention of stenosis of the esophagus. Timely conduct a set of preventive measures to prevent the development of cicatricial narrowing of the esophagus and stomach, require complex surgical treatment in most cases of acute poisoning with acetic acid. It includes:

• glucocorticoids: from the first day of use of methylprednisolone
• antispasmodics: papaverine, no-spa - to reduce the spasm of smooth muscle,
• adequate analgesia
• Local effect: used coating means Almagel
• special diet with total exclusion of hot, hard, mechanically unprocessed food
• antibiotic therapy

Burns to the esophagus and stomach of the first degree is practically no risk of complications and require no specific therapy.

3. Nutrition patients. The defeat of the digestive tract in chemical burns for a long time limit natural food intake, making it necessary to use auxiliary or total parenteral nutrition. Meeting the needs of the body in the water, energy and essential nutritional ingredients - an indispensable condition for the success of treatment. Enteral nutrition should begin after the onset of hunger, restore motility capabilities swallow saliva even in small quantities. Feeding should be a fraction and frequent small meals. Start with vegetable oil and liquid write. C 5-7 days added mechanically processed meat products, soups of different cereals with pureed meat, mashed potatoes, oatmeal, etc. Pathogenetically justified use in the acute stage of the complex of vitamins (A, E, C) at therapeutic doses. Ascorbic acid is involved in the synthesis of collagen connective tissue. With vitamin E, they act as antioxidants.

4. Management of complications, such as bleeding, acute renal failure, infectious complications - held on the same principles.

POISONING alkalis

The most common technique of poisoning caused by ammonia (NH_4OH - 10% aqueous ammonia NH_2; technical ammonia solution contains 28-29% NH_3), in rare cases - caustic soda (sodium hydroxide, NaOH).

The main route of alkalis in the body - oral. In accidents in equipment, piping possible inhalation of ammonia. Alkalis readily dissociate to form hydroxide ions.

Ammonia poisoning account for about 15-20% of all poisonings cauterizing liquids. Common cause of this medication is the use for the purpose of sobering up in alcoholic intoxication. Mortality in this condition - about 5%, 10% lethal dose of ammonia - 50-100 ml.

Pathogenesis. The mechanism of toxic action of alkalis on living tissues other than the acids. Alkalis dissolve mucus and protein substance cells, saponified fats, forming alkaline albuminates hoe, and crushed tissue, making them more accessible to the further penetration of the poison in the deep layers. The destructive effect of alkali on proteins is due to the formation of hydroxide ions entering into

chemical combination with the elements of living tissue.
Alkali leave a deep burns in the esophagus, while the stomach is suffering less than acid poisoning due to neutralizing the action of gastric juice. Resorbtive phase alkali poisoning lasts from 30 minutes to 2 hours, a period of intense resorption is 15 min.
Clinic. The clinical picture of poisoning syndrome is the leading burn the digestive tract. On examination, marked swelling of the mucous and submucous layers site desquamation of the mucous membrane, the absence of a sharp boundary with intact tissues. With deep burns may develop acute esophageal perforation with subsequent development periezofagitis, mediastinitis, pleurisy.
Severe burns of the esophagus usually end of stricture preferentially localized in the thoracic and lower third of the esophagus. Cicatricial obliteration of the esophagus develops within 1 - 2 years after the burn. At post mortem examination distinguishing feature is the nature of burns: The mucosa is swollen, loose, with a glassy touch, easy to tear, no sharp boundary with intact tissues.
Combined treatment is the same as that of a mineral acid poisoning. Correction of metabolic acidosis is much easier - infusion (glucose-novocaine mixture, etc.) in an amount to provide a moderate hemodilution.

