ACUTE AND CHRONIC PESTICIDE POISONING (PHOSPHORORGANIC, CHLORORGANIC AND MERCURORGANIC COMPOUNDS).

ETIOLOGY, PATHOGENY, CLASSIFICATION, POSITIVE AND DIFFERENTIAL DIAGNOSIS, TREATMENT, PROPHYLAXIS

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Definition

Pesticides are a series of chemicals with a particularly high biological action, intended and used in agriculture, forestry in order to prevent the action and / or combat some forms of plant or animal life, which cause direct and indirect damage to crops and animals. The term "pesticide" was taken from the English language, where "pest" means harmful insect, and the term "icide" reffers " destroy, kill."

Pesticides are classified as:

I. Depending on the destination;

II. Depending on the origin;

III. Depending on the chemical structure;

IV. Depending on the level of toxicity.

I. Depending on the destination of the pesticides, they are divided into the following groups:

1. Insecticides (pest control);

2. Zoocides (to control animal pests)

3. Rodenticides / Raticides (rodent control);

4. Molluscocides (mollusc control);

5. Nematocides (fighting nematodes);

6. Larvicides (fighting larvae);

7. Acaricides (pest control);

8. Ovicides (destruction of insect and mite eggs);

9. Algaecides (destruction of algae);

10. Herbicides (destruction of weeds in crops);

11. Fungicides (control of fungi that cause plant diseases);

12. Growth regulators (the processes of inhibition or stimulation of plant growth):

a) defoliation: means of defoliation of plants;

b) desiccant: means of drying the plants before harvesting;

c) deflowering: means of removing the excessive amount of flowers;

d) attractive: means to seduce;

e) repellents: means for rejection.

II. Depending on the origin:

1. Mineral origin: (As salts, Ba, Cu, Hg, Pb) etc.

2. Plant origin: (nicotine, veratrum, strychnine).

3. Synthetic origin: (organophosphorus esters, organohalogenated derivatives, aromatic nitroderivatives, carbamic derivatives, phenolic compounds and organometallic compounds).

III. Depending on the chemical structure:

1.Phosphororganic pesticides

2. Chlororganic pesticides

3. Mercury organic pesticides

4. Carbamic and thiocarbamic pesticides, respectively

5. Nitrophenolic pesticides

6.Sulfur preparations

7. Copper preparations

8. Arsenic preparations

9. Alkaloids, etc.

IV. Depending on the level of toxicity:

1.Group I: extremely toxic substances, induce fatal intoxications (lightning), LD50 <50 mg / kg, being marked with red labels.

2. Group II: substances with a strong toxic effect, LD50: 50-200 mg / kg, being marked with green labels.

3. Group III: substances with medium toxic effect, LD50: 200-1000 mg / kg, being marked with blue labels.

4. Group IV: substances with low toxic effect, LD50> 1000 mg / kg, being marked with black labels.

(LD50 - lethal dose value)

Toxicity can enter the body on different paths:

1. The respiratory tract,

2. The cutaneous route,

3. Digestive tract.

CLASSIFICATION OF PESTICIDE POISONING

According to the amount of toxic compound that enters the body, we can define the following:

• Acute intoxications (mild, moderate, severe form)

• Subacute intoxications.

• Chronic intoxications (stage I, II, III)

1. Acute intoxications.

They occur as a result of the penetration into the body of a large amount of pesticides. Characteristics:

- The early period, which includes the period of time from the penetration of the toxic in the body, until the appearance of the first signs of intoxication.
- The preclinical period, for which non-specific symptoms are characteristic such as: vomiting, nausea, headache, general weakness, which can also be found in other pathologies.
- The period of intoxication itself, has a series of specific clinical signs that appear as a result of the action of the toxic compound on the body.

2. Subacute intoxications

It depends on the amount and toxicity of the toxicant that has entered the human body. It is characterized by an insignificant reaction to the action of the toxicant on the body, compared to the reaction from acute intoxications, the pathological process having a more erased and longer evolution.

3. Chronic intoxications

They occur as a result of the over time sum up of the effects of small and repeated doses of the toxicant on the human body.

POISONING WITH PHOSPHORORGANIC COMPOUNDS

According to the chemical structure, the compounds of this group are ethers of phosphoric, thiophosphoric, dithiophosphoric and phosphonic acids. The representatives of this group are: Carbofos, Fazolon, Fosfamid, Methylnitrofos,

Metafos.

It is used for insecticides or acaricides in agriculture, fruit growing, viticulture and forestry, with intense effects even at low doses.

Pathogenesis

It is known that many Organophosphorus compounds can inhibit both Cholinesterase and other enzymes.

As a result of the inactivation of Acetylcholinesterase, the mediator of the Nervous System (synapse) - acetylcholine, "endogenous acetylcholine intoxication" accumulates, which leads to the disorder of the transmission of the nervous impulse through nerve cells and lymph node synapses.

The basic symptoms of intoxication with phosphoroorganic pesticides are determined by the presence of:

1. The muscarinic effect,

2. The nicotinic effect,

3. The central action of acetylcholine.

Muscarinic effect (parasympathomimetic action):

1. Ocular effects: miosis, decreased visual acuity and accommodation disorders.

2. Cardiovascular effects: short-term decrease in blood pressure, in the heart: an inotropic, chronotropic, tonotropic, dromotropic negative effect prolonging the P-Q interval; stimulates the excitability of the myocardium, especially the atrial one and predisposes to atrial arrhythmias; bradycardia.

3. Effects on smooth muscles, contracts the muscles:

• bronchi;

• gastrointestinal tract, including the intra- and extrahepatic bile ducts;

• ureters, bladder;

• uterus and fallopian tubes.

4. Effects on exocrine secretion, stimulates the secretion (increases the secretion of sweat, tear, salivary, bronchial, gastric, pancreatic and intestinal glands).

The nicotine effect

The disorder of the impulse transmission in the postganglionic fiber, which determines:

1. Hypertension (by generalized vasoconstriction).

2. Tachycardia (by acting on the sympathetic ganglions and releasing catecholamines from the adrenal medulla).

3. Apnea (by reflex mechanism starting from the large pulmonary vessels), followed by polypnea (by excitation of sinocarotid receptors).

4. Hyperglycemia (by releasing catecholamines).

5. Muscle fasciculations (by acting on nicotinic receptors and on the neuromuscular junction): eyelid contractions, tongue contractions, neck contractions.

Central action

It is determined by the toxic action of acetylcholine on the cerebral cortex and spinal bulb, which is manifested by:

- Headache,
- Anxiety,
- Dizziness,
- Insomnia,
- Loss of balance,

- Excitement,
- Psychological disorder,
- Consciousness alteration,
- Convulsions, coma,
- Paralysis of the centers with vital importance in the spinal bulb.

Clinical picture of acute intoxications

1. Light form

It occurs with the following symptoms: headache, dizziness, weakness in the limbs, decreased vision, anxiety, nausea, hypersalivation, abdominal colic, diarrhea.

Sufferers are worried, pupils are narrowed, reacting to low light, the accommodation spasm develops, which leads to decreased visual acuity, affected at dark adaptation and in dimly lit rooms.

Nystagmus, edema of the face, hypertranspiration occur.

Prolonged action leads to difficult breathing (predominantly in the inspiration), chest discomfort accompanied by respiratory failure, cough attacks.

Heavy breathing, dry rales are heard throughout the lung area.

Cardiovascular system: tachycardia, increased BP.

There is a notable decrease in the activity of erythrocyte cholinesterases in the blood serum.

2.Medium form

Excitement, anxiety, inadequate reactions to external stimulants, pronounced headache, muscle weakness are typical.

Breathing disorder becomes more intense both in the inspiratory act and in the expiratory act, the breathing becomes wheezing, “boiling”, wet rales are heard in the lungs throughout the entire lung area. Signs of respiratory failure (cyanosis) appear.

At this stage tachycardia can turn into bradycardia, BP remains high, hypertension accompanied by chills.

3. Severe form

A condition reminiscent of pulmonary edema: “boiling” breathing, numerous wet rales throughout the lung area, wet foamy cough associated with hypersecretion of bronchial glands.

The condition is aggravated by the appearance of paralysis of the intercostal muscles, breathing is based on the movement of the diaphragm and is similar to hiccups .

Short-term bradycardia turns into tachycardia, BP remains increased.

On this background the collaptoid state may appear: disturbed consciousness, narrowed pupils, do not react to light. Generalized convulsions occur periodically. A comatose state develops. At this stage, total inactivation of erythrocyte and serum cholinesterase occurs.

Death occurs as a result of asphyxia and decreased cardiac activity due to diffuse changes in the brain with predominance in the truncated and diencephalon.

Clinical picture of subacute intoxications

The symptoms of subacute intoxications with phosphororganic compounds are practically identical to acute intoxications. Subacute intoxications are less widespread compared to acute ones, the individual's ability to adapt and react to the body plays an important role, but also the amount and toxicity of the toxicant that has entered the human body. As with acute intoxication, the toxicant enters the body in one go, but the onset is slower.

Clinical picture of chronic intoxications

Stage I has a pronounced headache, predominantly in the temporal region, dizziness, decreased memory, sleep disorders, anorexia, nausea, general weakness, sometimes

miosis, disorder of the vegeto-vascular innervation with the predominance of the parasympathetic system.

Stage II shows a gradual decrease in intellect, short-term lipothemias may be present.

Stage III, especially in people who work with thiophos, it leads to livertoxicity. Determined in the general blood analysis: neutrophilic leukocytosis, toxic leukocyte granulation.

POISONING WITH CHLORORGANIC COMPOUNDS

These compounds are widely used in various branches of agriculture: as insecticides, acaricides, in seed processing, crops.

Organochlorine insecticides include: Chlorobenzene, Methoxychlor, Heptachlor, Chlordane, Polychlorinated, etc.

The particularities of these compounds are:

a) Environmental resistance;

b) High solubility in fats and lipids;

c) Ability to accumulate in the body tissues (fat-rich tissues: adipose tissue, brain, liver, pancreas, spleen, adrenal glands, thyroid).

Pathogenesis

The toxic action of chlororganic complexes is related to the modification of fermentative systems, interfering with the transmembrane transport of Na, K, Ca, Cl and the regulation of tissue respiration. Curciatov (1997) reffers to this group of compounds as fat-soluble non-electrolytes, which are able to penetrate all the body's protective barriers.

Toxic compounds act selectively on: CNS, reproductive system, parenchymal organs with their necrotic and fat degeneration, causing allergic sensitivity with irritant dermatitis.

The clinical symptomatology of acute and chronic intoxications with chlororganic compounds is characterized by a polymorphism of clinical signs and symptoms, which confirms their polytropism.

Clinical picture of acute intoxications with chlororganic compounds

The first clinical signs (after Aristotel Cocîrlă) appear between 30 min and up to 12 hours after the contact of the human body with the toxicant.

Clinical manifestations depend on the pathway of the toxicant into the body:

- When entering the respiratory tract, there are first signs of excitation of the upper respiratory tract and damage to the lower respiratory tract (bronchi) in the form of (acute tracheobronchitis).
- In case of penetration through the gastrointestinal tract - dyspeptic phenomena appear (nausea, vomiting, abdominal pain, accelerated transit), with the development of acute gastroenterocolitis.
- In case of penetration of the toxicant through the skin: it is accompanied by acute inflammation, hemorrhages and even tissue necrosis.

1.Light form:

Central Nervous System (CNS) Impairment: Toxic encephalitis with damage to the subcortical compartment, manifested by headache, vertigo, behavioral disorders, paresis, paresthesia of the extremities, tremor of the eyelids and upper extremities.

2. Medium form:

a) From the CNS, seizures may occur, sometimes epileptiform, colaptoid and comatose conditions.

b) Cardiovascular manifestations: cardiac pain, palpitations, dyspnea and chest

pressure feeling.

When large amounts of toxic substances enter the body, toxic-allergic myocarditis, toxic hepatitis and toxic nephritis can occur.

c) Hematopoietic system: sometimes, with repeated penetration of the toxicant, changes in the CBC it may appear as a hypo- and aplastic anemia

3. Severe form especially in intoxications with hexachloran or other analogous compounds may appear signs of damage to the peripheral nervous system with the development of vegeto-sensory polyneuritis. The pathological process in such cases diffusely affects the Nervous System (SN) as a encephalopolyneuritis, which has a rather unfortunate evolution.

