DEPARTMENT OF THE ARMY
UNITED STATES ARMY INTELLIGENCE AND SECURITY COMMAND
FREEDOM OF INFORMATION/PRIVACY OFFICE
FORT GEORGE G. MEADE, MARYLAND 20755-5995

REPLY TO
ATTENTION OF:

Freedom of Information/
Privacy Office

MAR 03 2010

Mr. John Greenewald, Jr.
[redacted]

Dear Mr. Greenewald:

This is in further response to your electronic Freedom of Information Act (FOIA) request of June 10, 2009, and supplements our response of August 4, 2009.

Coordination has been completed with other elements of our command and we have been informed by the National Ground Intelligence Center (NGIC) that the records are partially releasable and enclosed for your use.

Since the release of some of the information deleted from the record would result in an unwarranted invasion of the privacy rights of the individuals concerned, this information is exempt from the public disclosure provisions of the FOIA per Title 5 U.S. Code 552 (b)(6).

The withholding of the information described above is a partial denial of your request. This denial is made on behalf of Brigadier General Mary A. Legere, Commander, U.S. Army Intelligence and Security Command, who is the Initial Denial Authority for Army intelligence investigative and security records under the FOIA. You have the right to appeal this decision to the Secretary of the Army. Your appeal must be postmarked no later than 60 calendar days from the date of this letter. After the 60-day period, the case may be considered closed; however, such closure does not preclude you from filing litigation in the courts. You should state the basis of your disagreement with the response and provide justification for a reconsideration of the denial. An appeal may not serve as a request for additional or new information. An appeal may only address information denied in this response. Your appeal is to be made to this office, for forwarding, as appropriate to the Secretary of the Army, Office of the General Counsel.

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We are forwarding a copy of this letter to the Defense Technical Information Center DTIC-R (FOIA 2009-85) and the Department of the Army, Freedom of Information and Privacy Division (FOIA 09-0737).

There are no assessable FOIA fees.

If you have any questions regarding this action, feel free to contact this office at 1-866-548-5651 or email the INSCOM FOIA office at: INSCOM_FOIA_ServiceCenter@mi.army.mil and refer to case #591F-09.

Sincerely,

Joanne Benear
Chief
Freedom of Information/Privacy Office

Enclosure

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DEPARTMENT OF THE ARMY

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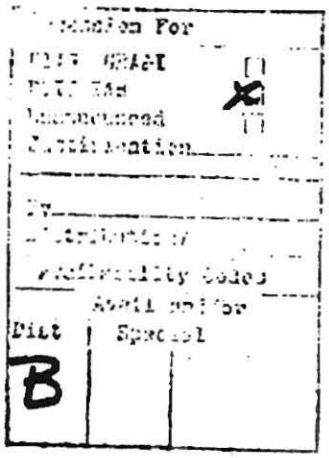

Injuries Caused by Chemical Agents

Militär Medizin Hochschullehrbuch für Studenten der Medizin und Stomatologie (Chapter 5 of Book) pp. 162--190 (Enclo. 1 to IR 2 215 2474 79)

Injuries caused by chemical agents (CA) are familiar from previous wars. Their mass incidence, the rapidly developing symptoms of intoxication, and the restricted possibilities for an efficient treatment impose great demands on the administration of medical aid. At present such intoxications are rare. The clinical treatment and therapy are thus relatively unknown. In view of the continuing threat of the use of chemical agents by the imperialist armies, it is therefore essential to become familiar with the characteristics and effects of chemical agents, described in this chapter, as well as the medical aid for intoxications that occur under combat conditions.

5.1. The Characteristics and Classification of Chemical Agents

Chemical agents are industrially produced chemical compounds for military use against living organisms.

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These are weapons of mass destruction, like nuclear devices; but contrary to the latter, chemical agents present certain basic differences for use, e.g.:

The material resources are preserved and can again be used after conquering the territory.

The effects of chemical agents can be easily varied appropriate to the particular military goals (death, injury with brief or long-term loss of combat or working capability, irritation).

Chemical agents can operate directly against humans, as well as indirectly, by destroying the important stocks of animals and agricultural crops for nutrition.

The manufacture of chemical agents is more cheap than that of nuclear devices with equivalent effects and can be concealed more easily.

The great importance that the imperialist nations still attribute to chemical agents is shown in part by the present re-outfitting of the combat forces of the USA with the "binary system". A feature of this system is the production of the chemical agent only when two components are brought together at the moment of use. These components are separately manufactured and stored in the particular delivery systems (missiles, bombs, projectiles) in isolation from each other. This re-outfitting is certainly not only for military and economic reasons. Apparently the decision has been influenced by considerations of side-stepping legal international agreements, which prohibits the manufacture and storage of chemical agents.

The number of suitable chemical substances is large; furthermore, an intensive research is being carried out primarily in the NATO countries to develop additional chemical agents. In any case, it is not yet possible at present to prepare beforehand a large number of chemical agents in sufficient

quantity for later use in the event of war. Thus at the present in the NATO armies there is only a limited number of standard agents for the various usage scenarios.

As can be seen from Table 5.1, these include the definite offensive agents (GB, BTX, AC, CG, BZ, CS-2), as well as defensive agents (VX, HD, CS-1). These "standard agents", furthermore, can vary the intensity of injury from irritation to death.

The classification of chemical agents in Table 5.1 is based on the medically-decisive pathophysiological effects on the human organism. The most important chemical agents at present are considered to be the nerve agents VX and GB and the blister agent HD. V-agent and Sarin have an exceptionally high toxicity and very quickly produce life-threatening functional disorders. On the other hand, sulfur-yperite causes protracted injuries that are therapeutically difficult to control.

The additional information in Table 5.1 will help an understanding of the use of chemical agents and the course of the damage caused by them. The further reading (5.5) should be consulted for the chemical formulas and physical properties of the chemical agents.

