HAZARDOUS AND TOXIC SUBSTANCES POISONING IN WARTIME AND PEACETIME. CLASSIFICATION OF HAZARDOUS CHEMICALS. MECHANISM OF TOXIC ACTION. CLINICAL MANIFESTATIONS OF POISONING WITH CHEMICAL AGENTS

Methodical instructions
for the 5th year students to the practical class

МІНІСТЕРСТВО ОХОРОНИ ЗДОРОВ'Я УКРАЇНИ
Харківський національний медичний університет

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ОТРУЄННЯ ШКІДЛИВИМИ ТА СИЛЬНОДІЮЧИМИ ХІМІЧНИМИ РЕЧОВИНАМИ У ВОЄННИЙ ТА МИРНИЙ ЧАС. КЛАСИФІКАЦІЯ ОТРУЙНИХ РЕЧОВИН. МЕХАНІЗМ ТОКСИЧНОЇ ДІЇ. КЛІНІЧНІ ПРОЯВИ УРАЖЕНЬ ОТРУЙНИМИ РЕЧОВИНАМИ

Методичні вказівки
для здобувачів вищої освіти V року навчання
до проведення практичного заняття

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Отруєння шкідливими та сильнодіючими хімічними речовинами у воєнний та мирний час. Класифікація отруйних речовин. Механізм токсичної дії. Клінічні прояви уражень отруйними речовинами : метод. вказ. для здобувачів вищої освіти V року навчання / упоряд. А.Я. Меленевич, Д.В. Мартовицький, Д.Г. Молотягін. Харків : ХНМУ, 2022. 16 с.

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Topic 2: «Hazardous and toxic substances poisoning in wartime and peacetime. Classification of hazardous chemicals. Mechanism of toxic action. Clinical manifestations of poisoning with chemical agents»

1. **Hours**: 4.

2. **Importance of the topic**: Exposure to toxic chemicals represents an important public health problem worldwide. According to a WHO estimate (WHO, 2 000), unintentional poisonings led to 300 000 deaths in the year 2 000. Over 70 000 deaths occurred in children up to 14 years old. Rural and industrial workers use sometimes with no protection large volumes of chemicals, some of which are extremely hazardous. Pregnant women may expose themselves and the developing fetus to the effects of chemicals in their environments at crucial development periods of their life. Elderly people are, due to physiological change, more susceptible to the toxic effects of some chemicals. Lack of information and education often results in people ignoring or neglecting simple measures that could help to prevent the toxic effects of chemicals. As a result, a large number of poisoning cases result from human carelessness, negligence and ignorance, all of which could be avoidable.

3. **Aim of studying**: The aim of this topic is to be able to recognize poisoning by dangerous and toxic chemicals in wartime and peacetime and to know the principles of emergency therapy for these poisoning.

Specific objectives to be achieved after conducting practical classes:

Students need to know:	Students will be able to:
1. Definition of chemical agents. 2. Classification of chemical agents. 3. Nerve agents: signs of poisoning, decontamination, antidotes. 4. Vesicants (blister agents): signs of poisoning, decontamination, management. 5. Choking agents: signs of poisoning, decontamination, management. 6. Blood agents (cyanide compounds): signs of poisoning, decontamination, general management, antidotes. 7. Incapacitating (behavior-altering) agents: signs of poisoning, decontamination, antidotes. 8. Prevention	1. List and describe five groups of chemical agents. 2. Identify and differentiate the signs of various chemical poisonings. 3. Provide emergency aid for nerve agents poisoning. 4. Provide emergency aid for blister agents poisoning. 5. Provide emergency aid for choking agents poisoning. 6. Provide emergency aid for blood agents poisoning. 7. Provide emergency aid for behavior-altering agents poisoning. 8. Prescribe patogenetically proved treatment. 9. Provide prevention

4. **Indicative syllabus**

- Chemical agents: definition, classification
- Nerve agents: signs of poisoning, decontamination, management
- Blister agents: signs of poisoning, decontamination, management

- Choking agents: signs of poisoning, decontamination, management
- Blood agents: : signs of poisoning, decontamination, management
- Behavior-altering agents: signs of poisoning, decontamination, management
- Protection, prevention and prognosis
- Chemical triage

5. Material and methodological support: Visual material, multimedia devices, Microsoft Power Point presentations, tables, posters. Training manuals. Link to the discipline page in MOODLE:
http://distance.knmu.edu.ua/course/view.php?id=2804.