POISONING oxidants

These poisonings most often caused by intake of hydrogen peroxide. Hydrogen peroxide, H2O2 - a clear, colorless liquid with no odor or with a faint odor, slightly acid reaction. When interacting with alkalis and organic matter releases oxygen. Available in the form of a concentrated solution of H2O2 (perhydrol) containing 27.5 - 31% H2O2 diluted solution of H2O2 (3% H2O2) gidroperita - tablets containing compound with urea H2O2 (1 tablet corresponds to 15 ml of a 3% solution of H2O2).
The main route of exposure - oral. When in contact with living tissues is decomposed with the liberation of oxygen. Is a strong oxidant. The most toxic properties has perhydrol. Lethal dose perhydrol - 50-100 ml.
Hydrogen peroxide is expressed destructive changes the digestive tract wall, which by nature are close to the action of alkalis. Deep mucosal damage, sometimes submucosal muscle layers in violation of the integrity of the vascular wall create the conditions for the penetration of gaseous oxygen in the bloodstream and the subsequent development of gas embolism of the brain and heart.
If poisoning develops hydrogen peroxide burn disease with characteristic major pathological syndromes. Severe complication of this disease is the gas embolism brain. In patients with marked impairment of consciousness, the appearance of focal neurological symptoms, respiratory failure the central type may create certain diagnostic difficulties.
Treatment of poisoning with hydrogen peroxide has the same features as poisoning by alkalis. With the development of the brain shows a gas embolism hyperbaric oxygenation.
Carbon monoxide poisoning
Carbon monoxide (chemical formula - CO) - a product of incomplete combustion of carbon, is where the carbon-containing substances are burned in low airflow. In its pure form, this gas is colorless and odorless, slightly lighter than air, so in areas primarily accumulates in the upper layers of air near the ceiling. In practice, the CO is found in a mixture with other gases: combustion of substances and atmospheric air.
The process of carbon monoxide poisoning has long been known as burning, it was the name of the domestic gas - carbon dioxide. CO poisoning can occur when you are in homes with furnace heating with

a defective chimney or premature closure of the stove damper, the fires from parties in smoky rooms with no means of defense, when in a poorly ventilated garage with the engine running.
The higher the concentration of CO in the air, the faster the life-threatening concentrations of carboxyhemoglobin in the blood. For example, if the concentration of carbon monoxide in the air is 0.02-0.03%, then 5-6 hours of inhalation of the air will create a carboxyhemoglobin concentration of 25-30%, and if the concentration of CO in the air is 0.3-0.5% , the deadly contents of carboxyhemoglobin level of 65-75% is reached in 20-30 minutes of being human in this environment.
Pathogenesis. Carbon monoxide has a high affinity for hemoglobin. Therefore, getting into the lungs with atmospheric air is then dissolved in the blood plasma into erythrocytes and enters into an irreversible bond with hemoglobin. , The complex - carboxyhemoglobin (SOHb), which can not bind and carry oxygen. If the flow of carbon monoxide in the human body is not stopped, the carboxyhemoglobin is gradually accumulated in an amount which prevents the normal transport of oxygen, which leads to severe hemic hypoxia. The high affinity of carbon monoxide to iron makes its entry into the reaction with tissue respiratory enzymes and impaired oxidation-reduction processes. Developing tissue hypoxia, especially CNS. After binding of carbon monoxide more than half of hemoglobin can be fatal if not treated qualified medical assistance.
Clinic and diagnostics. Depending on the intensity of poisoning are three degrees of poisoning:
• mild - the victim complained of a headache in the temporal and frontal areas, often encircling nature, dizziness, nausea and vomiting often occur. , A slight impairment. Patients complain of a sore throat, shortness of breath, dry cough. Increased blood pressure, pulse rate, there are unpleasant feelings in the heart. Consciousness is not impaired. Carboxyhemoglobin content 15-30%.
• medium - Violations of mental activity manifested excitation or stunning, shows a loss of consciousness (from 1-2 to 20 min.), Convulsions. Appear abnormal reflexes, coma, different pupils, persist tachycardia. Noteworthy carmine-red color of the skin. SONb content from 30% to 40%.
• severe degree - characterized by the development of a coma of varying depth and duration from a few hours to several days or more. Observed seizures, abnormal reflexes, paresis, and paralysis due to ischemic polyneuritis. Skin color changes from red to cyanotic. Due rinolaringitis and tracheobronchitis develops shortness of breath, marked pathological types of breathing to a stop from a paralysis of the respiratory center. From the CCC - left ventricular failure, ECG signs of hypoxia and ischemia. Subsequently, progressive renal failure as a result of severe hypoxia. Necrosis of tissue due to trophic disorders, bullous dermatitis manifests itself in 10-15 hours after ingestion. In the blood - metabolic acidosis. Carboxyhemoglobin content - 50-60%. The heart may be reduced for some time after the cessation of breathing. Cases of death from the effects of poisoning even after 2-3 weeks after the event of poisoning.
Of course, the rate of fatal carbon monoxide poisoning is influenced by many factors that accelerate or slow down the process. Sometimes there is increased resistance to carbon monoxide. For example, women and children under the age of carbon monoxide poisoning suffer more easily than men.
First aid for carbon monoxide poisoning.
• take steps to security: not light the fire, turn off the phone and electric bell (possible sources of explosion);
• Quickly remove victim to fresh air;
• If the ventilator breath hold method "mouth-to-nose", but do it carefully because of the risk of