Clinical picture of subacute intoxications with chlororganic compounds

Subacute intoxications with chlororganic compounds according to symptoms are practically identical to acute intoxications. Subacute intoxications are less widespread compared to acute ones, the individual's ability to adapt and react to the body is an important part of the process, but also the amount and toxicity of the toxicant that has entered the human body. As with acute intoxication, the toxicant enters the body in one go, but the onset is slower.

Clinical picture of chronic intoxications with chlororganic compounds

1. Stage I: is characterized by the development of astheno-vegetative or astheno-organic syndrome (microorganic symptoms, which indicate the location of the pathological process in the brainstem).

There are also signs of astheno-vegetative syndrome with angio-dystonic cerebral paroxysms: unexpectedly, headache occurs, accompanied by nausea, general weakness, hypertranspiration, dizziness, accessiform pallor, bradycardia.

2. Stage II: the clinical picture at this stage in the pathological process involves the peripheral nervous system with vegeto-sensory polyneuritis or mixed form.

In severe forms it is possible to diffuse damage to the NS (encephalopolyuritis) with organic symptoms, multiple sclerosis outbreaks, static disorders involved in the toxic process of the extrapyramidal and hypothalamic regions, auditory nerves, vegetative cervical lumps.

3. Stage III:

Nervous system disorders are accompanied by endocrine disorders (adrenal cortex secretion, pancreatic insulin system, thyroid hyperfunction).

Disorders of the cardiovascular system have a big significance: vegetative-vascular dystonia, hypo- or hypertension, myocardial dystrophy, toxic-allergic myocarditis.

POISONING WITH MERCURORGANIC COMPOUNDS

These pesticides are part of the group of chemicals with a high toxic effect, being very resistant and having the possibility to accumulate. Due to evaporation, most of them (Granozan, Mercuzan, Mercurhexane) are dangerous for people who are in contact with them.

Pathogenesis

When Mercuroorganic complexes enter the body, they bind to the –SH (sulfhydric) groups of cellular proteins, as a result of which the activity of the basic fermentative systems is disrupted, for the functioning of which free SH (sulfhydric) groups are needed. As a result, there are changes in the body with predominant CNS damage.

The capillarotoxic action of these substances has a very important role in the pathogenesis of intoxication. And, due to the peculiarities of mercuroorganic complexes to bind to cellular (tissue) proteins, forming the antigen-albumin complex because of which it is possible to have allergic reactions.

Clinical picture of acute intoxications

1.Mild form: the clinical picture of intoxication is characterized by the appearance of:

- The metallic taste
- Gingivitis
- Gastroenterocolitis
- Athteno-vegetative syndrome accompanied by dissociation between low muscle tone and exaggerated tendon reflexes.

2. Medium form:

Neurological disorders predominate: as acute toxic encephalitis with damage to the cerebellum, the truncated part of the brain, characterized by nystagmus, intentional tremor, positive Romberg picture.

Patients often develop myalgias, arthralgias, retrobulbar and trochlear neuritis, neuro-psychic disorders.

At this stage the pathological process is reversible, and in some cases has a recurrent character, with the aggravation of the general condition as a result of alcohol use, following the infections suffered, as well as other factors that influence the body.

3. The severe form of acute intoxication is characterized by:

Diffuse changes in the CNS, developing toxic encephalopolyneuritis. Important signs in the clinical picture of diencephalon damage: polyuria, adinamia, anorexia, progressive weight loss. The pathological process also includes the cranial nerves: oculomotor, facial, trigeminal, vagus, vestibulocochlear. Limb paralysis is possible due to damage to the peripheral nerves, pyramidal pathways and cerebellum.

Intentional tremor, dysarthria, ataxia appear when the cerebellum is affected. Some patients may have positive meningeal signs, limb hyperkinesia, epileptiform seizures.

The mental state often suffers: auditory and visual hallucinations, delusions,

schizophreniform syndrome.

It can also affect the cardiovascular system, for which capillary toxicosis, toxic myocardium or toxic-allergic myocardium are typical.

Toxic damage to the liver and kidneys are possible.

In the blood count: leukocytosis, accelerated ESR.

Clinical picture of subacute intoxications

- Subacute intoxications with mercuric compounds according to symptoms are practically identical to acute intoxications.
- Subacute intoxications are less widespread compared to acute ones, the individual's ability to adapt and react to the body is an important part of the process, but also the amount and toxicity of the toxicant that has entered the human body.
- As with acute intoxications, the toxicant enters the body in one go, but the onset is slower.

Clinical picture of chronic intoxications

1. Stage I is characterized by the appearance of astheno-vegetative syndrome and polyneuropathy.

On long term these pesticides increase the signs of neuro-vegetative dystonia and the tremor of the hands intensifies.

2. Stage II of intoxication in the clinical picture predomines the damage to the hypothalamic region: cachexia, vegetative-vascular crises.

3. Stage III:

First of all are signs of organic damage of the outbreak or diffuse of the CNS: toxic encephalopathy, rarely encephalopolyuritis.

When the spinal cord is included in the pathological process, the conduction pathways suffer, which leads to the appearance of spastic paraparesis, without sensitivity disorder. Disorders of the peripheral nervous system occur after those of the CNS.

Changes in sensitivity according to the polyneuritic type are accompanied by decreased carpo-radial and Achilles reflexes. The onset of polyneuritis is slow or progressive.

The pathological process at this stage acquires a resistant character.

In CSF (cerebrospinal fluid) the amount of protein and chlorides is low, and the level of glucose is increased.

In addition to CNS pathology, myocardial dystrophy is also associated, sometimes liver damage, which is distinguished by a progressive onset with the appearance of toxic jaundice.

Hypochromic anemia occurs often, at first moderate leukocytosis, then leukopenia occurs, in the leukocyte protoplasm toxic granulation is determined. The amount of platelets decreases, blood clotting is disturbed, ESR is accelerated.

The local action of mercuroorganic substances is poorly expressed, but long-term contact with them sometimes leads to the appearance of mucosal irritation, the development of blepharoconjunctivitis, allergic dermatitis.

The basic sign in intoxications with mercuroorganic substances is the presence of mercury in biological cultures: blood, urine, and in severe intoxications and in CSF.

TREATMENT OF PESTICIDE POISONING

At acute exposure of the human body to the toxicant, the first step is quickly stopping contact with the toxicant itself, aiming to restore the normal function of the contaminated organism.

When the toxicant enters by inspired air, it is necessary to transfer the patient from the polluted room, remove the clothes and destroy them, which would improve the respiratory act.

When it penetrates through the skin, the toxicant is removed with a cotton swab, the skin must be washed with warm water and soap or 2% sodium bicarbonate solution, wiped with 5-10% spirit solution or 2.5% chloramine solution. .

Removing the toxic from the stomach is performed by gastric lavage with warm water and absorbents (activated charcoal). For the extraction of the toxic from the intestine, purgative siphon enemas ($MgSO_4$) are applied.

The treatment is based on the following principles:

1. Administration of antidotes;

2. Pathogenetic treatment;

3. Symptomatic treatment.

The remedies used as antidotes can be medicinal preparations, which have the following properties:

a) to inactivate the toxicant in the blood,

b) to remove the toxic effect of its metabolites,

c) to speed up its excretion from the body.

For this purpose, physico-chemical preparations are used today to absorb the toxicant and decrease its absorption in the gastrointestinal tract (activated charcoal, amberlite). Lujnicov (1982) calls the property of making the toxic harmless with the help of physicochemical antidotes "gastrointestinal sorption".

The detoxifying action of some antidotes is based on the ability to intervene in chemical reactions with the toxicant or its metabolites, as a result of which the toxicant is inactivated, being eliminated from the body through urine and feces. This chemical group antitoxin - for parenteral administration refers to Unitiol and Succimeter.

Physiological antidotes are used in order to remove the toxic effect through antagonism on the same systems of the body, altering the metabolism of toxic complexes. The group of these antidotes includes: Methylene blue, Cholinolytics and

Cholinesterase Reactivators, Antioxidants. Antidotes can be administered as combinations of several preparations, mutually increasing the effect of the treatment.

Unitiol

(antidote in intoxications with chlororganic and mercuric compounds)

In case of acute intoxication, apply the antidote therapy with unitiol - proportions: 1 ml of 5% solution per 10 kg of the victim's weight according to the following scheme:

1. on the first day - 3-4 times a day, intramuscularly (i / m).

2. the next day - 2-3 times a day, i / m.

3. from the 3rd to the 7th day - 1-2 times a day, i / m.

In case of chronic intoxication, apply the antidote therapy with unitiol - 1 ml of 5% solution once a day for 7-10 days.

The combination with cholinolytics (atropine, scopolamine), which removes the muscarinic and nicotinic effects of phosphoroorganic complexes with cholinesterase reactivators (obidoxime, pralidoxime) is used in the treatment of intoxications with phosphororganic compounds.

Vitamins and aminoacids serve as antioxidants, which prevent the oxidation of the toxin and the formation of toxic derivatives and its transformation. Culaghin (2001) indicates the administration of Galascorbin and Alpha-tocopherol as antioxidants that not only reduce the signs of intoxication, but also normalizes the basic metabolic processes, liver function. Simultaneously with the administration of antidotes, pathogenic and symptomatic remedies are used in order to support and restore the function of organs and systems that have suffered from the toxin.

Atropine

(antidote in intoxications with phosphororganic compounds)

Atropine is the treatment base, we can reach the point of administration of huge doses (over 100 mg per day, meaning 100 ampoules).

In acute intoxications, mild form, 1-2 ml of 0.1% atropine sulphate solution is administered subcutaneously or intramuscularly; intravenous.

In the absence of a therapeutic effect or an increase in the intoxication symptoms, it is necessary to inject atropine intramuscularly(1-2 ml) every 1-2 hours until the poisoning symptoms disappear and the signs of overdose with atropine appear (dry mouth , blurred vision).

Pralidoxine

Its the substance capable of restoring the cholinesterase. It is part of the oxime group. Pralidoxime (2-PAM) regenerates cholinesterase by reversing phosphorylation. The loading dose for Pralidoxine is 30 mg / kg, then 8 mg / kg / h continuously.

The administration of oxime is only for patients with severe intoxications acusing acute respiratory failure, convulsions, coma. Pralidoxime is used in combination with atropine because atropine cannot regenerate cholinesterase.

If necessary, peritoneal dialysis, intestinal dialysis, hemodialysis, detoxifying hemosorption and blood replacement are possible. In the complex treatment, the basic methods that contribute to the increase of the resistance of the defense forces, would lead to the healing of the sufferings and the restoration of the work capacity. The treatment of intoxication recurrences is determined by the clinical basis of the syndrome (encephalopathic, polyneuritic, stable functional disorders in other systems).

Work capacity expertise

When establishing the work expertise in pesticide intoxications, the working conditions, the general and professional anamnesis, the clinical manifestations of the intoxication, the dynamics of the pathological process development will be taken into account.

In mild and moderate cases of intoxication the pathological process is usually reversible, the intoxication ends with full recovery, the work capacity is limited on a

period of time. But, at the appearance of high sensitivity to pesticides (compounds Hg, Cl), continuing to work with the same compounds is not recommended.

Sometimes in the moderately severe forms of intoxication asthenia and vegeto-vascular dystonia may still occur after a while. In this case it is not recommended to return to work, considering the influence of chemical preparations on the body.

Prolonged evolution or recurrences of toxic encephalopathy, damage to the peripheral nervous system, severe psycho-vegetative syndromes, these patients should not resume to working with pesticides, these being contraindicated along with other work related to the influence of other harmful factors on the body. Such patients require a rational work regime and, in case of decreased work quality, the medical commission must be advised to establish the work expertise. The level of work capacity decrease or IIIrd degree occupational disease associated disability is determined.

Severe forms of intoxication with diffuse CNS involvement, with involvement in the process of SCV, liver, blood usually lead to work capacity loss. These patients are diagnosed with group II or III disability work related occupational diseases. The decision of being in the disability group are established individually, taking into account the recurrences of intoxications.

Prevention

Paying the attention to sanitary rules at work, in the work process, in the storage and transportation of pesticides, their use in agriculture play an important role in the prophylaxis of pesticide poisoning.

Particular attention is required to the standards set for the return of field workers and objects processed with pesticides.

It is important to assess all occupational risk factors in the workplace, detailed job passporting is a key element in the management of occupational poisoning with pesticides.