6. Materials for practical classes:

Chemical agents is the general name of substances known to have toxic effects on the environment, which cause a large number of deaths and disabilities in a short period of time.

Hazardous chemicals are substances or mixtures that can pose a significant risk to health and safety if not managed correctly.

A chemical weapon agent is a chemical substance whose toxic properties are used to kill, injure or incapacitate human beings.

Chemical warfare is warfare using the toxic properties of chemical substances to kill, injure or incapacitate an enemy.

Among the existing methods of spreading chemical weapons the main threat is associated with their use as aerosols or vapors. In this regard, respiratory protection devices and means of predicting the potential airborne spread of the relevant agent can allow timely protective measures to be taken in those areas that may be affected by an attack. The problem of skin damage caused mainly by chemical agents and occurring in the immediate vicinity of the place of their distribution is relevant. In this case, protective clothing is the most important means of prevention.

Damage to personnel by chemical substances or weapons can occur both in combat and in peacetime conditions as a result of:

- using of chemical weapons during military operations or terrorist acts;
- accidents at processing plants and warehouses of combat toxic substances;
- destruction of chemical enterprises and storage facilities for hazardous chemicals;
- accidents during transportation of chemical agents;
- destruction of hazardous chemicals burial sites.

Depending on the duration of the interaction between the chemical substance and the organism, intoxications can be acute, subacute and chronic.

Intoxication that develops as a result of single or repeated exposure to a toxicant within a limited period of time (usually up to several days) is called acute.

Subacute intoxication is called intoxication that develops as a result of continuous or interrupted (intermittent) exposure to a toxicant lasting up to 90 days.

Intoxication that develops as a result of long-term (sometimes years) exposure to a toxicant is called chronic. As a rule, during any intoxication, four main periods can be distinguished: contact with the substance, the hidden period, the disease`s onset, and recovery. Sometimes there is a period of complications. The expression and duration of each of the periods depend on the properties of the substance that caused the intoxication, its dose and conditions of interaction with the body, etc.

Chemical agents are divided into 5 categories:

1. Nerve agents.
2. Vesicants (blister agents).
3. Choking (pulmonary-damaging) agents.
4. Asphyxiants/blood agents (cyanide compounds).
5. Incapacitating/behavior-altering agents.

Nerve agents are phosphorus compounds which inhibit the acetylcholinesterase enzyme (AChE). These agents are the most dangerous and lethal substances we have ever known. These agents are divided into subclasses known as G and V series agents. G series agents include; Sarin, Soman, Tabun. This is also classified because the initials of the short names of these titles begin with G.

Anticholinesterase and non-anticholinesterase theories of the toxic effect of nerve agents are distinguished in the pathogenesis. The basis of the pathogenesis of the nerve agent`s action on the body is inhibition of the AChE, which cleaves acetylcholine on the postsynaptic membrane, as a result of which an excess of acetylcholine accumulates in the synaptic cleft, resulting in overstimulation of the cholinergic structures of the nervous system.

Signs and symptoms of nerve agent poisoning

Receptor	Target	Signs and Symptoms
Muscarinic	Glands	
	Conjunctival mucosa	Hyperaemia
	Nasal mucosa	Rhinorrhoea, hyperaemia
	Bronchial mucosa	Bronchorrhea, bronchoconstriction, dyspnoea
	Sweat	Sweating
	Lacrimal	Lacrimation
	Salivary	Salivation
	Smooth muscle	
	Iris	Miosis, dim vision
	Ciliary muscle	Failure of accommodation, blurring of vision, frontal headache
	Gut	Nausea, vomiting, abdominal cramp, diarrhoea
	Bladder	Frequent, involuntary micturition
	Heart	Bradycardia, rhythm abnormalities
Nicotinic	Autonomic ganglia	Pallour, tachycardia, hypertension
	Skeletal muscle	Muscular twitching, fasciculation, weakness, paralysis
Central	Central nervous system	Giddiness, anxiety, restlessness, headache, tremor, confusion, failure to concentrate, excessive dreaming, convulsions, unconsciousness, respiratory depression

There are 3 degrees of severity of nerve agent poisoning: mild, moderate, and severe.