poisoning;
• implement immediately tracheal intubation and mechanical ventilation with high FiO2 (pure oxygen);
• the victim immediately hospitalized in the intensive care unit soon, preferably in a specialized hospital, being able to conduct hyperbaric oxygenation.
Treatment.
• stabilization of the patient.
• antidote therapy: in cases of poisoning by carbon monoxide or carbon monoxide poisoning antidote is oxygen, which is given by inhalation immediately after the evacuation of the victim from the source. When breathing oxygen SOHb half-life is reduced to 20 minutes. The most effective way to treat oxygen - hyperbaric oxygen (HBO) at a chamber pressure of 2 - 3 am for 50 - 60 min., Which is the method of choice for these poisonings. Expedient patient irradiation quartz lamp as UV rays accelerate the decay of carboxyhemoglobin, recommended the use of carbogen, which is a mixture of 95% oxygen and 5% carbon dioxide, as carbon dioxide displaces the oxyhemoglobin dissociation curve to the right.
• symptomatic treatment - anticonvulsant therapy, infusion therapy crystalloid p-set and colloidal substitutes, vitamin (vit. C, B1, B6, Vit E), hypothermia. When thermal injury of the upper respiratory tract: inhalation of a mixture consisting of 4% solution of sodium bicarbonate - 5 ml hydrocortisone - 125 mg, 5 ml of 3% solution of ephedrine. Correction of respiratory disorders.
In order to prevent complications from the respiratory system prescribed antibiotics.

Mushroom poisoning

Every year in our country registered cases of acute poisoning by poisonous mushrooms, and with the greatest frequency in August - September. On the severity of the clinical course of these poisonings are the leading among all non-bacterial food poisoning.
Mushrooms with hepatotropic ACTION
(Pale toadstool (Amanita phalioides), stinky mushroom (Amanita virosa).
Mushrooms containing hepatotropic poisons (death cup, green mushroom, amanita smelly), in forests, mostly coniferous, from June to October. Hat mushroom initially has a bell, then slightly convex (almost exactly), greenish or olive-green color. Hat death cup has a white or yellowish color. Plate of mushrooms - white. Foot also white, shiny, clavate at the base with a thickening in the upper part of the stem has a characteristic of these fungi fringed ring. Especially dangerous young specimens, which are mistaken for mushrooms or russules.
Toxicity. These mushrooms contain very strong toxins in cluster cyclopeptide: amanitotoxin (amanitin, amanina, amanullin) and fallotoksiny. Amanitotoxin - cyclic oligopeptides. Inhibiting RNA polymerase II, amanitotoxin violate DNA transcription and interrupt protein synthesis, which leads to cell death. The most vulnerable cells with intense protein synthesis - hepatocytes, enterocytes, nefrotsity, lymphocytes. The cells of the intestinal mucosa are the first defeat. Affects all the functions of the liver. Fallotoksiny - cyclic geptopeptidy. The experimental data, fallotoksiny lead to the destruction of the membrane of hepatocytes and inhibition of mitochondrial oxidative phosphorylation. Fallotoksiny largely destroyed by the enzyme systems of the gastrointestinal tract, and because of their low absorbability in the gut virtually no toxic effect. 100 g of fresh mushrooms (5 g dry) contains 10 mg of phalloidin, 13.5 mg amanitin. Severe poisoning develops at the use of mushrooms in 1 g per 1 kg of body weight. The lethal dose of amanitin - 0.1 mg / kg. The toxins are not destroyed by heat treatment and drying, are rapidly