One of the basic organisational measures for the prevention of pesticide poisoning is performing a medical control on people who have started work or those who work (or are in contact) with pesticides, early diagnosis of intoxication, treatment to increase the body's resistance.

KNOWLEDGE VERIFICATION QUESTIONS:

1. Define pesticides.

Pesticides are a series of chemicals with a particularly high biological action, intended and used in agriculture, forestry in order to prevent the action and / or combat some forms of plant or animal life, which cause direct and indirect damage to crops and

animals.

2. From which language does it come and what do the components of the word “pesticide” mean?

The term "pesticide" was taken from the English language, where "pest" means harmful insect, and the term "icide" means "to destroy, to kill."

3. How are pesticides classified?

I. Depending on the destination;

II. Depending on the origin;

III. Depending on the chemical structure;

IV. Depending on the degree of toxicity.

4. Depending on the destination of the pesticides into which groups are they divided?

1. Insecticides (pest control);

2. Zoocides (to control animal pests)

3. Rodenticides / Raticides (rodent control);

4. Molluscocides (mollusc control);

5. Nematocides (fighting nematodes);

6. Larvicides (fighting larvae);

7. Acaricides (pest control);

8. Ovicides (destruction of insect and mite eggs);

9. Algaecides (destruction of algae);

10. Herbicides (destruction of weeds in crops);

11. Fungicides (control of fungi that cause plant diseases);

12. Growth regulators (means that inhibit or stimulate plant growth processes):

    a) defoliation: means of defoliation of plants;

    b) desiccant: means of drying the plants before harvesting;

c) deflowering: means of removing the excessive amount of flowers;

d) attractive: means to lure;

e) repellents: means for rejection.

5. Depending on the origin of the pesticides, into which groups are they divided?

1. Mineral origin: (salts of As, Ba, Cu, Hg, Pb) etc.

2. Plant origin: (nicotine, veratrum, strychnine).

3. Synthetic origin: (organophosphorus esters, organohalogenated derivatives, aromatic nitroderivatives, carbamic derivatives, phenolic compounds and organometallic compounds).

6. Depending on the chemical structure of the pesticides, into which groups are they divided?

1.Phosphororganic pesticides

2. Chlororganic pesticides

3. Mercury organic pesticides

4. Carbamic and thiocarbamic pesticides

5. Nitrophenolic pesticides

6.Sulfur preparations

7. Copper preparations

8. Arsenic preparations

9. Alkaloids, etc.

7. Depending on the toxicity level of the pesticides, into which groups are they divided?

1.Group I: extremely toxic substances, induce fatal intoxications (lightning), LD50 <50 mg / kg, being marked with red labels.

2. Group II: substances with a strong toxic effect, LD50: 50-200 mg / kg, being marked with green labels.

3. Group III: substances with moderate toxic effect, LD50: 200-1000 mg / kg, being

marked with blue labels.

4. Group IV: substances with low toxic effect, LD50> 1000 mg / kg, being marked with black labels.

(LD50 - lethal dose value)

8. Trough which ways can toxicant enter the body?

1. The respiratory tract,

2. The cutaneous route,

3. Digestive tract.

9. According to the amount of toxic penetrated into the bodies, what types of intoxication do we know?

• Acute intoxications (mild, moderate, severe form)

• Subacute intoxications.

• Chronic intoxications (stage I, II, III)

10. What are the characteristics of acute pesticide poisoning?

They occur as a result of the penetration into the body of a large amount of pesticides. Characteristic being:

 The early period, which includes the period of time from the penetration of the toxic in the body, until the appearance of the first signs of intoxication.

 The preclinical period, for which non-specific symptoms are characteristic such as: vomiting, nausea, headache, general weakness, which can be found in other pathologies.

 The period of intoxication itself, has a series of specific clinical signs that appear as a result of the action of the toxic on the body.

11. What are the characteristics of subacute pesticide poisoning?

It depends on the amount and toxicity of the toxicant that has entered the human body. It has an insignificant reaction to the action of the toxicant on the body, compared to the reaction in acute intoxications, the pathological process having a more diminished and longer evolution.

12. What are the characteristics of chronic pesticide poisoning?

They occur as a result of small and repeated doses of the toxicant on the human body over time.

13. In which group do phosphorus-organic compounds belong to according to the chemical structure?

According to the chemical structure, phosphororganic compounds are ethers of phosphoric, thiophosphoric, dithiophosphoric and phosphonic acids.

14. What representatives of phosphororganic compounds do you know?

The representatives of this group are: Carbofos, Fazolon, Fosfamid, Methylnitrofos, Metafos.

15. For what purpose and where are phosphororganic compounds used?

It is used for insecticides or acaricides in agriculture, fruit growing, viticulture and forestry, with intense effects even at low doses.

16.Which enzyme is primarily inhibited by organophosphorus compounds when they enter the body?

It is known that many organophosphorus compounds can inhibit both cholinesterase and other enzymes.

17. What happens after acetylcholinesterase inactivation?

As a result of the inactivation of acetylcholinesterase, the mediator of the Nervous System (synapse) - acetylcholine, "endogenous acetylcholine intoxication" accumulates, which leads to the nerve impulse transmission disorder through nerve cells and ganglion synapses.

18. The basic symptoms of phosphoroorganic pesticide poisoning are determined by the presence of whose effects?

The basic symptoms of intoxication with phosphoroorganic pesticides are determined by the presence of:

1. The muscarinic effect,

2. The nicotinic effect,

3. The central action of acetylcholine.

19. What is the muscarinic effect (parasympathomimetic action) in organophosphorus pesticide poisoning?

1. Ocular effects: miosis, decreased visual acuity and accommodation disorders.

2. Cardiovascular effects: short-term decrease in blood pressure, on the heart it has an inotropic, chronotropic, tonotropic, dromotropic negative effect prolonging the P-Q interval; stimulates the excitability of the myocardium, especially the atrial one and predisposes to atrial arrhythmias; bradycardia.

3. Effects on smooth muscles, contracts the muscles:

• bronchi;

• the gastrointestinal tract, including the intra- and extrahepatic bile ducts;

• ureters, bladder;

• the uterus and fallopian tubes.

4. Effects on exocrine secretion, stimulates the secretion (increases the secretion of sweat, tear, salivary, bronchial, gastric, pancreatic and intestinal glands).

20. What is the nicotine effect of organophosphorus pesticide poisoning?

It contains the impulse transmission disorder in the postganglionic fiber, which determines:

1. Hypertension (by generalized vasoconstriction).

2. Tachycardia (by acting on the sympathetic nodes and releasing catecholamines from the adrenal medulla).

3. Apnea (by reflex mechanism starting from the large pulmonary vessels), followed by polypnea (by excitation of sinocarotid receptors).

4. Hyperglycemia (by releasing catecholamines).

5. Muscle fasciculations (by acting on nicotinic receptors and at the level of the neuromuscular junction): eyelid contractions, tongue contractions, neck contractions.

21. What is the central action in organophosphorus pesticide poisoning?

It is determined by the toxic action of acetylcholine on the cerebral cortex and spinal bulb, which is manifested by:

- Headache,
- Anxiety,
- Vertije,
- Insomnia,
- Balance disorders,
- Excitement,
- Psychological disorders,
- Knowledge disorder,
- Convulsions, coma,
- Paralysis of the centers of vital importance in the spinal bulb.

22.Describe the clinical picture of acute intoxications with phosphororganic pesticides, mild form.

It has the following accusations: headache, dizziness, weakness in the limbs, decreased vision, anxiety, nausea, hypersalivation, abdominal colic, diarrhea.

Patients are worried, pupils narrowed, the reaction to low light, the spasm of accommodation develops, which leads to decreased visual acuity, night adaptation disorder and difficulties in dimly lit rooms.

Nystagmus, edema of the face, hypertranspiration also occur.

Prolonged action is difficult breathing (predominantly in the inspiratory act), chest discomfort accompanied by respiratory failure, cough attacks.

Rough breathing, dry rales are heard throughout the lung area.

Cardiovascular system: tachycardia, increased BP.

There is a marked decrease in the erythrocyte cholinesterases activity in the blood serum.

23. Describe the clinical picture of acute intoxications with phosphororganic pesticides, the moderate form.

Excitement, anxiety, inadequate reactions to external stimulants, intense headache, muscle weakness are typical.

Breathing disorder is worsening both in the inspiratory act and in the expiratory act, the breathing becomes hissing, boiling, wet rales in the lungs are heard throughout the entire lung area. Signs of respiratory failure (cyanosis) appear.

At this stage tachycardia can turn into bradycardia, BP remains high, hypertension accompanied by chills.

24. Describe the clinical picture of acute intoxications with phosphororganic pesticides, the severe form.

A condition reminiscent of pulmonary edema: boiling breathing, numerous wet rales throughout the lung area, foamy wet cough accompanied by hypersecretion of bronchial warts.

The condition is aggravated through paralysis of the intercostal muscles, breathing is based on the diaphragm movement and is hiccup like.

Short-term bradycardia turns into tachycardia, BP remains increased.

Because of the collaptoid state, the following may appear: disturbed consciousness, narrowed pupils, do not react to light. Generalized convulsions occur periodically. A comatose state develops. At this stage, total inactivation of erythrocyte and serum cholinesterase occurs.

Asphyxia and decreased cardiac activity on the background of diffuse changes in the brain, predominantly in the truncated and diencephalon, can lead to death.

25. Describe the clinical picture of subacute intoxications with phosphororganic pesticides.

Subacute intoxications with phosphororganic compounds according to symptoms are practically identical to acute intoxications. Subacute intoxications are less common than acute ones, the individual's ability to adapt and react to the body has an important role, but also by the amount and toxicity of the toxicant that has entered the human body. As with acute intoxication, the toxicant enters the body in one go, but the onset is slower.

26.Describe the clinical picture of chronic intoxications with phosphororganic pesticides, stage I.

There is an intense headache, predominantly in the temporal region, dizziness, decreased memory, sleep disorders, anorexia, nausea, general weakness, can sometimes cause miosis, disorders of the vegeto-vascular innervation, predominantly

in the parasympathetic system.

27. Describe the clinical picture of chronic intoxications with phosphororganic pesticides, stage II.

There is a gradual decrease in the intellectual state, short-term lipothemias may be present.

28. Describe the clinical picture of chronic intoxications with phosphororganic pesticides, stage III.

Occurs especially in people who work with thiophos, leads to a toxic damage to the liver. Determined in the general blood analysis: neutrophilic leukocytosis, toxic leukocyte granulation.

29.What are the purposes of chlororganic compounds in different agriculture branches ?

These compounds are widely used in various branches of agriculture: as insecticides, acaricides, in seed processing, crops.

30.Name some representatives from the organochlorine compounds group .

Organochlorine insecticides include: Chlorobenzene, Methoxychlor, Heptachlor, Chlordane, Polychlorinated, etc.

31. What is the particularity of chlororganic compounds?

The particularity of these compounds is:

a) Environmental resistance;

b) High solubility in fats and lipids;

c) Ability to accumulate in the body tissues (fat-rich tissues: adipose tissue, brain, liver, pancreas, spleen, adrenal glands, thyroid).

32. What is specific to the toxic action of chlororganic complexes?

The toxic action of chlororganic complexes is associated to the modification of the fermentative systems, interfering with the transmembrane transport of Na, K, Ca, Cl and the tissue respiration disorder.

33. How does Curceatov describe chlororganic compounds?

Curciatov (in 1997) defines this group of compounds as fat-soluble non-electrolytes,

which are able to penetrate all the body's protective barriers.

34.On which systems and organs do chlororganic compounds act?

The compounds act selectively acting on: CNS, reproductive system, parenchymal organs with their necrotic and fatty degeneration, causing allergic reactions with irritated dermatitis.

35.Describe the clinical picture of acute intoxications with chlororganic pesticides.

The first clinical signs (after Aristotel Cocîrlă) appear between 30 min and up to 12 hours after the contact of the human body with the toxicant.

Clinical manifestations depend on the pathway how the toxicant enters the body:

- When entering the respiratory tract, the first signs of arousal of the upper respiratory tract appear and lead to damage to the lower respiratory tract (bronchi) as an (acute tracheobronchitis).
- In case of penetration through the gastrointestinal tract - dyspeptic phenomena appear (nausea, vomiting, abdominal pain, accelerated transit), developing acute gastroenterocolitis.
- In case of penetration of the toxicant through the skin, acute inflammation appears, hemorrhages and even tissue necrosis.