The degree of severity of nerve agent poisoning depends on the severity of cholinesterase inhibition. Mild poisoning occurs when the activity of

cholinesterase decreases to 50 % of the initial level, moderate – 50–80 %, and severe – more than 80 %. This indicator is determined by laboratory methods and is a prognostic criterion for the further course of poisoning.

The diagnosis of poisoning by nerve agents is based mainly on the clinical signs of intoxication. The most reliable signs are:

- inhalation exposure: miosis, spasm of accommodation, bronchospasm, salivation, sweating, fibrillation of certain muscle groups and convulsions;
- skin exposure: muscle twitching at the site of poison application, convulsions and paralysis;
- oral exposure: repeated vomiting, pain in epigastric region, widespread abdominal pain, frequent feaces (traces of the nerve agent can be detected), moist skin, muscle fibrillation, convulsions, miosis.

With mild clinical symptoms, laboratory blood tests for AChE activity are used.

Acute nerve agent poisoning is treated by **decontamination, respiratory support, and three antidotes: an anticholinergic** *(atropine is competitive inhibitor of acetylholine)*, **an oxime** *(pralidoxime – it reactivates acetylcholinesterase)* **and an anticonvulsant** *(diazepam).* In acute cases, all these forms of therapy may be given simultaneously.

Decontamination should be performed as soon as possible. For immediate spot decontamination, RSDL (reactive skin decontamination lotion) or other decontaminants should be used. In any case, before admission to a hospital, clothes should be removed and discarded, and exposed skin should be decontaminated to avoid cross-contamination of medical personal. If decontamination solutions or lotions are not available, copious amounts of water and soap should be used. Eyes should be rinsed with physiological saline or, if not available, with tap water.

Antidote recommendations after exposure to nerve agents

Patient's Age	Antidotes		Other Treatment
	Mild/Moderate Effects[1]	Severe Effects[2]	
Infants (0-2 years)	Atropine (0.05 mg/kg IM or 0.02 mg/kg IV) *and* 2-PAM Cl (15 mg/kg IM or *IV* slowly)	Atropine (0.1 mg/kg IM or 0.02 mg/kg IV) *and* 2-PAM chloride (25 mg/kg IM or 15 mg/kg *IV* slowly)	Assisted ventilation after antidotes for severe exposure
Child (2-10 years)	Atropine (1 mg IM or 0.02 mg/kg IV) *and* 2-PAM Cl[3] (15 mg/kg IM or *IV* slowly)	Atropine (2 mg IM or 0.02 mg/kg IV) *and* 2-PAM chloride[3] (25 mg/kg IM or 15 mg/kg *IV* slowly)	Repeat atropine (2 mg IM, or 1 mg IM for infants) at 5- to 10-min intervals until secretions have diminished and breathing is comfortable or airway resistance has returned to nearly normal.
Adoles-cent (>10 years)	Atropine (2 mg IM or .02 mg/kg IV) *and* 2-PAM Cl[3] (15 mg/kg IM or \IV slowly)	Atropine (4 mg IM or 0.02 mg/kg IV) *and* 2-PAM Cl[3] (25 mg/kg IM or 15 mg/kg IV slowly)	
Adult	Atropine (2-4 mg IM or IV*) and* 2-PAM Cl (600 mg IM or 15 mg/kg IV slowly)	Atropine (6 mg IM) *and* 2-PAM Cl (1800 mg IM or 15 mg/kg IV slowly	Phentolamine for 2-PAM-induced hypertension (5 mg IV for adults; 1 mg IV for children) Diazepam for convulsions (0.2-0.5 mg IV for infants < 5 years; 1 mg IV for children > 5 years; 5 mg IV for adults).