5.2. Principles for the Use of Chemical Agents

As already mentioned, chemical agents can be used with very different objectives. Table 5.2 provides a survey for the objectives and conditions of application. The selection of chemical agents appropriate to the particular military situations is based on the special properties of the individual agents (Table 5.3). From a tactical point of view, combat agents are distinguished from sabotage toxins.

Combat agents can be placed in use by means of various delivery systems

Table 5.1. Characteristics and Classification of Chemical Agents (CA)

Classification by Pathophysiological factors	Agent	USA Army Code	Intended Effect	Period of Latency After Inhalation	Period of Latency After Skin Contact	State of Aggregation	Effective Life (+15°C, Sun, Slight Wind)	Tactical Classification
Nerve Agents	V-agent	VX	Death	10--30 min.	40--120 min.	Liquid	3--21 days	D
	Sarin	GB	Death			Liquid	15--60 min.	O
Toxins	Botulin Toxin A	BTX	Death	3--36 hours	--	Solid	up to 12 hours	O
Tissue Respiration Poison	Hydrocyanic acid	AC	Death	Seconds	--	Liquid	several minutes	O
Lung-Damaging Agent	Phosgene	CG	Death	2--12 hours	--	Gaseous	several minutes	O
Head-Damaging Agent	Sulfur-yperite	HD	Long-term Injury	up to 1 hour	1--12 hours	Liquid	2--7 days	D
Psychochemical Agent		BZ	Temporary unfitness for combat	up to 1 hour	--	Solid	hours	O
Irritants		CS-1	Irritation	none	--	Solid (crystalline)	10--18 days	D
		CS-2	Irritation	none	--	Solid (micropulverized)	hours	O

Note: O = Offensive,
D = Defensive.

(missiles and aircraft with aerosol generators, bombs, projectiles, mines). To inflict mass casualties on the opponent, a surprise attack over a large area should be attempted. Therefc the use of chemical agents as an aerosol by means of missiles and aircraft is of special importance.

According to the information of the USA military, chemical agents as sabotage toxins should be used both in the preparations for a war as well as during its progress, to neutralize important political , economic, and military centers of the enemy. Sabotage poisons are put in use by diversion groups in the interior of the enemy country. Therefore in selecting such agents their high toxicity, possibilities for concealed use, and the poor chances for therapeutic control of th ir intoxications are of special importance. The primary importance in the use of sabotage poisons is the poisoning of foodstuffs and drinking water. Thus sabotage poisons are usually taken up in the human organism via the intestinal tract.

5.3. Important Chemical Agents

We shall describe the most important combat agents below, considering their characteristics and principles of use. Special emphasis is placed on the symptoms of the intoxications, as well as the diagnostic and therapeutic possibilities under combat conditions at the preliminary stages of medical evacuation. Most of the injury from chemical agents requires a very quick and efficient treatment as early as the stage of self-first aid and mutual first aid. Omissions in this initial phase of the injury usually cannot later be made up. This is especially so for the nerve agents.

Table 5.2. Principles for the Use of Chemical Agents (CA), According to the Views of the Imperialist Nations

Purpose of Use	Conditions of Use	CA Group	Most Probable Representative at Present
1. Annihilation by the surprise use of CA in high concentrations	a) A superior (attacking) enemy; b) A defensive enemy in well fortified positions; c) A stable enemy interior	Nerve poisons a) as lethal defensive CA b) as lethal offensive CA	VX Sarin
2. Immobilization by forcing protective measures, in order to create favorable conditions for warfare with conventional weapons	A superior (attacking) opponent; Enemy with slight CA capability	All CA, primarily those that injure the skin or penetrating CA, as in 1, and harmful defensive CA	VK Sarin S-Yperite
3. Exhaustion by forcing the use of protective clothing which greatly hinders bodily movements	When a (superior) enemy has employed (secure) protective equipment in time	As in 2	As in 2
4. Hindrance by creating danger areas (terrain blockade)	Necessity of covering retreat, securing flanks, or isolating enemy reserves from the battlefield	As in 2	As in 2
5. "Selective" killing, primarily for disorganization	When there is no other possibility of keeping decisive forces out of battle; critical situations of the enemy	Nerve poisons, sabotage poisons	VX (Bio-)toxins
6. Temporary unfitness for battle, enabling the capture of prisoners without risk	Superiority over the enemy; immediate contact with the enemy, but also necessity for simultaneous use against friend and foe	Non-inurious CA which produce combat unfitness, primarily psycho-chemical agents with predominant inhibitory effects	BZ

Table 5.3. Probable "Standard" CA of the Imperialist Armies

Standard	Properties	Goal of Use	Currently Most Probable Representative
1. lethal defensive CA	- stationary; - highly toxic percutaneously and by inhalation; - rapid death (without period of latency); - resistant to therapy	- to destroy; - to immobilize; - to exhaust; - to hinder	a "V-agent", e.g. VX
2. injurious defensive CA	- stationary; - highly toxic percutaneously and by inhalation; - causes long-term psychological injury which paralyzes men and equipment	- to injure; - to immobilize; - to exhaust; - to hinder; - to lower the combat morale; - to apply stress to the enemy interior	a S-yperite formula, e.g. with a "G-agent" (component) that has only a damaging effect
3. lethal offensive CA	- volatile; - as in 1.	- to destroy (without affecting the attack of the user)	a "G-agent", e.g. Sarin
4. sabotage poison	- highly-toxic; - no color, odor, or taste; - chemically and physically very stable; - damage resistant to therapy and providing no morphological clues, with - longest-possible period of latency; - metabolized without traces in the organism	- "selective" killing	a toxin
5. non-damaging CA which produces combat unfitness	- non-damaging; - effective in small doses (less than 10 mg)	- capture of prisoners without risk	a psychochemical CA with predominant inhibitory effects, e.g. BZ

5.3.1. Nerve Agents

By their chemical structure, nerve agents belong to the esters of phosphoric and phosphonic acids (organophosphates). Thus they are also known as organophosphate agents. The so-called organophosphates form the basis not only for the nerve agents Tabun, Sarin, Soman and V-agent, but also for a number of insecticides such as Wofatox, Delicia preparation, Bi-58, as well as for certain medications. Intoxications as a result of such insecticides are not uncommon at the present. In principle, they are comparable to the nerve agent intoxications with respect to pathophysiological processes, symptoms, and therapy. The differences in effects of the individual nerve agents on the human organism are so slight that they may be disregarded. We shall therefore discuss the nerve agents as a whole [2,3,4].