absorbed from the gastrointestinal tract and are deposited in the liver.
Milk of nursing mothers, poisoned by these fungi can be dangerous for the baby.
Dangerous as fried and cooked mushrooms, as these toxins are thermostable. In addition, given the water solubility of toxins, rather the presence of one death cup in a certain amount eatable mushroom poisoning to develop, as water treatment fungi (washing, cooking, etc.) takes place in a bowl.
Clinic. This type of mushroom is especially tricky. While in other mushroom poisoning symptoms of gastrointestinal disorders appear already at 0.5 - 1 hour after eating, for mushrooms hepatotropic action-long latency period - from 6 to 24 hours, sometimes longer, which is characterized minimum objective and subjectively complaints. Most often, the latent period lasts about 12 hours.
In the development of poisoning are three main stages.
The first stage begins with abdominal pain, vomiting and diarrhea, continuing 1-3 days. Mild forms may be limited to this stage. However, tenderness distinctly enlarged liver, as well as positive liver function tests indicate liver damage, indicating that poisoning falloidnymi mushrooms.
In more severe cases developed fulminant toxic gastroeneritis, diarrhea is choleriform character, often mixed with blood. Due to severe fluid and electrolyte loss develops exsicosis with hypovolemia, high values of hematocrit and circular collapse. According to some authors, a longer latent period causes intense absorption of toxins and more severe clinical picture.
The second stage of poisoning death cup - 1-2 day interval imaginary prosperity with less severe gastro-intestinal disorders.
In the third stage, marked enlargement of the liver, with its tenderness to palpation, jaundice parenchymal type with the presence of bile pigments in the urine, very high values of alanine (ALT) and aspartate (AST) transaminases (especially ALT). The level of glucose in the blood increases due to violations glycogen synthesis liver function, there is a decrease prothrombin activity with hemorrhagic manifestations. Often detected renal disease - oliguria, proteinuria, azotemia. In an unfavorable course of the disease patients die in the coming days with symptoms gepatargii.
Treatment.
• Given the fact that patients with hepatotropic poison mushrooms are symptomatic of toxic gastroenteritis (repeated vomiting and diarrhea), the need for gastric lavage not. Gastroenterosorbtsiya - activated charcoal inside.
• Infusion therapy crystalloid isotonic solutions for the normalization of water and electrolyte metabolism.
• Effective (especially early stage - the first 6-12 hours after ingestion) detoxification hemosorbtion and plasmapheresis.
• Specific therapy - silibinin (legalon) - 20 mg / kg / day. When using products containing silymarin (silibor, Kars), remember that 70 mg of silymarin approximate the performance of 30 mg silibinin. Essentiale - 15-20 ml / day, livomin (Liv-52) - 8-12 tablets / day, and prednisone 90-180 mg to 1000 mg / day. Lipoic acid 0.5% - 2 ml to 20-30mg/kg/sut. Use as a specific therapy for lipoic acid is now recognized not effective.
• Liver transplantation (best - at 4 days after poisoning).

Snakebite

In the CIS, there are 58 species of snakes, 10 of which are toxic to humans. According to statistics, most often bitten by people aged 20-50 years, the children in 30% of cases. Deaths from bites cobra and viper