36. Describe the clinical picture of acute intoxications with chlorogenic pesticides, the mild form.

Central Nervous System (CNS) Impairment: Toxic encephalitis with damage to the subcortical compartment, marked by headache, vertigo, behavior disorders, paresis, paresthesia of the extremities, tremor of the eyelids and upper extremities.

37. Describe the clinical picture of acute intoxications with chlororganic pesticides, the moderate form.

a) CNS: seizures may occur, sometimes epileptiform seizures, colaptoid and comatose.

b) Cardiovascular manifestations: cardiac pain, palpitations, dyspnoea and chest pressure sensation.

When large amounts of toxic substances enter the body, toxic-allergic myocarditis, toxic hepatitis and toxic nephritis can occur.

c) Hematopoietic system: sometimes, repeated penetration of the toxicant, the blood count may show changes like hypo- and aplastic anemia

38.Describe the clinical picture of acute intoxications with chlororganic pesticides, the severe form.

Severe form develops especially in hexachloran intoxications or other similar compounds. May show signs of damage to the peripheral nervous system developing vegeto-sensory polyneuritis. The pathological process in such cases diffusely affects the Nervous System (SN) - encephalopolyneuritis, which has a rather unfavorable evolution.

39.Describe the clinical picture of subacute intoxications with chlororganic pesticides.

Subacute intoxications with chlororganic compounds according to the symptoms are practically identical to acute intoxications. Subacute intoxications are less widespread compared to acute ones, the individual reaction ability of the body is an important part, but also to the amount and toxicity of the toxicant that has entered the human body. Similar to acute intoxication, the toxicant enters the body in one go, but the onset is slower.

40.Describe the clinical picture of chronic intoxications with chlororganic pesticides, stage I.

Developing astheno-vegetative or astheno-organic syndrome (microorganic symptoms, which indicate the location of the pathological process is in the brainstem).

There are also signs of astheno-vegetative syndrome with angio-dystonic cerebral paroxysms: unexpectedly, headache occurs, accompanied by nausea, general weakness, hypertranspiration, dizziness, accessiform pallor, bradycardia.

41.Describe the clinical picture of chronic intoxications with chlororganic pesticides, stage II.

The clinical picture at this stage in the pathological process involves peripheral nervous system with vegeto-sensory polyneuritis or mixed form.

In severe forms it is possible to diffuse the damage to the SN (encephalopolyuritis) with organic symptoms, multiple sclerosis outbreaks, static disorders involving the toxic process of the extrapyramidal and hypothalamic regions, auditory nerves, vegetative cervical nodes.

42. Describe the clinical picture of chronic intoxications with chlororganic pesticides, stage III.

Nervous system disorders are accompanied by endocrine disorders (adrenal cortex secretion, pancreatic insulin system, thyroid hyperfunction).

Cardiovascular system disorders play an important role: vegetative-vascular dystonia, hypo- or hypertension, myocardial dystrophy, toxic-allergic myocarditis.

43. What is the characteristic of mercury-organic pesticides?

Pesticides in this group are part of the chemicals with a high toxic effect, they are resistant and have accumulation capacity. Due to evaporation, most of them (Granozan, Mercuzan, Mercurhexane) are dangerous for people who are in contact with them.

44. Why do changes occur in the body, predominantly Central Nervous System damage in intoxications with mercuric compounds?

When mercuroorganic complexes enter the body, they bind to the –SH (sulfhydric) groups of cellular proteins, as a result of which the activity of the basic fermentative systems is disrupted, for the functioning of which free SH (sulfhydric) groups are needed. As a result, there are changes in the body with predominant CNS damage.

45. Are capillary toxicity and allergic reactions specific to intoxications with mercuric compounds?

Yes. The capillarotoxic action of these substances has a very important role in the pathogenesis of intoxication. And, due to the peculiarities of mercuroorganic complexes to bind to proteins (tissue), forming the antigen-albumin complex as a result of which allergic reactions are possible.

46.Describe the clinical picture of acute intoxications with mercuric pesticides, the mild form.

The clinical picture of intoxication is characterized by the appearance of:

- The metallic taste
- Gingivitis
- Gastroenterocolitis
- Asteno-vegetative syndrome with dissociation between low muscle tone and exaggerated tendon reflexes.

47. Describe the clinical picture of acute intoxications with mercuric pesticides, the moderate form.

Neurological disorders predominate: acute toxic encephalitis with damage to the cerebellum, the truncated part of the brain, characterized by nystagmus, intentional tremor, positive Romberg picture.

Often develop myalgias, arthralgias, retrobulbar and trochlear neuritis, neuro-psychic disorders.

At this stage the pathological process is reversible, and in some cases it has a recurrent character. The general condition can be worsening as a result of alcohol use, infections, as well as other factors that influence the body.

48.Describe the clinical picture of acute intoxications with mercuric pesticides, the severe form.

There are vague changes in the CNS, developing toxic encephalopolyneuritis. Predominantly in the clinical picture of the diencephalon damage: polyuria, adinamia, anorexia, progressive weight loss. The pathological process also includes the cranial nerves: ocolomotor, facial trigeminal, vague, vestibulo-cochlear. Limb paresis is possible due to damage to the peripheral nerves, pyramidal pathways and cerebellum.

Intentional tremor, dysarthria, ataxia appear when the cerebellum is affected. Some patients may have positive meningeal signs, limb hyperkinesia, epileptiform seizures.

Patient often suffers from a mental state disorders: auditory and visual hallucinations, delusions, schizophreniform syndrome.

It can also affect the cardiovascular system, for which capillary toxicosis, toxic myocardium or toxic-allergic myocardium are typical.

It can lead to toxic damage to the liver and kidneys.

In the blood count: leukocytosis, accelerated ESR.

49.Describe the clinical picture of subacute mercuric pesticide poisoning.

• Subacute intoxications with mercuric compounds according to the symptoms are practically identical to acute intoxications.

• Subacute intoxications are less widespread compared to acute ones,the individual's ability to adapt and react to the body is an important aspect, but also the amount and toxicity of the toxicant that has entered the human body.

• With acute intoxications, the toxicant enters the body in one go, but the onset is slower.

50.Describe the clinical picture of chronic intoxications with mercuric pesticides, stage I.

Usually astheno-vegetative syndrome and polyneuropathy appear. Long term, the action of these pesticides lead to increased neuro-vegetative dystonia and intensified tremor of the hands.

51.Describe the clinical picture of chronic intoxications with mercuric pesticides, stage II.

Clinical picture shows signs of damage to the hypothalamic region, predominantly: cachexia, vegetative-vascular crises.

52.Describe the clinical picture of chronic mercuric pesticide poisoning, stage III.

First of all come the signs of organic outbreak or diffuse CNS: toxic encephalopathy, rarely encephalopolyuritis.

When the spinal cord is included in the pathological process, the conduction pathways suffer, which leads to spastic paraparesis, without sensitivity disorder. Disorders of the peripheral nervous system occur after those of the CNS.

Changes in sensitivity according to the polyneuritic type are accompanied by decreased carpo-radial and Achilles reflexes. The onset of polyneuritis is slow or progressive.

The pathological process at this point has a resistant character.

In CSF (cerebrospinal fluid) the amount of protein and chlorides is low, and the level of glucose is increased.

In addition to CNS pathology, myocardial dystrophy is also associated, sometimes liver damage, which is distinguished by a progressive onset with the appearance of toxic jaundice.

Often hypochromic anemia occurs, at first moderate leukocytosis, then leukopenia occurs, in the leukocyte protoplasm toxic granulation is determined. The amount of platelets decreases, blood clotting is disturbed, ESR is accelerated.

The local action of mercuroorganic substances is poorly expressed, but long-term contact can lead to the appearance of mucosal irritation, development of

blepharoconjunctivitis, allergic dermatitis.

The basic sign in intoxications with mercuroorganic substances is the presence of mercury in biological environments: blood, urine, and in severe intoxications and in CSF.

53.What is the purpose of stopping the contact with the toxicant in case of acute exposure?

In case of acute exposure of the human body to the toxicant, the first step is quickly stopping contact with the toxicant itself, with the purpose of restoring the normal function of the contaminated organism.

54. What to do if the toxicant has penetrated the inspired air?

When the toxicant enters trough inspired air, it is necessary to transfer the patient from the polluted room, remove the clothes and destroy them, which would improve the respiratory act.

55. What to do if the toxicant has penetrated the skin?

When penetrating through the skin, the toxicant is removed with a cotton swab, skin is washed with warm water and soap or 2% sodium bicarbonate solution, wiped with 5-10% spirit solution or 2.5% chloramine solution. .

56. What to do if the toxin has entered the digestive tract?

Removing the toxic from the stomach is done by gastric lavage with warm water and absorbents (activated charcoal). For the extraction of the toxic from the intestine, purgative siphon enemas ($MgSO_4$) are applied.

57.Name the principles underlying treatment for pesticide poisoning.

The treatment is based on the following principles:

1.Antidotes administration ;

2.Pathogenetic treatment;

3. Symptomatic treatment.

58. What properties must the antidotes used in the treatment of pesticide poisoning have?

Medicinal preparations can be used as remedies/antidotes, they have the following properties:

a) to inactivate the toxicant in the blood,

b) to remove the toxic effect of its metabolites,

c) to speed up its excretion from the body.

59. What properties does the antidotes Unitiol and Succimeter have in the treatment of pesticide poisoning?

The detoxifying action of some antidotes is based on the ability to intervene in chemical reactions with the toxicant or its metabolites, as a result of which the toxicant is inactivated. It is eliminated from the body through urine and fecal masses. This chemical group antitoxin - for parenteral administration it refers to Unitiol and Succimeter.

60. What physiological antidotes do you know and how are they used in the treatment of pesticide poisoning?

Physiological antidotes are used in order to remove the toxic effect through antagonism on the same systems of the body, altering the metabolism of toxic complexes. This group of antidotes includes: Methylene blue, Cholinolytics and Cholinesterase Reactivators, Antioxidants. Antidotes can be administered as combinations of several preparations, mutually increasing the effect of the treatment.

61.What is the purpose of Unitiol in the treatment of pesticide poisoning?

Unitiol is used as an antidote in intoxications with chlororganic and mercuric compounds.

62.How is Unitiol used in the treatment of acute pesticide poisoning?

In case of acute intoxication, perform an antidote therapy with unitiol - proportions: 1 ml of 5% solution per 10 kg of the victim's weight according to the following scheme:

4. on the first day - 3-4 times a day, intramuscularly (i / m).

5. the next day - 2-3 times a day, i / m.

6. from the 3rd to the 7th day - 1-2 times a day, i / m.

63.How is Unitiol used in the treatment of chronic pesticide poisoning?

In case of chronic intoxication, an antidote therapy with unitiol - 1 ml of 5% solution once a day for 7-10 days.

64.What is the purpose of the combination with cholinolytics used in the

treatment of pesticide poisoning?

The combination with cholinolytics (atropine, scopolamine), which removes the muscarinic and nicotinic effects of phosphoroorganic complexes with cholinesterase reactivators (obidoxime, pralidoxime), is used in the treatment of intoxications with phosphororganic compounds.

65. What antioxidants are used in the treatment of pesticide poisoning?

Vitamins and aminoacids serve as antioxidants, which prevent the oxidation of the toxin and the formation of toxic derivatives, or its transformation.

66. For what purpose is Atropine used in the treatment of pesticide poisoning?

Atropine is the antidote used in intoxications with phosphororganic compounds.

67. What doses of Atropine are used in the treatment of phosphororganic pesticide poisoning?

Atropine is the basic treatment, it can be administered in huge doses (over 100 mg per day, meaning 100 ampoules).

68.How is Atropine used in the treatment of phosphororganic pesticide poisoning?

In acute intoxications, mild form, 1-2 ml of 0.1% atropine sulphate solution is administered subcutaneously or intramuscularly; intravenous.

69.Until when do we give Atropine in the treatment of phosphororganic pesticide poisoning?

Until the poisoning symptoms disappear and the atropine overdose signs appear (dry mouth, blurred vision).

70. What is Pralidoxine, what group does this medicine belong to and what contribution does it have to the treatment of pesticide poisoning?

It is the substance that is capable of restoring cholinesterase. It is part of the oxime group. Pralidoxime (2-PAM) regenerates cholinesterase by reversing phosphorylation.

71.What is the loading dose of Pralidoxine in the treatment of pesticide poisoning?