Patient's Age	Antidotes		Other Treatment
	Mild/Moderate Effects[1]	Severe Effects[2]	
Elderly, frail	Atropine (1 mg IM) *and* 2-PAM Cl (10 mg/kg IM or 5-10 mg/kg IV slowly)	Atropine (2-4 mg IM) and 2-PAM Cl (25 mg/kg IM or 5-10 mg/kg *IV* slowly)	

[1]*Mild/moderate effects include localized sweating, muscle fasciculations, nausea, vomiting, weakness, and dyspnea.*

[2]*Severe effects include unconsciousness, convulsions, apnea, and flaccid paralysis.*

[3]*If the calculated dose exceeds the adult IM dose, adjust accordingly.*

Abbreviation: 2-PAM Cl, 2-pralidoxime (or Protopam®) chloride

Vesicants (blister agents)

Blister agents include mustard agents and lewisite.

Vesicants burn and blister the skin or any other part of the body surface they contact. They act on the eyes, mucous membranes, lungs, skin, and bloodforming organs (bone marrow and spleen). They damage the respiratory tract when inhaled and cause vomiting and diarrhoea when ingested. Blister agents may also cause bone marrow suppression and have effects on other germ cells.

When the skin is affected by mustard agents, the latent period lasts 2–4 hours, followed by the appearance of erythema. With mild lesions, the course of erythematous dermatitis lasts up to 10 days with a typical clinical picture. With more severe lesions, erythematous bullous dermatitis occurs. In case of deep damage to the skin, ulcers are formed with very slow epithelization (2–3 months). Infection is possible. Severe bullous keratoconjunctivitis, damage to the cornea occurs when the eyes are damaged by drops of mustard agent. Reparative processes are very slow (4–6 months).

When the digestive organs are affected by mustard agents, the first signs of poisoning are pain in the epigastrium, nausea, vomiting, diarrhea, significant weakness, headache, tachycardia, hypotension. Swelling of the lips and mucous membrane of the oral cavity, pain during palpation in the epigastrium, flatulence are observed. With a significant lesion, gastric bleeding, ulceration, and later keloid stenoses are possible.

When affected by lewisite, the latent period is significantly reduced due to a stronger irritating effect, a pronounced pain syndrome, inflammation with massive exudation, but also a faster recovery.

When acting on the respiratory organs, reflectory respiratory arrest and toxic pulmonary edema are possible.

Common features for mustard gas injuries are that the victims do not feel direct contact with the poison, so the medical workers who provide assistance to them must work in protective equipment. Affected persons seek medical help after the end of the hidden period – within a day.

Unlike mustard agents, when affected by lewisite, the victim feels intense pain and skin irritation. The absence of a latent period allows you to immediately start decontamination and leave the infected area. A gray spot

appears on the skin at the place of application of liquid lewisite, after 20–30 minutes a blister forms. In contact with vaporous Lewisite, severe erythema with small foci of hemorrhages is determined on the skin. The focus of necrosis is formed faster than when mustard agents are applied. The full clinical picture develops over 12–18 hours.

Treatment of mustard lesions

There is no specific therapy available for the treatment of mustard lesions. The aim of therapy is to relieve symptoms, prevent infections, and promote healing.

Treatment of lewisite lesions

The antidote for lewisitis is unitiol intramuscularly, intravenously, in the form of applications and ointments. If the eyes are damaged, wash them with water or 0.25 % chloramine solution, use 30 % unitiol ointment. Specific antidote – British anti-Iewisite – may decrease systemic effects of lewisite also. The tactics of treating lesions with lewisite correspond to the principles of treating lesions with mustard agents, taking into account the absence of bone marrow supression and the additional need to control the balance of fluid and the blood coagulation system.

Choking (pulmonary-damaging) agents

Choking agents are chemical substances which cause pulmonary edema, reduced pulmonary compliance and altered gas exchange as a result of pulmonary inflammation when they are inhaled. Choking agents which are used as chemical warfare agents are phosgene (carbonyl dichloride), diphosgene, chlorine and chloropicrin. They are irritant, corrosive and/or chemically highly reactive gases or aerosols. Water-soluble choking agents tend to damage the upper airways and alveoli while water-insoluble choking agents tend to damage the lower airways.