- 8-15% of cases, from the bites of vipers - 2-12% of cases, but 25% of children.
Acute poisoning is caused by the specific action of snake venom - Product snake venom gland. Poisons can be divided into two groups:
• hemotoxic (viper, rattlesnakes);
• neurotoxic (cobra).
Hemocoagulation effect poison of vipers and rattlesnakes leads to the development of thrombo-hemorrhagic or DIC for 1-3 hours after being bitten by a snake. As a result, growing consumption coagulopathy, and the internal blood loss reaches 50% of BCC, which is also the main cause of death. Under the influence of poisons and enzymes lecithinase and phospholipase A developing hemolysis in areas of hemorrhage. Poisons help release histamine, bradykinin, and other biologically active substances in violation hemodynamics.
Features intoxication cobra bites. In venom contains two non-enzymatic polypeptide toxin - a neurotoxin and cardio, as well as enzymes: phospholipase A, hyaluronidase, and cholinesterase.
The main toxic principle is a neurotoxin, has curariform action, inhibits reflex activity, paresis, paralysis. Particularly sensitive bulbar centers and spinal cord.
In contrast to the venom of vipers and cobras venom has anticoagulant activity (phase 1 inhibits clotting) and increases fibrinolytic activity.
Clinic. Bites of vipers - petechial or macular hemorrhage in the bite site, rapidly progressive hemorrhagic edema of the affected limb. In the first 10-15 minutes - the appearance of the first symptoms and further progression of shock (mostly due to hypovolemia). In the future, can join the phenomenon of intravascular hemolysis with the development of disseminated intravascular coagulation, and acute renal failure.
The bite of a cobra - neurotoxicity associated with impaired sensitivity parastezii, ptosis, dysphagia, ascending motor paralysis (curariform effect), disruption of the central nervous system, respiratory and cardiovascular system - impaired consciousness, respiratory depression, cardiac arrhythmias and conduction.
Snake bites may be complicated by sepsis, necrosis of soft tissue with the formation of ulcers, gangrene of the extremities.
Treatment. You cannot do: drink alcohol, scarify to the bite site, enter $KMnO_4$ solutions to the bite, apply a tourniquet.
• Prehospital: immobilization limbs, horizontal position of the patient. Suctioning venom by mouth or by special devices (effective in the first minutes after the bite, after more than 30 minutes - inefficient.) Then disinfect wounds common method applied aseptic bandage.
• the prevention and treatment of shock and gemoglobinuriynogo nephrosis - early onset of a massive infusion therapy with the introduction of crystalloid solution, fresh frozen plasma, alkalization, forced diuresis. In case of impossibility of infusion - excessive drinking.
• Corticosteroids - 2-3 mg / kg prednisone, followed by 1 mg / kg per day.
• Specific therapy is conducted mono-and polyvalent sera - "antigyurza", "antikobra" etc. Monovalent serum preferable because less likely anaphylactic reactions. Bites of vipers and rattlesnake serum application is most effective in the early hours (not a panacea). Cobra bites injected serum in any terms.
• symptomatic treatment
• prevention of tetanus.

Insect bite

The most dangerous in our band for man hymenopteran - bees, wasps, hornets. The components of the venom of insects:

• amines (histamine, dopamine, serotonin, etc.)
• peptides (mellitin, aspen and hornetkinin)
• enzymes (phospholipase A and B, hyaluronidase)

Hyaluronidase promotes the spread of venom from the site of sting. Phospholipase A is similar to snakeskin, an indirect hemolytic factor. Wasp and hornetkinin - similar to the human bradykinin. Mellitin (peptide), phospholipase A, have a direct hemolytic effect and narrowing of blood vessels and expand, reduce skeletal and smooth muscle, the myocardium, increase vascular permeability, disrupt metabolism. Apamin - neurotoxic venom for spinal and bulbar centers.

Despite the differences in the venoms of bees, wasps and hornets, and similar clinical toxicity is dose of poison (number of bites), bites, on individual sensitivity. Local Clinic: burning pain in the site of the bite, pale papule, with a belt of hyperemia and edema of the tissues. Bites to the face, neck lymphadenitis phenomenon, and stings the eyes and mucous membranes: a lot of pain, conjunctivitis, blepharitis, epiphora, muco-purulent discharge, laryngeal edema, asphyxia. Common symptoms: chills, fever, shortness of breath, dizziness, headache, tachycardia, nausea, vomiting, fainting, decreased blood pressure, blockade of autonomic ganglia and neuromuscular synapses.

Mortality:

- If he had been stung by wasps and hornets are rare and occur from heart failure and asphyxia;
- If he had been stung by bees, from paralysis of the respiratory center.

Treatment - symptomatic

• remove the stinger (if any) as soon as possible, wash the wound with alcohol or other antiseptic, apply cold, novocaine blockade bites
• ensuring the airway (endotracheal intubation, tracheotomy);
• infusion therapy. Forced diuresis with the introduction of 4% solution of sodium bicarbonate. V / IV infusion of plasma, albumin and plasma substitutes. Intensive fluid administration continues to stabilize hemodynamic parameters, then move on drip solutions

• Drug therapy (similar to treatment of anaphylactic reactions) - corticosteroids in / in, antihistamines (diphenhydramine, pipolfen).