The loading dose for Pralidoxine is 30 mg / kg, then 8 mg / kg / h continuously.

72.What is the purpose of Pralidoxine in the treatment of pesticide poisoning?

The administration of oximes is reserved for patients with severe intoxications manifested by acute respiratory failure, convulsions, coma.

73.Which medicinal preparations are associated with the use of Pralidoxine in the treatment of pesticide poisoning?

Pralidoxime is used in combination with atropine because atropine cannot regenerate cholinesterase.

74.What will be taken into account in the work capacity expertise in pesticide poisonings?

When establishing the work expertise in pesticide intoxications, the working conditions, the general anamnesis and the professional path, the clinical manifestations of the intoxication, the development of the pathological process will be taken into account.

75. What are the cases of mild and medium severity in work capacity expertise, in pesticide poisonings?

In mild and moderate cases of intoxication, the pathological process is usually reversible, the intoxication ends with full recovery, the work capacity is limited for a period of time. But, when a high sensitivity to pesticides is noticed (compounds Hg, Cl), resuming work with the same compounds is not recommended.

76.What do the cases of medium gravity in work capacity expertise, in the intoxications with pesticides, include?

In case of prolonged evolution or toxic encephalopathy recurrences, peripheral nervous system damage, severe psycho-vegetative syndromes, the patients should not resume work with pesticides, these being contraindicated and other work related to the influence of other harmful factors on the body. Such patients require rational work regime and in case of decrease in work quality , patient is directed to the medical commission for establishing the work expertise. The level of work capacity loss or occupational disease IIIrd degree disability is determined.

77.What do the serious cases in the work capacity expertise, in the pesticide poisonings, foresee?

Severe forms of intoxication with diffuse CNS damage, involving the SCV process, liver, blood usually lead to stable work capacity loss. These patients are diagnosed with group II or III occupational diseases related disability. The terms the disability group are established individually, taking into account the intoxications recurrences of

each patient.

78. What is the importance of complying with health rules in the prevention of pesticide poisoning?

An important role in the prophylaxis of pesticide intoxications is paying attention to sanitary rules. Usually the lack of adequate sanitary equipment and behaviour when working with pesticides leads to intoxications at work, in the work process, storage and transportation of pesticides, in agriculture.

79.Is the declaration of all occupational risk factors a measure of prophylaxis of pesticide poisoning?

It is important to assess all occupational risk factors in the workplace, detailed job passporting is a key element in the management of occupational poisoning with pesticides.

80. What is the importance of medical control in the prevention of pesticide poisoning?

Among the basic organizational measures related to the prevention of pesticide poisoning, there is the medical control on people who have started work or those who work (or are in contact) with pesticides, early diagnosis of intoxication, treatment to increase the body's resistance.

KNOWLEDGE VERIFICATION TEST:

1. Give the definition of pesticides.

a. [*] Pesticides are a series of chemicals with a particularly high biological action, intended and used in agriculture, forestry in order to prevent the action and / or control of plant or animal life, which cause direct and indirect damage to crops agricultural and animal health.

b. [] Pesticides are a series of chemicals with no high biological action, intended and used in agriculture, forestry to prevent and / or combat plant or animal life, which cause direct and indirect damage to crops, and animals.

c. [] Pesticides are a series of substances that do not cause direct or indirect damage to crops and animals.

d. [] Pesticides are a series of chemicals used only in the synthetic chemical industry.

e. [] Pesticides are a series of chemicals with a particularly high biological action, intended and used only in forestry for the purpose of preventing animal damage, and which cause direct damage to wild animals.

2. From which language was it taken and what do the components of the word "pesticide" mean?

a. [*] The term "pesticide" was taken from the English language, where "pest" means harmful insect, and the term "icide" means "to destroy, to kill".

b. [] The term "icide" was taken from the Greek, where "pest" means harmful insect, and the term "pesticide" means "to destroy, to kill".

c. [] The term "pesticide" was taken from the English language, where "pest" means

“to destroy, to destroy”, and the term “icide” means harmful insect.

d. [] The term “pesticides” was taken from the French language, where “pest” means harmful insect and the term “icide” means chemical ”.

e. [] The term “pesticide” was taken from the English language, where “pest” is detected, and the term “icide” means “to stop”.

3. In how many groups are pesticides classified and what are they?

a. [*] I. Depending on the destination.

b. [*] II. Depending on the origin.

c. [*] III. Depending on the chemical structure.

d. [] IV. Depending on the manufacturer.

e. [*] IV. Depending on the degree of toxicity.

4. Depending on the destination of the pesticides into which groups are they divided?

a. [*] Insecticides (pest control).

b. [*] Zoocides (for controlling animal pests).

c. [*] Rodenticides / Raticides (rodent control).

d. [*] Molluscocides (mollusc control).

e. [] Molluscocides (pest control).

5. Depending on the destination of the pesticides into which groups are they divided?

a. [*] Nematocides (fighting nematodes).

b. [*] Larvicides (fighting larvae).

c. [] Acaricides (fighting nematodes).

d. [*] Ovicides (destruction of eggs by insects and mites).

e. [*] Acaricides (mite control).

6. Depending on the destination of the pesticides into which groups are they divided?

a. [*] Algaecides (destruction of algae).

b. [*] Herbicides (destruction of weeds in crops).

c. [*] Fungicides (control of fungi that cause plant diseases).

d. [] Acaricides (fighting nematodes).

e. [] Molluscocides (pest control).

7. Growth regulators (means that inhibit or stimulate plant growth processes) include:

a. [*] defoliating: means of defoliating plants.

b. [*] desiccant: means of drying plants before harvesting.

c. [*] deflowering: means of removing the excessive amount of flowers.

d. [*] attractive: means to lure.

e. [] repellents: means for luring.

8. Depending on the origin of the pesticides into which groups are they divided?

a. [*] Mineral origin: (salts of As, Ba, Cu, Hg, Pb) etc.

b. [*] Plant origin: (nicotine, veratrum, strychnine).

c. [*] Synthetic origin: (organophosphorus esters, organohalogenated derivatives, aromatic nitroderivatives, carbamic derivatives, phenolic compounds and organometallic compounds).

d. [] Mineral origin: (nicotine, veratrum, strychnine) etc.

e. [] Mineral origin: (organophosphorus esters, organohalogenated derivatives, aromatic nitroderivatives, carbamic derivatives, phenolic compounds and organometallic compounds).

9. Depending on the chemical structure of the pesticides into which groups are they divided?

a. [*] Phosphororganic pesticides

b. [*] Chlororganic pesticides

c. [*] Mercurorganic pesticides

d. [*] Carbamic and thiocarbamic pesticides, respectively

e. [] Selenorganic pesticides

10. Depending on the chemical structure of the pesticides into which groups are they divided?

a. [*] Nitrophenolic pesticides

b. [*] Sulfur preparations

c. [*] Copper preparations

d. [*] Arsenic preparations

e. [] Alcohol.

11. Depending on the toxicity degree of the pesticides into which groups are they divided?

a. [*] Group I: extremely toxic substances, induce deadly poisoning (lightning), LD50 <50 mg / kg, being marked with red labels.

b. [*] Group II: substances with a strong toxic effect, LD50: 50-200 mg / kg, being marked with green labels.

c. [] Group III: substances with moderate toxic effect, LD50: 200-1000 mg / kg, being marked with green labels.

d. [*] Group IV: substances with a reduced toxic effect, LD50> 1000 mg / kg, being marked with black labels.

e. [*] Group III: substances with moderate toxic effect, LD50: 200-1000 mg / kg, being marked with blue labels.

12. What are the pathways trough which the toxicant can enter the body?

a. [*] Respiratory tract.

b. [*] The cutaneous route.

c. [*] Digestive tract.

d. [] The parenteral route.

e. [*] Mixed route.

13. According to the amount of toxic penetrated into the bodies, what types of intoxications can we differentiate?

a. [*] Acute intoxications (mild, moderate, severe form).

b. [*] Subacute intoxications.

c. [*] Chronic intoxications (stage I, II, III).

d. [] Acute intoxications (stage I, II, III).

e. [] Subacute intoxications (stage I, II, III).

14. What are the characteristics of acute pesticide poisoning?

a. [*] The early period, from the penetration of the toxicant in the body, until the first intoxication signs appear.

b. [*] The preclinical period, for which non-specific symptoms are characteristic such as: vomiting, nausea, headache, general weakness, which can be found in other pathologies.

c. [*] The period of intoxication itself, has a series of specific clinical signs that are the result of the toxic's action on the body.

d. [] The early period, for which nonspecific symptoms are characteristic such as: vomiting, nausea, headache, general weakness, which can be found in other pathologies.

e. [] The period of intoxication itself, for which nonspecific symptoms are characteristic such as: vomiting, nausea, headache, general weakness, which can be found in other pathologies.

15. What are the characteristics of subacute pesticide poisoning?

a. [*] Depends on the amount and toxicity of the toxicant that has entered the human body.

b. [*] It is defined by an insignificant reaction to the action of the toxicant on the organism.

c. [*] Compared to the reaction of acute intoxications, the pathological process has a more mild and longer evolution.

d. [*] Compared to the reaction of acute intoxications, the pathological process has a more accentuated and longer evolution.

e. [] It is defined by a significant reaction to the action of the toxicant on the organism.

16. What are the characteristics of chronic pesticide poisoning?

a. [*] Occur as a result of over time summed up effects of small and repeated doses of the toxicant on the human body.

b. [] They occur as a result of the summation over time of the effects of high and repeated doses of the toxicant on the human body.

c. [] They occur as a result of summing up over time the effects of small and single doses of the toxicant on the human body.

d. [] Occur spontaneously with the action of small and repeated doses of toxic to the human body.

e. [] Occur as a result of an accident without repeating over time.

17. Which phosphorus-organic compounds belong to the chemical structure?

a. [*] According to the chemical structure, phosphororganic compounds are ethers of phosphoric, thiophosphoric, dithiophosphoric and phosphonic acids.

b. [] According to the chemical structure, phosphororganic compounds are alcohols.

c. [] According to the chemical structure, phosphororganic compounds are strong bases.

d. [] According to the chemical structure, phosphororganic compounds are chloride derivatives.

e. [] According to the chemical structure, phosphororganic compounds are benzene derivatives.

18.What representatives of phosphororganic compounds do you know?

a. [*] The representatives of this group are: Carbofos, Fazolon, Methylnitrofos, Metafos.

b. [] The representatives of this group are: Chlorbophos, Metichlorophos, Metamercurophos.

c. [] The representatives of this group are: Arsenite, Mercury.

d. [] The representatives of this group are: Trichlorophosphogem.

e. [] The representatives of this group are: Phosphamidchlorate, Metaphosparsenate.

19. For what purpose and where are phosphororganic compounds used?

a. [*] Used for insecticide or acaricide purposes in agriculture, fruit growing, viticulture and forestry, with intense effects even at low doses.

b. [] Used for animal killings in agriculture, fruit growing, viticulture and forestry, with intense effects even at low doses.

c. [] Used for algicidal purposes in agriculture, fruit growing, viticulture and forestry, with intense effects even at low doses.

d. [] Used for mycelicide purposes in agriculture, fruit growing, viticulture and forestry, with intense effects even at low doses.

e. [] Used for fumigation purposes in agriculture, fruit growing, viticulture and forestry, with intense effects even at low doses.

20.Which enzyme is primarily inhibited by organophosphorus compounds when they enter the body?

a. [*] It is known that many organophosphorus compounds can inhibit both cholinesterase and other enzymes.

b. [] It is known that many organophosphorus compounds can inhibit both alcohol dehydrogenase and other enzymes.

c. [] It is known that many organophosphorus compounds can inhibit both synthases and other enzymes.

d. [] It is known that many organophosphorus compounds can inhibit both ATPases and other enzymes.

e. [] It is known that many organophosphorus compounds cannot inhibit both cholinesterase and other enzymes.

21. What happens after acetylcholinesterase inactivation?

a. [*] As a result of inactivation of acetylcholinesterase, the mediator of the Nervous System (synapse) - acetylcholine accumulates.

b. [*] Endogenous acetylcholine intoxication leads to disorders of nerve impulse transmission through nerve cells and ganglion synapses.

c. [] Endogenous acetylcholine intoxication does not lead to disorder of nerve impulse

transmission through nerve cells and ganglion synapses.

d. [] As a result of inactivation of acetylcholinesterase, the mediator of the Nervous System (synapse) - dopamine accumulates.

e. [] As a result of inactivation of acetylcholinesterase, the mediator of the Nervous System (synapse) - acetylcholine does not accumulate.