Nerve agents are primarily used as an aerosol in combat conditions.

Nerve agents may get into the organism by

the respiratory tract,

the intestinal tract,

or the injured or intact skin or mucosa.

The toxicity of the nerve agents is exceptionally high. Table 5.4 shows that even a minute drop of V-agent, almost too small to be seen, can produce a lethal intoxication.

Depending on the type and the concentration of the agent, as well as the point of entry into the organism, the intoxication symptoms appear several minutes after a period of latency.

Table 5.4. The Toxicity of Nerve Agents

Kampfstoff (A)	Dosis und Art der Einwirkung (B)		
	(C) mittlere tödliche Einatmungsdosis in min · mg m^3	(D) Kampfunfähigkeit verursachende Einatmungsdosis in min · mg m^3	(E) tödlich wirkende Dosis auf ungeschützte Haut in mg
Tabun	100 .. 200	10 ... 100	200 .. 400
Sarin	50 .. 100	5 .. 50	100 .. 200
Soman	25 .. 50	5 ... 25	50 .. 100
V-Stoffe	5 .. 15	0,5 ... 5	2 ... 10

Key: a, agent; b, dose and type of effects; c, medium lethal inhalation dose in min·mg/m^3; d, unfitness for battle produced by an inhalation dose in min·mg/m^3; e, lethal dose on unprotected skin in mg.

The pathogenetic processes in nerve agent intoxication are not yet familiar in all their details. But it is clear that they produce a blocking of esterases, in particular acetylocholinesterase, which results in dysfunction of transmission of the nerve impulse at the so-called cholinergic synapses. To better understand the effect of nerve agents, we shall review the functions of the cholinergic synapses.

As can be seen in fig. 5.1, acetylcholine acts as a "transmitter substance" for the impulse. The intensity of the impulse transmitted at the synapse is a function of the quantity of acetylcholine influencing the cholinergic receptors. The process of impulse transmission is controlled by the enzyme acetylcholinesterase. This enzyme prevents too great a build-up of acetylcholine by rendering an excess of acetylcholine ineffective through hydrolytic splitting.

The action of nerve agents consists in their prolonged accumulation on acetylcholinesterase, thus blocking the enzyme. The result is an excessive increase in acetylcholine ("endogenous acetylcholine intoxication"). The resulting pathophysiological processes at the synapses and their effects on the

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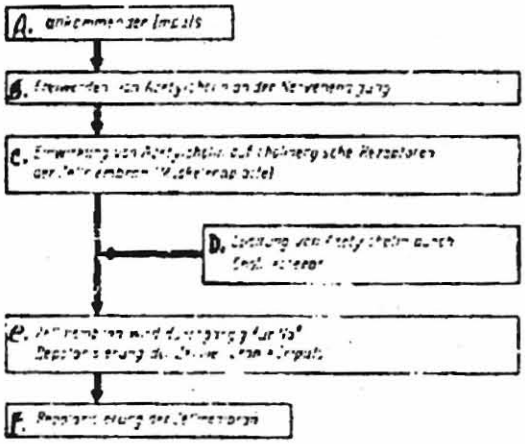

Fig. 5.1. The Functions of a Cholinergic Synapse (Nerve/Muscle). Key: a, arriving impulse; b, liberation of acetylcholine at the nerve ending; c, action of acetylcholine on the cholinergic receptors of the cellular membrane (muscular endplate); d, cleavage of acetylcholine by cholinesterase; e, the cellular membrane becomes permeable to Na^+, depolarization of the cellular membrane = impulse; f, repolarization of the cellular membrane.

particular terminal organs of the nerves are shown in Fig. 5.2 [1].

The functional increase of the terminal organ may be converted to a functional breakdown with increasing accumulation of acetylcholine. Both functional disorders may co-exist, even in different organ systems.

5.3.1.1. The Clinical Picture

The cholinergic synapses are widely distributed in the organism (central nervous system, N. parasympathicus, preganglion sections of the N. sympathicus), so that a disturbance in the transmission of excitation produces dysfunctions in the most diverse organ systems. Depending on the type of affected synapse, the resulting dysfunctions are divided into muscarine-type and nicotine-type effects (Table 5.5).

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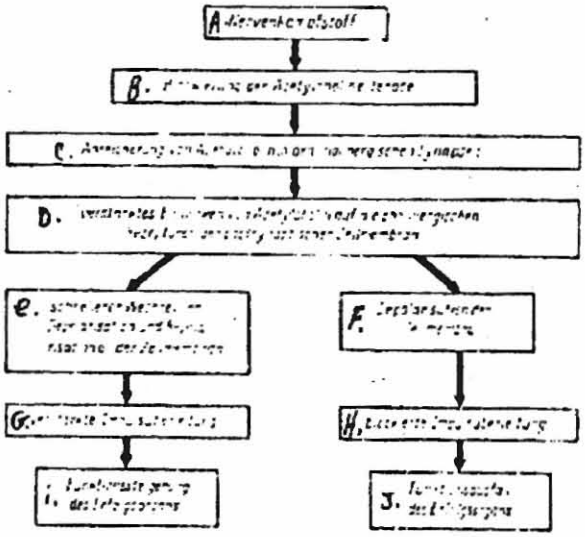

Fig. 5.2. Diagram for the Functional Disorders of a Cholinergic Synapse by the Effect of Nerve Agents. Key: a, nerve agent; b, blocking of acetylcholinesterase; c, accumulation of acetylcholine in the cholinergic synapses; d, intensified action of acetylcholine on the cholinergic receptors of the postsynaptic cellular membrane; e, quickened alternation of depolarization and repolarization at the cellular membrane; f, depolarization of the cellular membrane; g, intensified transmission of the impulse; h, blocked transmission of the impulse; i, functional increase of the terminal organ; j, functional breakdown of the terminal organ.