Bites of spiders and other arthropods. Karakurt - poisonous spider. Length of female - to 2 cm. Body is black, male abdomen with red spots. Animals and man does not attack unless it is disturbed. Female bites can be deadly for animals (camel, horse) and human. Found in the southern regions of Ukraine. Venom of carakurt contains alpha latrotoksin that is extremely durable connect specific receptor proteins synaptic membranes, causing the opening of nonspecific cation channels, leading to increased calcium entry, a massive release of acetylcholine.

Karakurt bites accompanied by significant systemic toxicity response without significant local manifestations. During the first 30-120 minutes after the bite marked effects of ataxia, muscle weakness, pain in the extremities, back, in the anterior abdominal wall. Karakurt bites can simulate hospital myocardial infarction, acute abdomen. Besides the body temperature to 39-400C, chills,

sweating, hypertension occurs. In the most severe poisoning (especially in children) - coma, convulsions, pulmonary edema.

Treatment. Wound treatment, cold on the bite, the prevention of tetanus. Observation for at least 6-8 hours after the bite, with symptoms of respiratory disorders - tracheal intubation, mechanical ventilation.

Specific therapy - calcium supplements (calcium chloride, 10% of 0.1-0.2 ml / kg. Remember that calcium gluconate contains about three times less than milliequivalents of calcium, so the dose should be 3 times higher).

In severe muscle rigidity, muscle relaxants. In severe pain, use of narcotic analgesics. In the most severe cases - specific antikarakurt serum.

Tarantulas. Venom of this spider is a colorless, somewhat turbid and viscous liquid. Per person tarantula venom has little effect and, apparently, only in rare cases can cause fatal poisoning. Coming after the bite wound, yawl produces mainly local action, which rarely align general symptoms as fever, rapid pulse, sweating and collapse. Probably Severe and deaths related to exposure to the wound pathogens that are a spider on the hooks and chelicerae.

Treatment for the bite of this spider is reduced to symptomatic treatment and sterilization of the sting by the same treatment as the infected wound. Special serum no, but you can enter antikarakurt's.

Scorpio, a poisonous spider. Length - 1-20 cm legs are large, armed with claws on the end of the abdomen Hooked poisonous sting. About 750 species, found in tropical and subtropical regions, including Central Asia, Kazakhstan, South Crimea and the Caucasus. Viviparous (young larvae remain in the mother's body). Are nocturnal. Large scorpion venom is dangerous to humans and animals. Scorpions - the oldest terrestrial arthropods.

Scorpion venom contains digestive enzymes (giauloronidazu, phospholipase) and, in addition, strong neurotoxins that cause damage to the sodium channels with the stimulation of neuromuscular synapses and the autonomic nervous system.

Scorpion stings, in most cases, cause severe pain in the site of the bite with irradiation on nerve trunks. The pain is accompanied by hyperemia and edema. When mild local reactions, general intoxication usually more pronounced. Symptoms of intoxication, usually expressed only in children under the age of 10 years. It is noted headache, dizziness, vomiting, shortness of breath, palpitations, agitation with the transition to drowsiness, sweating and thoroughly with increased salivation, bronchospasm, diplopia, nystagmus. Sometimes there is a coagulopathy, pancreatitis, acute renal failure. Symptoms of poisoning are most pronounced in 3-4 hours after the bite, appear no more than 24-36 hours.

Treatment. Cold compress on the bite, analgesics. Prevention of tetanus. Do not use excessive sedation of patients. Specific antitoxic serum needed tropical scorpion stings.

Self-study.

Task 1.

Inspect the patient with poisoning with acetic acid. Ground possible mechanisms for the development of clinical and laboratory syndrome, further examination, intensive care.

Task 2.

Learn the history of the patient with methemoglobin poisoning. Examine the patient's treatment strategy. To do this:

• Find out the data history of the history of the state
• Perform analysis of objective data on the diaries
• Analyze data from laboratory and instrumental methods in dynamics
• The differential diagnosis
• Evaluate treatment strategy worksheets appointments

Clinical problems

Objective number one

Patient K., 40 years old. He was in the office with a diagnosis of acute poisoning: acetic acid poisoning, burns mucous oropharynx and esophagus. On day 3, the patient appeared profuse gastric bleeding, should determine the tactics of therapy.