22. The basic symptoms of phosphoroorganic pesticide poisoning are determined by the presence of whose effects?

a. [*] Muscarinic effect.

b. [*] The nicotinic effect.

c. [*] The central action of acetylcholine.

d. [] Neurological effect.

e. [] Peripheral action of acetylcholine.

23. What is the muscarinic effect (parasympathomimetic action) in organophosphorus pesticide poisoning?

a. [*] Ocular effects: miosis, decreased visual acuity and accommodation disorders.

b. [*] Cardiovascular effects: short-term decrease in blood pressure.

c. [*] Effects on smooth muscles.

d. [*] Effects on exocrine secretion, stimulates secretion (increases the secretion of sweat, tear, salivary, bronchial, gastric, pancreatic and intestinal glands).

e. [] Eye effects: mydriasis and increased visual acuity.

24. What determines the nicotinic effect in organophosphorus pesticide poisoning?

a. [*] Hypertension (by generalized vasoconstriction).

b. [*] Tachycardia (by acting on the sympathetic nodes and releasing of catecholamines from the adrenal medulla).

c. [*] Apnea (by reflex mechanism starting from the large pulmonary vessels), followed by polypnea (by excitation of sinocarotid receptors).

d. [] Hyporglycemia (by releasing catecholamines).

e. [*] Muscle fasciculations (by action on nicotinic receptors and at the level of the neuromuscular junction): eyelid contractions, tongue contractures, neck contractions.

25. What determines the central action in organophosphorus pesticide intoxications?

a. [*] Headache.

b. [*] Anxiety.

c. [*] Vertigo.

d. [] Drowsiness.

e. [*] Balance disorders.

26.What determines the central action in organophosphorus pesticide intoxications?

a. [] Inhibition.

b. [*] Psychological disorder.

c. [*] Knowledge disorder.

d. [*] Convulsions, coma.

e. [*] Paralysis of the vital importance centers in the spinal bulb.

27. Describe the clinical picture of acute intoxications with phosphororganic pesticides, the mild form.

a. [*] Patient suffering from: headache, dizziness, limb weakness.

b. [*] Dealing with the following accusations: decreased vision, anxiety, nausea, hypersalivation, abdominal colic, diarrhea.

c. [*] Sufferers are worried, pupils narrowed, reaction to dim light, accommodation spasm develops, which leads to decreased visual acuity.

d. [] Occurs without nighttime adjustment and in dimly lit rooms.

e. [*] Nystagmus, edema of the face, hypertranspiration occur.

28. Describe the clinical picture of acute intoxications with phosphororganic pesticides, the mild form.

a. [*] Long term consequences are breathing difficulties (predominantly in the inspiratory act), chest discomfort accompanied by respiratory failure, cough attacks.

b. [*] Rough breathing, dry rales are heard throughout the lung area.

c. [*] Cardiovascular system: tachycardia, increased BP.

d. [*] There is a marked decrease in erythrocyte cholinesterase activity in the blood serum.

e. [] Cardiovascular system: bradycardia, decreased BP.

29. Describe the clinical picture of acute intoxications with phosphororganic pesticides, the average form.

a. [*] Excitation, anxiety, inadequate reactions to external stimulants, intense headache, muscle weakness are typical.

b. [*] The breath disorder intensifies both in the inspiratory act and in the expiratory act, breathing becomes hissing, boiling, in the lungs wet rales are heard on the entire lung area.

c. [*] Signs of respiratory failure (cyanosis) appear.

d. [*] At this stage tachycardia can turn into bradycardia, BP remains high, hypertension accompanied by chills.

e. [] Signs of respiratory failure (cyanosis) disappear.

30. Describe the clinical picture of acute intoxications with phosphororganic pesticides, severe form.

a. [*] A condition reminiscent of pulmonary edema: boiling breathing, numerous wet rales throughout the lung area, foamy wet cough associated with hypersecretion of bronchial warts.

b. [*] The condition is aggravated by the intercostal muscle paralysis, breathing is based on the movement of the diaphragm and has a hiccup character.

c. [] Short-term tachycardia changes to bradycardia, BP remains low.

d. [*] On this background the collaptoid state may appear: disturbed consciousness, narrowed pupils, does not react to light.

e. [*] Generalized seizures occur periodically. A comatose state develops.

31. Describe the clinical picture of subacute intoxications with phosphororganic pesticides.

a. [*] According to symptoms of the subacute intoxications with phosphororganic compounds, they are practically identical to acute intoxications.

b. [*] Subacute intoxications are less widespread compared to acute ones, an important role is played by the individual's ability to adapt and react to the body, but also by the amount and toxicity of the toxicant that has entered the human body.

c. [*] As with acute intoxication, the toxicant enters the body in one go, but the onset is slower.

d. [] According to the symptomatology of subacute intoxications with phosphororganic compounds, they are practically identical to chronic intoxications.

e. [] As with chronic intoxications, the toxicant enters the body in one go, but the onset is slower.

32.Describe the clinical picture of chronic intoxications with phosphororganic pesticides, stage I.

a. [*] There is an intense headache, predominantly in the temporal region.

b. [*] Dizziness, decreased memory, sleep disorders.

c. [*] Anorexia, nausea, general weakness.

d. [] Sometimes causes mydriasis.

e. [*] Disorder of the vegeto-vascular innervation with predominance on the parasympathetic system.

33.Describe the clinical picture of chronic intoxications with phosphororganic pesticides, stage II.

a. [*] There is a gradual decrease in intellect.

b. [*] Short-term lipothemias may be present.

c. [] Sometimes causes mydriasis.

d. [] Neutrophilic leukocytosis, toxic leukocyte granulation is determined.

e. [] It is especially found in people who work with thiophos.

34.Describe the clinical picture of chronic intoxications with phosphororganic pesticides, stage III.

a. [*] It is especially found in people who work with thiophos.

b. [*] Leads to toxic liver damage ,

c. [*] in the blood count, we can notice neutrophilic leukocytosis, toxic leukocyte granulation.

d. [] There is a gradual decrease in intellect.

e. [] Short-term lipothemias may be present.

35. For what purpose are chlororganic compounds used in different branches of agriculture?

a. [*] As insecticides, acaricides, in the processing of seeds, crops.

b. [] As a nutrient support for crops.

c. [] Not used since 1970.

d. [] They were used only as chemical weapons.

e. [] No answer is correct.

36. List some representatives from the organochlorine compounds group.

a. [*] Chlorobenzene.

b. [*] Methoxychlor.

c. [*] Heptachlor.

d. [*] Chlordane.

e. [] Polyflordan.

37. What is the specificity of chlororganic compounds?

a. [*] Environment resistance.

b. [*] High solubility in fats and lipids.

c. [*] Ability to accumulate in body tissues (fat-rich tissues: adipose tissue, brain, liver, pancreas, spleen, adrenal glands, thyroid).

a. [] Lack of resistance in the environment.

d. [] Low solubility in fats and lipids.

38. What is the toxic action of chlororganic complexes characterized by?

a. [*] Modification of fermentation systems.

b. [*] Impairment of transmembrane transport of Na, K, Ca, Cl.

c. [*] Disorder of tissue respiration.

d. [] Does not affect fermentation systems.

e. [] Does not affect tissue respiration.

39. How does Curceatov describe chlororganic compounds?

a. [*] Fat-soluble non-electrolytes that are able to penetrate all the body's protective barriers.

b. [] Fat-soluble electrolytes, which are able to penetrate all the protective barriers of the body.

c. [] Some fat-soluble non-electrolytes, which are not able to penetrate all the protective barriers of the body.

d. [] Some fat-soluble electrolytes, which are not able to penetrate all the protective barriers of the body.

e. [] Water-soluble electrolytes, which are able to penetrate all the body's protective barriers.

40. On which systems and organs do chlororganic compounds act?

a. [*] Central Nervous System.

b. [*] Reproductive System,

c. [*] Parenchymal organs with necrotic and fatt degeneration.

d. [] The Vascular System.

e. [] Muscular System.

41. Describe the clinical picture of acute intoxications with chlororganic pesticides.

a. [*] When entering the respiratory tract, first of all, there are signs of excitation of the upper respiratory tract and damage of the lower respiratory tract (bronchi) in the form of (acute tracheobronchitis).

b. [*] In case of penetration through the gastrointestinal tract - dyspeptic phenomena occur (nausea, vomiting, abdominal pain, accelerated transit), developing acute gastroenterocolitis.

c. [*] In case of penetration of the toxicant through the skin, it is accompanied by acute inflammation, hemorrhages until tissue necrosis occurs.

d. [] In case of penetration through the gastrointestinal tract - signs of excitation of the upper respiratory tract and impairment of the lower respiratory tract (bronchi) in the form of (acute tracheobronchitis) appear.

e. [] In case of penetration of the toxicant through the skin, dyspeptic phenomena appears (nausea, vomiting, abdominal pain, accelerated transit), developing acute gastroenterocolitis.

42. Describe the clinical picture of acute intoxications with chlororganic pesticides, the mild form.

a. [*] Headache, vertigo.

b. [*] Behavioral disorders.

c. [*] Paresis, paresthesias of the extremities.

d. [*] Tremor of the eyelids and upper extremities.

e. [] Vestibular disorders.

43. Describe the clinical picture of acute intoxications with chlororganic pesticides, the average form.

a. [*] Seizures of the CNS may occur, sometimes epileptiform, colaptoid and comatose.

b. [*] Cardiovascular manifestations: cardiac pain, palpitations, dyspnea and chest pressure sensation.

c. [*] When large amounts of toxic enter the body, toxic-allergic myocarditis, toxic hepatitis and toxic nephritis may occur.

d. [*] Hematopoietic system: sometimes, with repeated penetration of the toxicant,

changes in the blood count may occur in the form of hypo- and aplastic anemia.

e. [] There are no CNS changes.

44. Describe the clinical picture of acute intoxications with chlororganic pesticides, the severe form.

a. [*] The severe form especially develops in intoxications with hexachloran or other analogous compounds.

b. [*] Signs of weakened peripheral nervous system may occur with the development of vegeto-sensory polyneuritis.

c. [*] The pathological process in such cases, diffusely affects the Nervous System with encephalopolyneuritis, which has a rather unfavorable evolution.

d. [] The severe form does not develop in hexachloran poisoning.

e. [] The pathological process in such cases does not diffusely affect the Nervous System.

45.Describe the clinical picture of subacute intoxications with chlororganic pesticides.

a. [*] According to symptoms are subacute intoxications with chlororganic compounds practically identical to acute intoxications.

b. [*] Subacute intoxications are less widespread compared to acute ones, an important role is played by the individual's ability to adapt and react to the body, but also by the amount and toxicity of the toxicant that has entered the human body.

c. [*] As with acute intoxication, the toxicant enters the body in one go, but the onset is slower.

d. [] As with chronic poisoning, the toxicant enters the body in one go, but the onset is slower.

e. [] Subacute intoxications with chlororganic compounds by symptomatology are practically identical to chronic intoxications.

46.Describe the clinical picture of chronic intoxications with chlororganic pesticides, stage I.

a. [*] It is typical to develop astheno-vegetative or astheno-organic syndrome (microorganic symptoms, which indicate the location of the pathological process in

the brainstem).

b. [*] There are also signs of astheno-vegetative syndrome with angio-dystonic cerebral paroxysms.

c. [*] Sudden headache occurs, accompanied by nausea, general weakness, hypertranspiration, dizziness, pallor, accessibility, bradycardia.

d. [] There are also signs of asthenic syndrome with anxious cerebral paroxysms.

e. [] Suddenly miosis occurs.

47. Describe the clinical picture of chronic intoxications with chlororganic pesticides, stage II.

a. [*] In the clinical picture at this stage of the pathological process the peripheral nervous system is involved with vegeto-sensory polyneuritis or mixed form.

b. [*] In severe forms, diffuse involvement of the SN (encephalopolyuritis) with organic symptoms is possible.

c. [*] Multiple sclerotic outbreaks.

d. [*] Static disorders with toxic process involvement of the extrapyramidal and hypothalamic regions, of the auditory nerves, of the vegetative cervical nodules.

e. [] Multiple necrosis outbreaks.