Table 5.5. Muscarine-Type and Nicotine-Type Effects of Organophosphates

M-Effects	N-Effects
Myosis	Excitation of the CNS
Accommodation weakness	Excitation of the vegetative ganglia
Intensified secretion of the excretory glands	
Bronchospasmus	Tachycardia
Bradycardia	
Hypotonia	Hypertonia
Intensified intestinal peristalsis	Fibrillation of the muscular fibers
General pain	General pain

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The dysfunctions of the following organ systems are decisive for the course of the intoxication:

the respiratory system;

the cardio-vascular system;

the central nervous system.

The various dysfunctions of other organ systems are less important.

The Respiratory System

An acute respiratory insufficiency may develop as soon as several minutes from the beginning of the intoxication. At first there is a depression and acceleration of the respiration. Afterwards bronchospasmus and an increased bronchorrhoea may produce a primarily expiratory insufficiency with protraction of the expiration phase and continual coughing up of secretions. For severe intoxication there is a paralysis of the breathing or of the respiratory muscles. The acute respiratory insufficiency produced by these dysfunctions is the most frequent cause of death in intoxication by nerve agent (Table 5.6).

Table 5.6. Effects of Nerve Agents on the Respiration

Symptoms	Dysfunction	Affected Region
Accelerated depressed breathing, increasing expiratory deficit, rough breathing, coughing up of secretions, symptoms of hypoxia	Excitation of the breathing center, Bronchospasmus, Bronchorrhoea, weakness of the respiratory muscles	Chemoreceptors, breathing center, vagus center, parasympathetic ganglia, bronchial muscles, bronchial glands, respiratory muscles
Cessation of respiration	Central or peripheral paralysis of respiration	Breathing center, respiratory muscles

The Cardio-Vascular System

The dysfunctions of the cardio-vascular system are also decisive for the progress of the intoxication during the first hours and days. A first symptom of the incipient disorder is often a decrease in the pulse rate. The blood pressure may be elevated at first, but later drops off to the shock level in the case of severe intoxication (decrease in the heart time volume and peripheral vascular tonus).

Cardio-vascular failure is an equally important cause of death in nerve agent intoxication (Table 5.7).

Table 5.7. Effects of Nerve Agents on the Cardio-Vascular System

Symptoms	Dysfunction	Affected Area
Blood pressure, normal or elevated, incipient bradycardia	Negative chronotropic effects, heart time volume and peripheral vascular resistance normal or elevated	Cardio-vascular center, excitatory system of the heart
Drop in blood pressure (shock), increasing bradycardia	Drop in the heart time volume (negative ionotropic and chronotropic effects), drop in the peripheral vascular resistance	Cardio-vascular center, vegetative ganglia, heart, arterioles and capillaries

The Central Nervous System

Depending on the variable fluid action of the nerve agents, especially in the case of severe intoxication, dysfunctions of the central nervous system may determine the clinical picture. Of special importance is the disabling of important control centers, e.g. the breathing center and the cario-vascular center. As a symptom of central disorder there also occur tonic-clonic cramps, frequently involving unconsciousness. Also typical are disturbances of the equilibrium apparatus, leading to vertigo, nausea, and vomiting, as well as

agitation, heightened feelings of anxiety, and disturbances of the sleeping/waking rhythm.

Further symptoms of intoxication are:

dysfunctions of the intestinal tract, such as gastro-intestinal spasms, elevated intestinal motility with uncontrollable defecation, increased salivation, nausea, and vomiting.

visual disorders caused by myosis, Argyll-Robertson pupil, accommodation incapability, flowing of tears, conjunctivitis and pain in the visual field.

increased sweating and muscular fibrillation, both generalized and localized at the area of penetration of the nerve agent in the skin.

general muscular weakness.

Depending on the quantity of ingested nerve agent, there are 3 degrees of severity of the nerve agent intoxication of primary concern for medical classification in the event of mass casualties. These are based on the type and intensity of the resulting dysfunctions.

More than half of nerve agent casualties (roughly 70%) are of the second and third degree.

Characteristics of the Degrees of Severity

First degree. A mild form (myotic form) with no serious influencing of the vital organ functions. Symptoms: good general condition, myosis, possibly bradycardia, slightly increased salivation, feeling of pressure in the thorax, slight abdominal discomfort.

Second degree. A medium-severe form (bronchospastic form), in which acute, primarily expiratory insufficiency is the main factor, caused by bronchospasmus and accumulation of secretions in the respiratory passages. Symptoms: general health considerably affected; mental agitation; headache

(in the region of the eyes), gastro-intestinal spasms; vertigo, nausea, possible vomiting and uncontrollable defecation; prolonged, difficult expiration with involvement of the accessory respiratory muscles; loud, bubbling noises during breathing; coughing up of bronchial secretions; increasing symptoms of cyanosis (acren, lips); blood pressure initially normal or raised; possible drop in blood pressure later on; no convulsive conditions. In the absence of medical attention, death by suffocation occurs for a number of the patients.

Third degree. A severe form (generalized or convulsive form), in which life-threatening dysfunctions of the central nervous system determine the clinical picture. Symptoms: tonic-clonic convulsions; increased fading of consciousness or complete loss of consciousness; the initial accelerated and deepened breathing becomes irregular, followed by cessation; drop in blood pressure to the shock level; all other symptoms above occur, but intensified. In the absence of medical attention, death occurs in the first few hours by cessation of breathing or cardio-vascular failure.