Chemical Warfare Agent	Signs and Symptoms	Pharmacological Treatment
Phosgene Diphosgene	Choking, dyspnea, coughing, burning in the throat and eyes	Supportive treatment N-acetylcysteine and ibuprofen
Chlorine Chloropicrin	Dyspnea, burning in the upper airways and eyes, chough, nausea, chest pain	Supportive treatment

Poisoning by choking agents is characterized by a certain cyclical nature. In the intoxication clinic, the following stages are distinguished: reflex (initial), hidden (fake well-being, latent period), development of toxic pulmonary edema, and remote consequences.

The pathogenesis of phosgene poisoning is complex. One of the main ones is toxic pulmonary edema Ammonia, chlorine, formaldehyde mainly affect the upper respiratory tract.

Toxic pulmonary edema should be taken into account when providing emergency care to a patient. It reaches its maximum at the end of the 1st day, the phenomena of pulmonary edema persist for 2 days. This period accounts for 70–80 % of cases of mortality from phosgene damage. On the 3rd day, the

patient's condition improves and recovery occurs within 4–6 days. However, during this time various complications can happen. Pneumonia develops most often as a result of joining a secondary infection. The second peak of mortality in phosgene poisoning, which occurs on the 9th–10th day, is caused by the progression of pneumonia in the affected patients Another complication is acute myocardial dystrophy, which is manifested by signs of acute heart failure and leads to death on the 4–11th day of the disease from congestive pulmonary edema. Other complications include thrombosis of blood vessels (mainly of the lower extremities, pelvic plexus) and thromboembolism (pulmonary infarctions).

Treatment includes strict bed rest and warming of the victim, oxygen inhalation with foam extinguishers, fight against bronchospasm, pulmonary edema and infectious complications.

Protection

In-service military respirators afford full protection from choking agents.

Blood agents (cyanide compounds)

Cyanide exists in several forms, including the gases hydrogen cyanide and cyanogen chloride, which are classified as blood agents due to the fact that they damage the oxygen-carrying capacity of red blood cells.

Time-dependent symptoms of cyanide poisoning		
System	**Early Signs**	**Later Signs**
Central nervous system	Headache, nausea and vomiting, anxiety, confusion, drowsiness	Altered consciousness, seizure, delirium, lethargy, convulsions, cerebral death
Cardiovascular system	Tachycardia, hypertension	Bradycardia, heart blocks, ventricular arrhythmias, cardiac arrest
Respiratory system	Dyspnoea, tachypnoea	Respiratory depression, non- cardiogenic pulmonary oedema, respiratory arrest
Blood	Bright red venous blood, pH < 7.35 (metabolic acidosis)	
Skin and eyes	Perspiration, bright red skin, cyanosis, mydriasis, eye irritation (following exposure to cyanogen chloride)	

General management of acute cyanide poisoning

Termination of exposure	• Inhalation exposure: removal from the scene of exposure (using appropriate personal protective equipment) • Ingestion exposure: gastric lavage, activated charcoal within 30 minutes • Dermal exposure: decontamination of skin with soap and water
Basic life support	• 100% oxygen (hyperbaric if possible) • Cardiopulmonary support or resuscitation
Advanced life support	• Sodium bicarbonate for metabolic acidosis • Anticonvulsants for seizures • Epinephrine for cardiovascular collapse
Antidotal treatment	• Methaemoglobin-forming agents (4-DMAP! amyl nitrite, or sodium nitrite), not recommended in smoke victims • Sodium thiosulphate • Hydroxycobalamin (smoke-inhalation victims)

Antidote recommendations after exposure to cyanide[1]

Patient	Mild (Conscious)	Severe (Unconscious)	Other Treatment
Child	If patient is conscious and has no other signs or symptoms, antidotes may not be necessary.	Sodium nitrite[2]: 0.12-0.33 ml/kg, not to exceed 10 mL of 3% solution,[3] by slow IV over no less than 5 min, or more slowly if hypotension develops ***and*** Sodium thiosulfate: 1.65 mL/kg of 25 % solution IV over 10-20 min	Nasal oxygen supplementation For sodium nitrite-induced orthostatic hypotension, normal saline infusion and a supine position are recommended. If patient is still apneic after antidote administration, consider sodium bicarbonate for severe acidosis
Adult		Sodium nitrite[2] 10-20 mL of 3% solution[3] by slow IV over no less than 5 min, or more slowly if hypotension develops ***and*** Sodium thiosulfate: 50 mL of 25% solution IV over 10-20 min Alternative: 5 g of hydroxocobalamin in reconstituted solution IV over 15 min	For amyl nitrite,[2] inhaled ampoules titrated to need or until other forms of IV therapy are initiated