48.Describe the clinical picture of chronic intoxications with chlororganic pesticides, stage III.

a. [*] Nervous system disorder is accompanied by endocrine disorders (disorder of the adrenal cortex, pancreatic insulin system, thyroid hyperfunction).

b. [*] A special place is occupied by cardiovascular system disorders: vegetative-vascular dystonia, hypo- or hypertension, myocardial dystrophy, toxic-allergic myocarditis.

c. [] The peripheral nervous system is not involved in the clinical picture at this stage in the pathological process.

d. [] In severe forms, diffuse involvement of the SN (encephalopolyuritis) with organic symptoms is not possible.

e. [] Multiple foci of necrosis.

49. What are the characteristics of mercury-organic pesticides?

a. [*] Pesticides in this group are part of the chemicals with a high toxic effect.

b. [*] Possessing resistance and accumulation capacity.

c. [*] Due to evaporation, most of them (Granozan, Mercuzan, Mercurhexane) are dangerous for people who are in contact with them.

d. [] Pesticides in this group do not fall into the group of chemicals with a high toxic effect.

e. [] It does not possess resistance and the ability to accumulate.

50. Why do changes occur in the body with the predominance of Central Nervous System damage in intoxications with mercuric compounds?

a. [*] When the mercuroorganic complexes enter the body, they bind with the –SH (sulfhydric) groups of the cellular proteins, as a result of which the activity of the basic fermentative systems is disturbed, for the functioning of which the free SH (sulfhydric) groups are necessary.

b. [] When the mercuroorganic complexes enter the body, they do not bind with the –SH (sulfhydric) groups of cellular proteins, as a result of which the activity of the basic fermentative systems is disturbed, for the functioning of which free SH (sulfhydric) groups are necessary.

c. [] When the mercuroorganic complexes enter the body, they bind with the –SH (sulfhydric) groups of cellular proteins, as a result of which the activity of the basic fermentative systems is not disturbed, for the functioning of which free SH (sulfhydric) groups are necessary.

d. [] When the mercuroorganic complexes enter the body, they bind to the –SH (sulfhydric) groups of cellular proteins, as a result of which the activity of the basic fermentative systems is not disturbed, for the functioning of which the related SH (sulfhydric) groups are necessary.

e. [] When the mercuroorganic complexes enter the body, they do not bind to the –SH (sulfhydric) groups of cellular proteins, as a result of which the activity of the basic fermentative systems is not disturbed, for the functioning of which the related SH (sulfhydric) groups are necessary. .

51. Are capillary toxicity and allergic reactions specific to intoxications with mercuric compounds?

a. [*] Yes.

b. [] Maybe.

c. [] Yes, but very rarely.

d. [] Not all the time.

e. [] No answer is correct.

52.Describe the clinical picture of acute intoxications with mercuric pesticides, the mild form.

a. [*] Metallic taste.

b. [*] Gingivitis.

c. [*] Gastroenterocolitis.

d. [*] Astheno-vegetative syndrome.

e. [] Cerebral edema.

53. Describe the clinical picture of acute intoxications with mercuric pesticides, the average form.

a. [*] Neurological disorders predominate: acute toxic encephalitis with damage to the cerebellum, the truncated part of the brain, characterized by nystagmus, intentional tremor, positive Romberg picture.

b. [*] Myalgia, arthralgia, retrobulbar and trochlear neuritis, neuro-psychic disorders often develop.

c. [*] At this stage the pathological process is reversible, and in some cases has a recurrent character, with the worsening of the general condition as a result of alcohol use, following infections, as well as other factors that influence the body.

d. [] Myalgia, arthralgia, retrobulbar and trochlear neuritis, neuro-psychic disorders often do not develop.

e. [] At this stage the pathological process is irreversible.

54. Describe the clinical picture of acute intoxications with mercuric pesticides, the severe form.

a. [*] There are diffuse changes in the CNS, toxic encephalopolyneuritis develops.

b. [*] With predominance in the clinical picture of diencephalon damage: polyuria, adinamia, anorexia, progressive weight loss.

c. [*] The pathological process also includes the cranial nerves: ocolomotor, facial trigeminal, vagus, vestibulo-cochlear.

d. [*] Paresis of the limbs is possible due to peripheral nerves, pyramidal pathways and cerebellum damage.

e. [] There are no diffuse changes in the CNS.

55. Describe the clinical picture of acute intoxications with mercuric pesticides, severe form.

a. [*] Intentional tremor, dysarthria, ataxia appear when the cerebellum is affected.

b. [*] In some patients there may be positive meningeal signs, limb hyperkinesia, epileptiform seizures.

c. [*] Patients often suffer from influenced mental state: auditory and visual hallucinations, delusions, schizophreniform syndrome.

d. [] There are no diffuse changes in the CNS.

e. [] There are no psychic changes.

56.Describe the clinical picture of acute intoxications with mercuric pesticides, the severe form.

a. [*] It may also affect the cardiovascular system for which capillarotoxicosis, toxic myocardium or toxic-allergic myocardium is typical.

b. [*] It is possible that the phenomena of toxic damage to the liver, kidneys occurs.

c. [*] In the blood count: leukocytosis, accelerated ESR.

d. [] There are no diffuse changes in the CNS.

e. [] There are no psychic changes.

57.Describe the clinical picture of subacute intoxications with mercuric pesticides.

a. [*] Subacute intoxications with mercuric compounds according to the symptoms are practically identical to acute intoxications.

b. [*] Subacute intoxications are less widespread compared to acute ones, a decisive role is played by the individual's ability to adapt and react to the body, but also by the amount and toxicity of the toxicant that has entered the human body.

c. [*] As with acute intoxication, the toxicant enters the body in one go, but the onset is slower.

d. [] As with chronic poisoning, the toxicant enters the body in one go, but the onset is slower.

e. [] Subacute intoxications with mercuric compounds according to symptoms are practically identical to chronic intoxications.

58.Describe the clinical picture of chronic intoxications with mercuric pesticides, stage I.

a. [*] Astheno-vegetative syndrome and polyneuropathy will normally appear.

b. [*] With long-term action of these pesticides, the signs of neuro-vegetative dystonia increase.

c. [*] The trembling of the hands intensifies.

d. [] At the long-term action of these pesticides the signs of neuro-vegetative dystonia decrease.

e. [] The trembling of the hands does not intensify.

59. Describe the clinical picture of chronic intoxications with mercuric pesticides, stage II.

a. [*] cachexia.

b. [*] vegeto-vascular crises.

c. [] At the long action of these pesticides the signs of neuro-vegetative dystonia decrease.

d. [] Hand tremor does not intensify.

e. [] The appearance of astheno-vegetative syndrome and polyneuropathy is not characteristic.

60. Describe the clinical picture of chronic intoxications with mercuric pesticides, stage III.

a. [*] First of all are signs of organic outbreak or diffuse CNS: toxic encephalopathy, rarely encephalopolyneuritis.

b. [*] When the spinal cord is included in the pathological process, the conduction pathways suffer, which leads to the appearance of spastic paraparesis, without sensitivity disorder.

c. [*] Disorders of the peripheral nervous system occur after those of the CNS.

d. [*] Changes in sensitivity according to the polyneuritic type are accompanied by decreased carpo-radial and Achilles reflexes.

e. [] CNS disorders occur after those of the peripheral nervous system.

61.Describe the clinical picture of chronic intoxications with mercuric pesticides, stage III.

a. [*] The onset of polyneuritis is slow or progressive.

b. [*] The pathological process at this stage acquires a resistant character.

c. [*] In CSF (cerebrospinal fluid) the amount of protein and chlorides is low and glucose is increased.

d. [*] In addition to the pathology of the CNS, myocardial dystrophy is also associated, sometimes affecting the liver, which is distinguished by a progressive onset with toxic jaundice.

e. [] In CSF (cerebrospinal fluid) the amount of protein and chlorides is increased and the glucose level is low.

62.Describe the clinical picture of chronic intoxications with mercuric pesticides, stage III.

a. [*] There is often a hypochromic anemia, at first moderate leukocytosis, then leukopenia occurs, in the protoplasm of leukocytes toxic granulation is determined.

b. [*] Platelet count decreases, blood clotting is upset, ESR is accelerated.

c. [] CNS disorders occur after those of the peripheral nervous system.

d. [] In CSF (cerebrospinal fluid) the amount of protein and chlorides is increased, and the level of glucose is low.

e. [] Platelet count increases, ESR accelerates.

63.Describe the clinical picture of chronic intoxications with mercuric pesticides, stage III.

a. [*] The local action of mercuroorganic substances is poorly expressed, but at prolonged contact with them the appearance of mucosal irritationis is possible, the development of blepharoconjunctivitis and allergic dermatitis.

b. [*] The basic sign in intoxications with mercuroorganic substances is the presence of mercury in biological environments: blood, urine, and in severe intoxications and in CSF.

c. [] CNS disorders occur after those of the peripheral nervous system.

d. [] In CSF (cerebrospinal fluid) the amount of protein and chlorides is increased and the glucose level is low.

e. [] Platelet count increases, ESR accelerates.

64.What is the purpose of stopping contact with the toxicant in case of acute exposure?

a. [*] In acute exposure of the human organism to the toxicant, the first step is to rapidly stop the contact with the toxicant itself, aiming to restore the normal function of the contaminated organism.

b. [] In chronic exposure of the human organism to the toxicant, the first step is to slowly stop the contact with the toxicant itself, aiming to restore the normal function of the contaminated organism.

c. [] In acute exposure of the human body to the toxicant, the first step is to slowly stop contact with the toxicant itself, aiming to gradually restore the normal function of the contaminated organism.

d. [] In the acute exposure of the human body to the toxicant, the first step is to maintain contact with the toxicant itself.

e. [] In the subacute exposure of the human organism to the toxicant, the first step is to slowly stop contact with the toxicant itself, aiming to gradually restore the normal function of the contaminated organism.

65. What should be done if the toxicant has penetrated the inhaled air?

a. [*] When the toxicant enters with the inspired air, it is necessary to transfer the patient from the polluted room, remove the clothes and destroy them, which would

improve the respiratory act.

b. [] When the toxicant enters with the inhaled air, it is not necessary to transfer the patient from the polluted room.

c. [] When the toxicant enters with the inhaled air, it is necessary to transfer the patient from the polluted room, remove the clothes and destroy them, but this will not improve the respiratory act.

d. [] When the toxicant enters with the inspired air, it is necessary to transfer the patient from the polluted room, without removing the clothes and destroying them.

e. [] When the toxicant enters with the inhaled air, it is not necessary to transfer the patient from the polluted room, do not remove the clothes and do not destroy them.

66. What to do if the toxicant has penetrated the skin?

a. [*] Upon penetration through the skin, the toxicant is removed with a cotton swab.

b. [*] Wash skin with warm soapy water or 2% sodium bicarbonate solution.

c. [*] Wipe the skin with 5-10% or 2.5% chloramine solution.

d. [] When penetrating the skin, the toxicant is not removed with a cotton swab.

e. [] Wash your skin with warm water and soap or 20% sodium bicarbonate solution.

67. What should be done if the toxin has entered the digestive tract?

a. [*] Toxicity is removed from the stomach by gastric lavage with warm water and absorbents (activated charcoal).

b. [*] For the extraction of the toxic from the intestine, purgative siphon enemas (MgSO4) are applied.

c. [] Toxic removal from the stomach is performed by gastric lavage with warm water and absorbents (spirit solution).

d. [*] For the extraction of the toxin from the intestine, purgative siphon enemas (2.5% chloramine solution) are applied.

e. [*] For the extraction of the toxin from the intestine, purgative siphon enemas (20% sodium bicarbonate) are applied.

68. Name the principles underlying treatment for pesticide poisoning.

a. [*] Administration of antidotes.

b. [*] Pathogenetic treatment.

c. [] Monomedicine treatment.

d. [*] Symptomatic treatment.

e. [] No administration of antidotes.

69. What properties should antidotes have in the treatment of pesticide poisoning?

a. [*] To inactivate the toxicant in the blood.

b. [*] To remove the toxic effect of its metabolites.

c. [*] To hasten its excretion from the body.

d. [] Do not inactivate the toxicant in the blood.

e. [] not to hasten its excretion from the body.