5.3.1.2. Diagnostics

The goal of diagnostics is to answer the following questions on the basis of the clinical picture:

1. Is the casualty a nerve agent intoxication?
2. How severe is the intoxication?

There are usually no problems in answering the first question on the basis of the typical symptoms. As diagnostic criteria we should consider: myosis combined with pain in the region of the eyes, bradycardia, increased secretion (salivary glands, nose, eyes), muscular fibrillation, gastro-intestinal spasms, respiratory or cardio-vascular insufficiency, convulsions, loss of consciousness. It is possible to confirm the diagnosis by determination of cholinesterase activity

in the laboratory. But this is not usually necessary, because of the distinct clinical pattern, and under combat conditions at the bandaging stations of the troop units it is not even feasible.

Eliminated by differential diagnosis are:

Exogenous hyperthermia (symptoms of overheating, e.g. bodily strain under protective clothing at high environmental temperatures, tachycardia, mydriasis, body temperature over 40°C).

Intoxication by lung-damaging agents (toxic pulmonary edema after a latent period of several hours, tachycardia, often fever, pupils normal or expanded).

General resorptive injury from yperite intoxication (concurrent local injuries to the skin, mucosa, eyes, latent period of several hours).

Psychoreactive disorders (nonspecific dysfunctions such as nausea, vomiting, uncontrollable defecation, headache, vertigo; absence of myosis and bradycardia).

In order to answer the second question, or to determine the severity, the following attendant symptoms are important:

First degree: myosis, bradycardia, intact respiration, stable blood pressure, no convulsions, no loss of consciousness.

Second degree: primarily expiratory insufficiency (breathing noises, coughing up of secretions, possible symptoms of hypoxia), no convulsions, loss of consciousness only in the final stage.

Third degree: tonic-clonic convulsions, loss of consciousness, cessation of breathing, drop in blood pressure (shock!).

5.3.1.3. Therapy

Among the therapy for nerve agent intoxication there are:

Measures to remove the nerve agent from the surface of the body or from the organism (detoxication, cf. 7.1.4.2, and alleviation of resorption),

Administration of antidotes,

Measures to maintain the vital body functions.

Antidote therapy produces

a reactivation of the esterase

the shielding of the cholinergic receptors against the excessive action of acetylcholine.

The reactivation of the cholinesterase is done by the so-called cholinesterase reactivators, which release the bond between the enzyme and the nerve agent. Those most commonly used are:

obidoxim (S-100);

trimedoxim (TMB-4);

pralidoxim (2-PAM).

When using cholinesterase reactors, it is important to take the following into account:

Their action is limited in time; their application is effective only in the first few hours of nerve agent intoxication.

Cholinesterase blocked by Soman can only be reactivated to a very slight extent.

The reactivators have undesirable side-effects. Therefore no more than two single doses should be given.

At present the average choice is obidoxim (relatively slight side-effects, good effectiveness in the central nervous system).

It is possible to shield the cholinergic receptors in the synapses against the increased quantity of available acetylcholine by the administration of cholinolytics. Foremost is atropine. The following factors are important:

The individual dose for atropine is extremely high for nerve agent intoxication (2 to 3 mg).

The administration of atropine, depending on the severity of the intoxication, must be renewed every 2 to 4 hours, or even more frequently for severe intoxications.

The dose should be selected so that there is dryness of the mucosa.

The atropine medication is continued until the symptoms abate (on the average, 2 to 3 days, as many as 10 to 14 days for severe cases).

During the first few days the symptomatic treatment measures are concentrated on maintaining the vital functions of the organs. Most important are:

insurance of the respiration. In the event of respiratory insufficiency as a result of bronchospasmus and bronchorrhoea:

the secretions must be suctioned out of the respiratory pathways;

the bronchia should be expanded by atropine/ephedrine spray;

oxygen insufflation (nasal catheter).

in the event of a threatening cessation of the respiration (peripherally or centrally caused):

intubation or tracheotomy;

forced breathing (manual or machine).

insurance of the cardio-vascular function

infusion therapy (shock control), administration of medication to increase the blood pressure (angiotensin, noradrenaline) in the event of a drop in blood pressure.

control of convulsive and excited conditions

administration of faustan i.v. or short-term narcotics.

buffering of the frequently occurring metabolic acidosis

infusion therapy, e.g. sodium bicarbonate.

Antibiotics should also be administered to protect against secondary infections during the standard treatment. If there are strong gastro-intestinal spasms, papaverin should be given in addition, while atropine oil for the eyes (1%) should be given for eye pains.

At present the optimum therapy for nerve agent intoxication is:

earliest possible combination antidote dose, e.g. 250 mg obidoxim plus 2 mg atropine (within the first minutes after contact with the agent).

the combined antidote dose should be repeated after 20 to 30 minutes.

afterwards, carry out the antidote treatment with atropine again, depending on the symptoms (subcutaneous or intravenous injections, or as a component of intravenous drop infusion).

symptomatic measures to maintain the important organ functions (cf. above) should accompany the antidote treatment.

The Administration of Medical Aid Under Combat Conditions

The administration of medical aid for those injured by nerve agents under combat conditions places extremely high demands on the personnel of the medical service, especially at the bandaging stations. This is a result of:

the rapid commencement of the intoxication symptoms and

the high proportion of severely injured casualties for this type of injury.

In keeping with the above, the first aid measures are carried out at the preliminary stages of medical evacuation (Tables 5.8 to 5.10).

Table 5.8. Self-Help and Mutual Aid, as Well as Medical First Aid in a Contaminated Territory for Nerve Agent Injuries

Important:
- The prompt and proper execution of the measures for self-help and mutual aid are of decisive importance for preserving the life of the casualties.
- The sequence of carrying out the measures is important.

Measures	Medication/Remedy
1. Partial sanitary treatment (decontamination of the uncovered parts of the skin)	Decontamination packet (skin decontaminant, cloth)
2. Implacement of the protective mask	
3. Intramuscular injection of antidote (through the clothing)	Medical protection packet (plastic quick-action syringe)
4. Removal of perceptible spatters of agent from the clothing	Decontamination packet (skin decontaminant, cloth)
5. Covering with protective clothing	
6. Rescue from the contaminated territory	

Table 5.9. Medical First Aid Outside the Contaminated Territory for Nerve Agent Casualties

Important:
- Artificial respiration requires that the protective mask be taken off after completing a partial sanitary treatment (only possible in special cases).