[1]Victims whose clothing or skin is contaminated with hydrogen cyanide liquid or solution can secondarily contaminate response personnel by direct contact or through off-gassing vapors. Dermal contact with cyanide-contaminated victims or with the gastric contents of victims who may have ingested cyanide-containing materials should be avoided. Victims exposed only to hydrogen cyanide gas do not pose contamination risks to rescuers. If the patient is a victim of recent smoke inhalation (and thus may have high carboxyhemoglobin levels), only sodium thiosulfate should be administered.

[2]If sodium nitrite is unavailable, administer amyl nitrite by inhalation from crushable ampoules.

[3]Available in cyanide antidote kits, which can be purchased from various manufacturers.

Doses and adverse effects of currently available antidotes

Antidote	Dose	Mechanism	Adverse Effects
4-DMAP	3-4 $mg*kg^{-1}$ 5 ml (50 $mg*ml^{-1}$) intravenously (only one ampoule)	Methaemoglobin formation	Reduction of oxygen carrying capacity, overdose, haemolysis
Amyl nitrite pearls	1 pearl per minute via inhalation	Methaemoglobin formation	Reduction of oxygen carrying capacity
Sodium nitrite	4 $mg*kg^{-1}$ 10 ml (30 $mg*ml^{-1}$) intravenously	Methaemoglobin formation	Reduction of oxygen carrying capacity
Sodium thiosulphate	Ca. 100 $mg*kg^{-1}$ 30 ml (250 $mg*ml^{-1}$) intravenously	Enhancement of metabolism	Concentration > 10 $mg*dl^{-1}$: vomiting, psychosis, arthralgia, myalgia
Hydroxycobalamin	Initial: 5 g Additional: 10 g intravenously	Chelation of cyanide	Transient discoloration (skin, mucous membranes, urine), allergic reactions
Dicobalt edetate	4 $mg*kg^{-1}$ 20 ml (15 $mg*ml^{-1}$) intravenously	Chelation of cyanide	Severe hypotension, cardiac arrhythmias, convulsions

Protection, decontamination and treatment

Exposure to chemical agents is a damaging event, therefore protective measures must be taken, exposed people must be removed from the area, decontaminated and treated to minimize the damage. Rescuers must be use personal protective equipment during decontamination and removal of exposed persons. In the treatment, primarily, ABC (Airway, Breath and Circulation) approach and supportive treatment should be applied. In case of need, specific antidote may be administered, unfortunately only a few agents have specific antidote.

Recognizing and diagnosing health effects of chemical agents

Agent Category	Agent name	Unique Characteristics	Initial effects
Nerve	Cyclohexyl sarin (GF)[1]. Sarin (GB). Soman (GD). Tabun (GA). VX. VR.	Miosis (pinpoint pupils). Copious secretions. Muscle twitching/ fasciculations	Miosis (pinpoint pupils). Blurred/dim vision. Headache. Nausea, vomiting, diarrhea. Copious secretions/sweating. Muscle twitching/ fasciculations. Breathing difficulty. Seizures
Asphyxiant/ Blood	Arsine (SA). Cyanogen chloride (CK). Hydrogen cyanide (AC)	Possible cherry-red skin. Possible cyanosis Possible frostbite[2]	Confusion. Nausea. Gasping for air in some cases; similar to asphyxiation but more abrupt onset. Seizures before death
Choking/ Pulmonary-Damaging	Chlorine (CL). Hydrogen chloride. Nitrogen oxides Phosgene (CG)	Chlorine is a greenish-yellow gas with a pungent odor. Phosgene gas smells like newly mown hay or grass. Possible frostbite[2]	Eye and skin irritation. Airway irritation. Dyspnea, cough. Sore throat. Chest tightness
Blistering/ Vesicant	Mustard (HD)/sulfur mustard (H). Mustard gas (H). Nitrogen mustard (HN-1, HN-2, HN-3). Lewisite (L). Phosgene oxime (CX)	Mustard has an odor like burning garlic or horseradish. Lewisite has an odor like penetrating geranium. Phosgene oxime has a pepperish or pungent odor	Severe irritation. Redness and blisters of the skin. Tearing, conjunctivitis, corneal damage. Mild respiratory distress to marked airway damage. May cause death
Incapacitating/ Behavior-altering	3-Quinuclidinyl benzilate, or Agent 15 (BZ)	May appear as mass drug intoxication with erratic behaviors, shared realistic and distinct hallucinations, disrobing, and confusion. Hyperthermia. Mydriasis (dilated pupils)	Dry mouth and skin. Initial tachycardia. Altered consciousness, delusions, denial of illness, belligerence. Hyperthermia. Ataxia (lack of coordination). Hallucinations. Mydriasis (dilated pupils)