Objective number two

The injured man 43 years old, oral poisoning in production (seed treatment Dostum). Ingested castor oil, was getting worse abdominal pain, sudden excitement, leg cramps, muscle weakness, and severe hypotension. Diagnosis, tactics? What a mistake was made?

Task number 3

The patient, aged 38, an auto mechanic, was taken to the emergency room in a deep stunning. Contact is not available, the response to painful stimuli is preserved. OBJECTIVE: severe flushing of the skin, eyes narrowed, anisocoria, tendon reflexes are increased. Notes shallow breathing, tachypnea. Heart sounds are muffled, rhythmic, heart rate - 118ud/min. In the study of CBS: pH - 7.33, BE - (-5) mmol / L, SoHb - 36%. What is your diagnosis and treatment policy?

Test control:

1. Treatment of caustic ingestion should be to:
A. vomiting
B. endoscopy in the first 24-48 hours *
C. drinking acidic solution
D. Heavy drinking milk or water for dilution of caustic substances
2. It is a cholinesterase reactivator:
A. atropine
B. lipoic acid
C. Neostigmine free radicals;
D. Physostigmine carboxyhemoglobin
E. dipiroksim *
3. The presence of free hemoglobin in the blood poisoning cauterizing poison causes the development of:
A. respiratory failure;
B. liver failure;
C. renal failure *
D. cardiovascular disease.
4. With carbon monoxide poisoning, severe observed:
A. coma *
B. arterial hypertension
C. myoplegia

D. tonic convulsions *
E. respiratory disorders *
F. arterial hypoxemia *
G. drowsiness
5. The sequence of urgent measures for poisoning acetic essence provides everything except:
A. introduction of analgesics;
B. tube gastric lavage with water;
C. bezzondovoe lavage alkaline solution *
D. forced diuresis;
6. Clinical signs of poisoning with alkali:
A. Hypersalivation. *
B. Loose scabs on the mucosa.
C. The searing pain along the gastrointestinal tract. *
D. Metabolic acidosis. *
7. Choose the selective removal of toxin:
A. sorption *
B. filtration;
C. apheresis;
D. phototherapy.
8. Complications hemosorption all, except:
A. sorbent DIC;
B. trauma and hemolysis of red blood cells;
C. diarrhea *
D. pyrogenic reactions.
9. If lavage stomach activated charcoal used in a dose:
A. 1 mg \ kg
B. 5 mg \ kg
C. 1 g \ kg *
D. 10 g \ kg
E. 0.1 mg \ kg
10. Which event counter with a viper bite:
A. Drink plenty of warm
B. immobilization of the affected limb
C. applying tourniquet above the bite *
D. imposition of aseptic dressings
E. introduction of tetanus toxoid
ANSWERS
Objective number one
Burns to the digestive tract of the second degree deeper damage, usually accompanied by bleeding. To assess the hemostatic disorders need to control the number of platelets, fibrinogen concentration, determination of prothrombin and activated partial thromboplastin time. Introduction to conventional doses of fresh frozen plasma (10 mL / kg) may be enough to eliminate the deficit of fibrinogen and

prothrombin. In addition, the depletion of the bone marrow results in a high risk of thrombocytopenia. Therefore, the number of platelets should be monitored along with red blood. It decreased less than 100,000 - 75000/mm3 - indication for the use of platelets. To eliminate early secondary bleeding from the esophagus and stomach using local hypothermia. For this purpose, use one - or two-channel probe, giving c / o them chilled with ice up to 2-4 ° C water. The water is fed at a rate of 0.5 l / min. for 1-2 hours.

Objective number two.

Organochlorine poisoning. Castor oil is contraindicated.

Task number 3.

CO poisoning, moderate. Oxygen therapy. When the tongue - duct. In severe respiratory failure - mechanical ventilation, sanitation tracheo-bronchial tree. Antidote therapy: giberbaricheskaya oxygenation (HBO). Infusion therapy. Aminophylline 2.4% 10ml. When hypotension - cardiotonic. Treatment of brain edema. Antibiotics. Vitamin therapy