Measures	Medication/Remedy
Partial sanitary treatment, if an insufficient treatment or none at all has been performed	Decontamination packet (skin decontaminant, cloth)
Rinsing of the eyes, mouth, and throat	Water, contents of a canteen (tea, coffee)
Intramuscular injections of antidote (2 doses)	Plastic quick-action syringe
Dose of antitussins for the appropriate Dose of spasmolytics symptoms	Eucopon, 1 tablet Papverin, 0.2 g
Artificial respiration (manual) in the case of respiratory cessation	Small respirator
In the event of peroral ingestion/ inhalation: inducement of vomiting and/or non-catheter rinsing of the stomach, dose of saline purgative	Suspension of activated charcoal (50 g/1.5 NaCl solution or contents of canteen, magnesium sulfate (1 tablespoon per glass of water)

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Table 5.10. Medical First Aid for Nerve Agent Casualties

Important:
Only minutes are available for the classification and treatment of a victim when there are mass casualties. This requires a concentration on the execution of simple and quick life-saving measures to enable transport (critical measures).

Measures	Medication/Remedy
Urgent:	
Continuation of the partial sanitary treatment and of the measures to alleviate resorption (cf. medical first aid)	Decontamination kit, also medical first aid
Intramuscular injection of antidote if this has not yet been done twice. Otherwise repeated atropine injections (ED 0.002 g)	Plastic quick-action syringe
Insurance of the respiratory function - keeping clear the breathing passages (suctioning out of secretions) - oxygen insufflation, if necessary manual forced breathing after intubation or coniotomy - insurance of the cardio-vascular function, simple shock control measures	Oxygen inhalation device, small respirator, intubation/coniotomy case pholedrin or depotpholedrin 0.02
Contingent:	
Application of:	
- spasmolytics	Papaverin Tabl. 0.02 g
- antitussins	Eucopon 1 Tablet
- sedatives/transquilizers	Sinophenin 0.05--0.15 g i.m./i.v. or Faustan 0.01 g i.m./i.v.
- atropine oil for the eyes	Atropine oil for the eyes (1%)

The medical classification of nerve agent casualties is based on the severity of the intoxication. The following principles are important:

All nerve agent casualties are directed from distribution points to the sanitary treatment area. Only after the sanitary treatment can the treatment and evacuation classification be carried out.

The sanitary treatment and the treatment classification are coincident with the administration of critical first aid.

Second and third degree casualties always require urgent treatment measures and have priority over first degree casualties.

The evacuation of second and third degree casualties has first priority and is done with the victim lying down, as directly as possible to a medical facility that has specialized or at least qualified internists. The antidote treatment should be continued during the evacuation.

First degree casualties are to be evacuated seated, by second priority, when absolutely necessary.

5.3.2. Skin-Damaging Agents

Among the skin-damaging agents (sulfur and nitrogen yperite, lewisite, phosgene oxime), sulfur yperite is the most important at present. Therefore the following remarks concern this agent.

Sulfur yperite can get into the organism by many pathways:

through the intact or damaged skin, as well as through the conjunctiva,

across the respiratory tract

across the intestinal tract.

Its toxicity is shown in Table 5.11. This is substantially less than that of nerve agents.

The pathogenesis of yperite intoxication is not absolutely clarified. Among the various hypotheses proposed to account for the intoxication symptoms, only the following deserve mention.

The hypothesis of alkylation of important natural body substances: in particular, the alkylation of guanine (a component of DNA) is supposed to be responsible for the disturbed protein synthesis.

The hypothesis of cation effects, which supposedly lead to a reversible blocking of cholinesterase, which is only important in severe intoxication.

Table 5.11. Toxicity of Sulfur Yperite

Injury	Percutaneous Intake mg/kg	Inhalative Intake Dose in mg/l air	Inhalative Intake Exposure Time in Min.
Irritation of the eyes		0.0005	10--25
Combat unfitness		0.001	8--10
Exitus letalis	40--60	0.7	30

Fig. 5.3. S-Yperite Injury Roughly 1 Day After Contamination. Amber-colored coronoid vesicular eruption. The grayish-yellow ischemic center stands out clearly against the dark-red hyperemic erythema of the surrounding skin.

Fig. 5.4. Severe Vesicular Yperite Damage to the Skin 8 Hours Afterwards. By the increased exudation in the anemic center the coronoid vesicle has expanded centripedally. The livid red, edematous, sharply defined erythema of the surrounding skin can be clearly seen.

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Fig. 5.5. After Removal of the Covering Vesicle, the Necrotic, Greasy Wound Surface Can Be Seen.

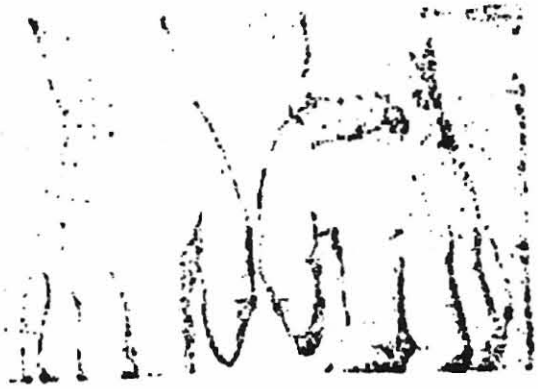

Fig. 5.6. Under Involution of the Erythema and Edema, There is a Gradual Demarcation of the Adherent Necrotic Tissue in the Region of Ischemic Injury After Two Weeks.

Fig. 5.7. Continuing Demarcation of the Tissue Necrosis, Four Weeks After S-Yperite Injury.