[1]*Letters in parentheses indicate NATO codes for designated agents.*
[2]*Frostbite may occur from skin contact with liquid arsine, cyanogen chloride, or phosgene*

Decontamination and treatment

Agent category	Decontamination	First aid	Other considerations
Nerve	Remove clothing immediately. Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline	Atropine before other measures. Pralidoxime (2-PAM) chloride	Onset of symptoms from dermal contact with liquid forms may be delayed. Repeated antidote administration may be necessary
Asphyxiant/blood	Remove clothing immediately if no frostbite.[1] Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline	Rapid treatment with oxygen. For cyanide, use antidotes (sodium nitrite and then sodium thiosulfate	Arsine and cyanogen chloride may cause delayed pulmonary edema
Choking/ Pulmonary-damaging	Remove clothing immediately if no frostbite.[1] Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline	Fresh air, forced rest. Semiupright position. If signs of respiratory distress are present, oxygen with or without positive airway pressure may be needed. Other supportive therapy, as needed	May cause delayed pulmonary edema, even after a symptom-free period that varies in duration with the amount inhaled

Agent category	Decontamination	First aid	Other considerations
Blistering/Vesicant	Immediate decontamination is essential to minimize damage. Remove clothing immediately. Gently wash skin with soap and water. Do not abrade skin. For eyes, flush with plenty of water or normal saline	Immediately decontaminate skin. Flush eyes with water or normal saline for 10-15 min. If breathing is difficult, give oxygen. Supportive care	Mustard has an asymptomatic latent period. There is no antidote or treatment for mustard. Lewisite causes immediate burning pain, with blisters developing later. Specific antidote —British anti-lewisite — may decrease systemic effects of lewisite. Phosgene oxime causes immediate pain. Possible pulmonary edema
Incapacitating/ Behavior-altering	Remove clothing immediately. Gently wash skin with water or soap and water. Do not abrade skin	Remove heavy clothing. Evaluate mental status. Use restraints as needed. Monitor core temperature carefully. Supportive care	Hyperthermia and self-injury are the greatest risks. Hard to detect because it is an odorless and nonirritating substance. Possible serious arrhythmias. Specific antidote (physostigmine) may be available

[1]*For frostbite areas, DO NOT remove any adhering clothing. Wash area with plenty of warm water to release clothing*

In a chemical mass casualty situation medical resources will be overwhelmed. Triage is a medical decision process used to arrange patients in priority order to ensure the most effective use of limited medical resources and minimize morbidity and mortality. Triage is a dynamic process through the patient care chain used to assign priority for treatment, evacuation and decontamination.

There are different systems for chemical triage. One of the most commonly used contains four categories:

- *Immediate:* This category includes patients requiring emergency life-saving treatment. Treatment should not be time consuming or require numerous, highly trained personnel, and the patient should have a high chance of survival with therapy.
- *Delayed:* The general condition of the patients in this category permits some delay in medical treatment, although some continuing care and pain relief may be required before definitive care is given.
- *Minimal:* This category includes those patients with relatively minor signs and symptoms who can care for themselves or who can be helped by untrained personnel.
- *Expectant:* Patients in this category have a low chance of survival. Life threatening conditions of these patients will be beyond the treatment capabilities of the available medical personnel.