70. What properties does the antidote Unitiol and Succimeter use in the treatment of pesticide poisoning?

a. [*] The detoxifying action of these antidotes is the ability to intervene in chemical reactions with the toxicant or its metabolites as a result of which the toxicant is inactivated.

b. [*] Eliminating the toxic from the body through urine and feces.

c. [] Eliminating the toxicant from the body through the exhaled air.

d. [] Eliminating the toxicant from the body only through urine.

e. [] The detoxifying action of these antidotes is based on the ability to intervene in chemical reactions with the toxicant or its metabolites as a result of which the toxicant dissolves.

71.What physiological antidotes do you know and how are they used in the treatment of pesticide poisoning?

a. [*] Physiological antidotes are used in order to remove the toxic effect of antagonism on the same systems of the body, altering the metabolism of toxic complexes.

b. [*] These antidotes include: Methylene blue, Cholinolytics and Cholinesterase Reactivators, Antioxidants.

c. [*] Antidotes can be administered in different combinations of several preparations, mutually increasing the effect of the treatment.

d. [] Physiological antidotes are not used to remove the toxic effect.

e. [] Antidotes cannot be administered in the form of combinations of several preparations, they mutually decrease the effect of the treatment.

72.What is the purpose of Unitiol in the treatment of pesticide poisoning?

a. [*] Unitiol is used as an antidote in intoxications with chlororganic and mercuric compounds.

b. [] Unitiol is used as an antidote in intoxications with phosphororganic compounds.

c. [] Unitiol is used as an antidote in intoxications with flororganic compounds.

d. [] Unitiol is used as an antidote in intoxications with arsenic compounds.

e. [] Unitiol is not used as an antidote in intoxications with chlororganic and mercuric compounds.

73.How is Unitiol used in the treatment of acute pesticide poisoning?

a. [] In case of acute intoxication, perform the antidote therapy with unitiol - proportion : 1 ml of 10% solution to 5 kg of the victim's weight.

b. [*] In case of acute intoxication, perform the antidote therapy with unitiol - proportion : 1 ml of 5% solution per 10 kg of the victim's weight.

c. [*] On the first day - 3-4 times a day, intramuscularly (i / m).

d. [*] The next day - 2-3 times a day, i / m.

e. [*] From the 3rd to the 7th day - 1-2 times a day, i / m.

74.How is Unitiol used in the treatment of chronic pesticide poisoning?

a. [*] In case of chronic intoxication, perform the antidote therapy with unitiol - 1 ml of 5% solution once a day for 7-10 days.

b. [] In case of chronic intoxication, perform the antidote therapy with unitiol - 1 ml of 15% solution once a day for 7-10 days.

c. [] In case of chronic intoxication, perform the antidote therapy with unitiol - 1 ml of 2.5% solution once a day for 7-10 days.

d. [] In case of chronic intoxication, perform the antidote therapy with unitiol - 1 ml of 30% solution once a day for 7-10 days.

e. [] In case of chronic intoxication,perform the antidote therapy with unitiol - 1 ml of 20% solution once a day for 7-10 days.

75.What antioxidants are used in the treatment of pesticide poisoning?

a. [*] Vitamins and aminoacids serve as antioxidants. They prevent the oxidation of the toxin and the formation of toxic derivatives, its transformation.

b. [] Mannitol and unitiol serve as antioxidants. They prevent the oxidation of the toxin and the formation of toxic derivatives, its transformation.

c. [] Only antioxidants serve as antioxidants. They prevent the oxidation of the toxin and the formation of toxic derivatives, its transformation.

d. [] Only antioxidants serve as antioxidants. They prevent the oxidation of the toxin and the formation of toxic derivatives, its transformation.

e. [] As antioxidants. They prevent the oxidation of the toxin and the formation of toxic derivatives, its transformation, except for vitamins and amino acids.

76. For what purpose is Atropine used in the treatment of pesticide poisoning?

a. [*] Atropine is the antidote used in intoxications with phosphororganic compounds.

b. [] Atropine is the antidote used in intoxications with mercuric compounds.

c. [] Atropine is the antidote used in intoxications with chlororganic compounds.

d. [] Atropine is the antidote used in intoxications with fluororganic compounds.

e. [] Atropine is the antidote used in intoxications with all compounds except phosphororganics.

77. What doses of Atropine are used to treat phosphororganic pesticide poisoning?

a. [*] Atropine is the basis of treatment, reaching the administration of huge doses (over 100 mg per day, meaning 100 ampoules).

b. [] Atropine is the basis of treatment, reaching the administration of huge doses

(over 1000 mg per day, meaning 100 ampoules).

c. [] Atropine is the basis of treatment, reaching the administration of huge doses (over 10 mg per day, meaning 10 ampoules).

d. [] Atropine is the basis of treatment, reaching the administration of huge doses (over 100 mg per day, meaning 1000 ampoules).

e. [] Atropine is the basis of treatment, reaching the administration of huge doses (over 1 mg per day, meaning 100 ampoules).

78.How is Atropine used in the treatment of phosphororganic pesticide poisoning?

a. [*] In acute intoxications, the mild form, 1-2 ml of 0,1% atropine sulphate is administered subcutaneously or intramuscularly.

b. [*] In acute intoxications the moderate or severe form is administered 3-5 ml - 0.1% intramuscular or intravenous atropine sulfate.

c. [] In acute intoxications, the mild form, 12 ml of 0,1% atropine sulphate is administered subcutaneously or intramuscularly.

d. [] In acute intoxications, the moderate or severe form is administered 35 ml - 0.1% intramuscular or intravenous atropine sulfate.

e. [] In acute intoxications, the moderate or severe form is administered 0.05 ml - 1% atropine sulphate intramuscularly or intravenously.

79. How long will we take Atropine in the treatment of phosphororganic pesticide poisoning?

a. [*] Until the poisoning symptoms disappear and the atropine overdose signs appear (dry mouth, blurred vision).

b. [] Until symptoms of poisoning occur.

c. [] Until the symptoms of poisoning disappear.

d. [] Until the atropine overdose symptoms disappear (dry mouth, blurred vision).

e. [] Until symptoms of poisoning and signs of overdose with atropine (dry mouth, blurred vision) occur.

80. What is Pralidoxine, what group does this medicine belong to and what contribution does it make to the treatment of pesticide poisoning?

a. [*] It is the substance capable of restoring cholinesterase.

b. [*] It belongs to the oxime group.

c. [*] Pralidoxime (2-PAM) regenerates cholinesterase by reversing phosphorylation.

d. [] It is the substance capable of restoring SH groups.

e. [] It belongs to the group of antioxidants.

81. What is the loading dose of Pralidoxine in the treatment of pesticide poisoning?

a. [*] The loading dose for Pralidoxine is 30 mg / kg, then 8 mg / kg / h continuously.

b. [] The loading dose for Pralidoxine is 30 mg / kg, then 80 mg / kg / h continuously.

c. [] The loading dose for Pralidoxine is 3 mg / kg, then 8 mg / kg / h continuously.

d. [] The loading dose for Pralidoxine is 30 mg / kg, then 3 mg / kg / h continuously.

e. [] The loading dose for Pralidoxine is 80 mg / kg, then 8 mg / kg / h continuously.

82.What is the purpose of Pralidoxine in the treatment of pesticide poisoning?

a. [*] The administration of oxime is reserved for patients with severe intoxications manifesting acute respiratory failure, convulsions, coma.

b. [] The administration of oxime is not reserved for patients with severe intoxications manifesting acute respiratory failure, convulsions, coma.

c. [] The administration of oxime is prohibited.

d. [] The administration of oxime is obligatory to all patients.

e. [] The administration of oxime is reserved for patients without signs of severe intoxication manifesting acute respiratory failure, convulsions, coma.

83.Which medicinal preparations are associated with the use of Pralidoxine in the treatment of pesticide poisoning?

a. [*] Pralidoxime is used in combination with atropine because atropine cannot regenerate cholinesterase.

b. [] Pralidoxime is used in combination with unitiol, as unitiol cannot regenerate cholinesterase.

c. [] Pralidoxime is used in combination with atropine because pralidoxine cannot regenerate cholinesterase.

d. [] Pralidoxime should not be used in combination with atropine, as atropine may regenerate cholinesterase.

e. [] Pralidoxime is used in combination with atropine, as atropine may regenerate cholinesterase.

84.What will be taken into account in the work capacity expertise in pesticide poisonings?

a. [*] Working conditions will be taken into account.

b. [*] General anamnesis and professional path.

c. [*] The character of the clinical manifestations of intoxication.

d. [*] The development of the pathological process.

e. [] Working conditions are considered less important.

85.What are the mild and medium severity cases in the work capacity expertise, in pesticide poisonings?

a. [*] In mild and moderate cases of intoxication the pathological process is usually reversible, the intoxication ends with full recovery, the work capacity is limited for a period of time.

b. [*] When high sensitivity to pesticides (Hg, Cl compounds) occurs, resuming work with the same compounds is not recommended.

c. [] In mild and medium cases of intoxication the pathological process is usually irreversible, the intoxication ends with incomplete recovery, the work capacity is limited for a long period of time.

d. [] When high sensitivity to pesticides (Hg, Cl compounds) occurs, resuming work with the same compounds is recommended.

e. [] In mild and moderate cases of intoxication, the pathological process is usually reversible, the intoxication ends in full recovery, but the work capacity is lost.

86.What are the medium gravity cases in the work capacity expertise, in the intoxications with pesticides, what do they include?

a. [*] Prolonged evolution or recurrences of toxic encephalopathy, peripheral nervous

system damage, severe psycho-vegetative syndromes, these patients should not resume working with pesticides, being contraindicated as well as other work related to the influence of other harmful factors on the body.

b. [*] Such patients require a rational work regime, in case of decreased work capacity they go to the medical commission to establish the work expertise.

c. [*] The work capacity level loss or IIIrd degree disability occupational disease related is determined.

d. [] When high sensitivity to pesticides (Hg, Cl compounds) occurs, resuming work with the same compounds is recommended.

e. [] In cases of moderate intoxication, the pathological process is usually reversible, intoxication ends in full recovery, but the ability to work is lost.

87. What are the serious cases in the work capacity expertise, in the intoxications with pesticides, what do they include?

a. [*] Severe forms of intoxication with diffuse CNS damage, involving the SCV process, liver, blood usually lead to stable loss of work capacity.

b. [*] These patients are diagnosed with group II or III occupational diseases related disability .

c. [*] The disability group is established individually, taking into account the recurrences of the pacients intoxications.

d. [] When high sensitivity to pesticides (Hg, Cl compounds) occurs, resuming work with the same compounds is recommended.

e. [] In severe cases of intoxication, the pathological process is usually reversible, the intoxication ends in full recovery, but the ability to work is lost.

88. What is the importance of respecting the health rules in preventing pesticide poisoning?

a. [*] To a large extent, the lack of adequate sanitary behavior when working with pesticides leads to the development of intoxications at work, in the work process, in the storage and transportation of pesticides or in their use in agriculture.

b. [] To a large extent, the lack of adequate sanitary behavior when working with pesticides does not lead to the development of intoxications at work, in the work process, in the storage and transportation of pesticides or in their use in agriculture.

c. [] Largely the lack of adequate sanitary behavior, when working with pesticides, leads to protection against the development of intoxications at work, in the work process, in the storage and transportation of pesticides or in their use in agriculture.

d. [] No answer is correct.

e. [] Observance of sanitary rules is a problem of the worker.

89. Is declaring all occupational risk factors a prophylactic measure for pesticide poisoning?

a. [*] It is important to assess all occupational risk factors in the workplace, detailed job passporting is a key element in the management of occupational pesticide poisoning prevention.

b. [] It is not important to assess all occupational risk factors in the workplace, detailed job passporting is not a key element in the management of occupational pesticide poisoning prevention.

c. [] It is important to assess all occupational risk factors in the workplace, but this does not help us to prevent poisoning.

d. [] It is important to assess all occupational risk factors in the workplace, but this is not done without the employee's consent.

e. [] It is important to assess all occupational risk factors in the workplace, but this is not done without the consent of the doctor.

90. What is the importance of medical controls in the prophylaxis of pesticide poisoning?

a. [*] One of the basic organizational measures for preventing pesticide poisoning is performing a medical control on the people who have entered work or those who work (or are in contact) with pesticides, early diagnosis of intoxication, treatments for increasing the resistance of the body.

b. [] Medical control when working with pesticides is not mandatory.

c. [] Medical control in working with pesticides is no more important than compliance with the sanitary regime.

d. [] Medical control in working with pesticides is no more important than monitoring risk factors for employers.

e. [] Medical control when working with pesticides is not helpful.