Fig. 5.8. Seven Weeks After S-Yperite Contact the Necrotic Tissue is Almost Entirely Shed. In the region of skin damaged by the agent there is delicate cicatrization with partially restored cleavages of the skin. In the region of the cicatrix there are depigmentation and hyperpigmentation, with isolated telangiectases.

5.3.2.1. The Clinical Picture

The clinical picture of yperite intoxication is determined by

local injuries at the point of entry of the agent into the organism and by

toxic general symptoms after resorption of the agent.

Yperite injuries display a number of characteristic features:

The presence of yperite on the body surface causes at first no irritation.

The symptoms of the injury occur after a latent period of 2 to 12 hours.

A short latent period and the presence of toxic general symptoms indicate a severe injury.

The frequent occurrence of secondary infections and the poor healing tendency of the local injuries necessitate a lengthy (several weeks or months continuously) treatment.

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Local injuries may be present at the following regions of the organs/body:

skin/external mucosa;

eyes;

respiratory tract;

intestinal tract.

Skin injuries manifest different symptoms in dependence on the concentration of the agent and the length of its action on the skin. Table 5.12 and Figs. 5.3 to 5.8 provide an overview.

Table 5.12. Forms of Skin Damage by Yperite

Type of Skin Damage	Latent Period		
Erythematous form	roughly 12 hours	Light burning or itching, increasing sensitivity to touch, erythema	Healing after 5--10 days without cicatrization, leaving behind a pigmentation
Superficial bullous form	6--12 hrs.	Begins as the above. A perceptible infiltrate (wheallike) results from the erythema. After 10 to 12 hours pale ischemic foci occur in the infiltrate. In this region small, afterwards confluent bubbles (amber-yellow contents) subsequently develop	Healing occurs after a stage of erosion in 3--4 weeks without cicatrization, leaving behind a pigmentation
Deep bullous form	2--6 hrs.	Beginning as the erythematus form. In the center of the injury there is at first swelling and considerable infiltration, which is then grayish-yellow colored and surrounded by bubbles arranged like a string of pearls. In this region there later develops an ulcerative-necrotic process.	Healing results after 2 to 3 months, with formation of a depigmented and hyperpigmented cicatrix. Secondary infection is typical

Injuries to the eyes by yperite are relatively frequent. Even small yperite concentrations (aerosol) produce an irritation with a sensation of foreign objects, burns, flow of tears, and photophobia. More severe effects of yperite (yperite drops, viscous yperite) involve destructive changes which often lead to loss of the eye.

Injuries of the respiratory tract occur after inhalation of yperite (aerosol, yperite vapors). For a small concentration of yperite or a short exposure, the inflammatory-necrotic alterations that develop are primarily restricted to the upper air passages (toxic rhino-pharyngitis and tracheitis). Stronger yperite effects also lead to the formation of toxic bronchitis or bronchopneumonia. A toxic lung edema may occur at the same time.

Clinical symptoms are: irritation-excited cough, scratching in the neck, retrosternal pain, bloody/purulent sputum, possible symptoms of toxic lung edema.

The healing process requires weeks to months. Lung abscesses may occur during the course of the pneumonia. Peeling membranes may cause a closure of the upper air passages.

An interstitial lung emphysema and chronic bronchitis have been observed as later consequences.

The injuries of the intestinal tract are a consequence of the ingestion of poisoned food and drink. In this region there also develop inflammatory-necrotic processes, accompanied by stomach pain, salivation, nausea and vomiting, as well as bloody diarrhea on occasion. More intense intestinal bleeding and perforation are possible. For these injuries the healing process is greatly delayed and there often remain degenerative changes in the mucosa with chronic dyspeptic complaints.

The toxic general injury of severe yperite casualties is manifested by an increasing worsening of the general condition, attended by a decrease in body weight, lack of appetite, weakness and disturbed sleep. Furthermore, there are functional disturbances of various organ systems:

the cardio-vascular system: initial increase in blood pressure, later a drop in blood pressure (bradycardia, vertigo, sweating, headache, feeling of weakness).

the respiratory organs: respiratory insufficiency due to bronchospasmus and increased bronchial secretion (accelerated rough breathing with protracted expiration, coughing up of secretions, involvement of the accessory respiratory musculature, possibly cyanosis).

the gasto-intestinal tract: increased motility of the gastro-intestinal tract (nausea, vomiting, body pain, diarrhea).

the hemopoietic organs: toxic damage to the bone marrow (leucopenia at the seventh or eighth day after intoxication).

the central nervous system: lack of movement, somnolence, contact receptor impairment, and general weakness (in the case of nitrogen yperite, compulsive movement, possibly tonic-clonic convulsions).

The proper assessment of the severity of an yperite intoxication may be difficult in view of the multifarious symptoms of the injury. Of decisive importance are the extent of damaged skin surface and the presence or absence of symptoms of toxic general injury (Table 5.13).

Table 5.13. Severity of Yperite Injury

Severity	Characteristics
I	Slight local injuries erythema, possible formation of isolated small blisters, reddened conjunctiva
II	Moderately pronounced local injuries blistering in the region of extensive skin sections. reddened and swollen conjunctiva. - after inhalation of the agent: scratching in the neck, hoarseness, irritation excited cough. - after peroral ingestion of the agent: nausea, body pain
III	Severe local injuries/symptoms of general injury damage of extremely large skin sections. considerable swelling of the eyelids and conjunctiva. - after inhalation of the agent: symptoms of toxic lung edema, cyanosis. - after peroral ingestion of the agent: strong body pain, repeated vomiting, diarrhea (mixed with blood). increasing worsening of the general condition. Bradycardia, drop in blood pressure, loss of body weight, apathy.

5.3.2.2. Diagnostics

The recognition of an yperite injury presents few difficulties, due to the typical skin changes after the period of latency. Nonetheless an yperite contact is frequently not recognized.

By differential diagnosis there should be eliminated:

nerve agent intoxication (no local indications of injury).

intoxication by lung-damaging agents (possible inflammations of the skin occur immediately, toxic lung edema only after 10 to 15 hours).

lewisite intoxication (skin changes occur basically without a latent period).

acute radiation sickness (detection of radiation exposure, typical periodic progress with initial primary and subsequent latency periods).