7. Practical skills:

1. Provide the clinical examination of the patient to identify toxic substances poisoning.
2. Distinguish the signs of nerve agents poisoning.
3. Provide emergency aid for nerve agents poisoning.
4. Antidote recommendations after exposure to nerve agents.
5. Identify the signs of blister agents poisonings. Principles of decontamination. Management.
6. Recognize the signs of choking agents poisoning. First aid. Management strategy.
7. Time-dependent symptoms of cyanide poisoning.
8. General management of acute cyanide poisoning.
9. Antidote recommendations after exposure to cyanide.
10. Differential diagnosis.
11. Protection.

8. Questions for control of knowledge

1. Give the definition of chemical agents.
2. Subclasses of chemical agents.
3. Consequences of toxic substances poisoning.
4. Describe the nerve agents poisoning.
5. Describe the blister agents poisoning.
6. Describe the choking agents poisoning.
7. Describe the blood agents poisoning.
8. Describe the behavior-altering agents.
9. Prevention of toxic substances poisoning.

9. Tests for self-assessment of knowledge:

1. What agents can cause bone marrow suppression?

A. Lewisite.	*C Sarin.*	*E. Cyanide.*
B. Arsine.	*D. Phosgene.*	*F. Mustard.*

2. What chemical agents have specific antidote?

A. Phosgene.	*C. Cyanide.*	*E. Lewisite.*
B. Sarin.	*D. Chlorine.*	*F. Mustard.*

3. Nerve agents are ...

A. Cyanide.	*C. Chlorine.*	*E. Sarin.*	*G. Arsine.*
B. Phosgene.	*D. Soman.*	*F. Tabun.*	*H. Sulfur mustard.*

4. In what poisoning N-acetylcysteine and ibuprofen can be useful?

A. Lewisite. *C. Phosgene.* *E. Cyanide.*
B. Nitrogen mustard. *D. Chloropicrin.* *F. Soman.*

5. In what poisoning Diazepam will be useful?

A. Phosgene. *C. Lewisite.* *E. Sulfur. mustard.* G. Arsine.
B. Hydrogen chloride. *D. Tabun.* *F. Cyclohexyl sarin.*

6. Symptoms of muscarinic overstimulation are ...

A. Sweating. *C. Tachycardia.* *E. Salivation.*
B. Paralysis. *D. Vomiting.*

7. What are the most dangerous agents?

A. Blister. *C. Nerve.* *E. Choking.*
B. Behavior altering. *D. Blood.*

8. There is no specific therapy available for the treatment of lewisite lesions.

A. True. *B. False.*

9. In-service military respirators afford full protection from choking agents.

A. True. *B. False.*

10. Intoxication that develops as a result of single or repeated exposure to a toxicant within a limited period of time (usually up to several days) is called subacute.

A. True. *B. False.*

Answers:

1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
A, F	B, C, E	D, E, F	C	D, F	A, D, E	C	B	A	B

10. References and recommended reading:

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4. WHO Library Cataloguing-in-Publication Data. International Programme on Chemical Safety. Guidelines on the prevention of toxic exposures: education and public awareness activities, 2004. Available from: https://www.who.int/ipcs/features/prevention_guidelines.pdf.

5. Disaster Medicine. Comprehensive Principles and Practices / Jonathan Newmark. Edited by Kristi L. Koenig and Carl H. Schultz: 2016 by Cambridge University Press, pp. 499 – 521.
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Навчальне видання

ОТРУЄННЯ ШКІДЛИВИМИ ТА СИЛЬНОДІЮЧИМИ ХІМІЧНИМИ РЕЧОВИНАМИ У ВОЄННИЙ ТА МИРНИЙ ЧАС. КЛАСИФІКАЦІЯ ОТРУЙНИХ РЕЧОВИН. МЕХАНІЗМ ТОКСИЧНОЇ ДІЇ. КЛІНІЧНІ ПРОЯВИ УРАЖЕНЬ ОТРУЙНИМИ РЕЧОВИНАМИ

Методичні вказівки
для здобувачів вищої освіти V року навчання
до проведення практичного заняття

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