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5.3.2.3. Therapy

At present there is no antidote for yperite. The symptomatic complex therapy involves:

measures to remove the agent from the body surface or from the organism;

early administration of antibiotics (second and third degree severity);

therapy for the local injuries;

therapy for the toxic general injury.

The measures for removing the yperite from the body surface or from the organism are shown in Tables 5.14 through 5.16, and also discussed in Chapter 7.

Early sanitary treatment is also crucial for the severity of the developing injury. Important in this regard are:

Decontamination of the skin within the first 15 minutes after contact with the yperite can greatly minimize or even prevent the injury.

Yperite that has penetrated to the bloodstream can be neutralized within the first hours after contact by sodium hyposulfite (10 to 15 ml of 30% solution i.v.).

Table 5.14. Self-Help and Mutual Aid as Well as Medical First Aid in a Contaminated Territory for Yperite-Caused Injury

Important:
The sequence for carrying out the measures is important. They must be executed as quickly as possible.

Measures	Medication/Remedy
1. Partial sanitary treatment (decontamination of uncovered skin areas)	Decontamination packet (skin decontaminant, cloth)
2. Emplacement of the protective mask	
3. Removal of noticeable spatters of agent from the clothing	Decontamination packet (skin decontaminant, cloth)
4. Covering with protective clothing	
5. Rescue of the casualties from the contaminated territory	

Table 5.15. Medical First Aid Outside the Contaminated Territory for Yperite Casualties

Measures	Medication/Remedy
Continuation of the partial sanitary treatment	Decontamination packet (skin decontaminant, cloth)
Intense rinsing of the eyes, mouth cavity, and nose	Chloramine solution (0.25%), in an emergency water, tea, coffee
If an injection of the agent by the respiratory or intestinal tract is suspected:	
mechanical inducement of vomiting, if possible after plentiful intake of fluids with adsorbents.	Activated charcoal suspension (50 g/1.5 l) or contents of canteen
Administration of saline purgative	Magnesium sulfate (20 g per 1 glass water)

Table 5.16. Medical First Aid for Yperite Casualties

Important:
Of first priority are the measures for alleviation of resorption and for maintenance of the vital organ functions.

Measures	Medication/Remedy
Urgent:	
Continuation of the partial sanitary treatment and other measures for resorption alleviation (if this has not yet been satisfactorily achieved)	Decontamination kit, activated charcoal suspension, magnesium sulfate
Stabilization of the circulation	Pholedrin/deporpholedrin (0.02 g s.c./i.m.)
Clearing of the respiratory passages	Foot suction pump
Application of protective bandages	Bandage kit, decontaminant
Alleviation of pain	Dolcontral (0.1 g i.m.)
Administration of eye drops	Tolazolin eye drops (1 drop per hour)
Contingent:	
Application of	
antemetics	Marophen (0.054 g i.m.)
antitussins	Eucopon (1 tablet)
antibiotics	Berlicetin or OTC (2 g/day p.o.)
spasmolytics	Atropine (0.001 g s.c.)

The therapy for local injuries

The skin: cover the skin damage using a bandage with 2% chloramine or 1% rivanol or potassium permanganate solution, or a sterile bandage at the least. The blisters are to be left alone or, if necessary, punctured under sterile conditions. The wounds are treated with "skin-sparing" salve.

The eyes: intense rinsing of the eyes (0.25% chloramine solution, water in an emergency)! Then 1 drop of tolazoline (10%) eye drops per hour. Cleaning of the eyes (agglutinations!). Application of OTC salve.

The respiratory tract: The patient is to be placed in a position of bodily and mental rest. The irritated coughing is to be controlled (antitussins). For symptoms of hypoxia, O_2 insufflation. For toxic lung edema, cf. 5.5. Respiratory exercises for the later course of the treatment.

The intestinal tract: Alleviation of the pain and nausea by spasmolytics and antemetics. Plentiful liquid intake, by infusion in an emergency. Easily-digestible food (liquid or semi-fluid), rich in calories, protein, and vitamins. In severe cases, parenteral feeding.

The therapy for the toxic general injury. In the initial phase of the intoxication, the maintenance of the vital body functions is of primary importance.

Cardio-Vascular Activity: Administration of glycosides, stabilization of blood pressure (medication, if necessary infusion treatment).

Respiration: Suctioning out of bronchial secretions, expansion of the bronchia by medication (e.g. ephedrine spray), oxygen insufflation, in an emergency artificial respiration following intubation.

Measures during the later course of the intoxication

Making up the protein losses (disturbed protein synthesis) by protein-rich food, possible administration of protein hydrolysates.

Administration of easily-absorbable carbohydrates and polyvitamin preparations.

For hemopoietic disorder, several blood transfusions (each 150 to 200 ml).

Medical assistance under combat conditions

After yperite has been used, the thorough execution of all measures to restrict the further effects of the agent is of first priority.

The administration of medical aid at the preliminary stages of medical evacuation includes the measures shown in Tables 5.14 through 5.16.

For the medical classification of yperite victims, the following principles apply:

The victims are to be classified only after sanitary treatment.

Third degree casualties receive priority treatment.

Third degree casualties are to be evacuated in recumbent position by first priority, second degree casualties in the seated or recumbent position by second priority, to a medical facility that has qualified or specialized internists. First degree casualties are evacuated to a field hospital only in exceptional cases.

5.3.3. Lung-Damaging Agents

Among the lung-damaging agents (phosgene, diphosgene, chloropicrin), phosgene still retains special military significance. Therefore the remarks concern this agent [2,3,4].

Phosgene is taken into the organism only across the respiratory tract.

We do not yet possess comprehensive information on the pathogenesis of phosgene intoxication. It is assumed that it produces a disturbance of the metabolism in the pulmonary alveoli and capillaries by inhibition of the natural bodily enzymes.

5.3.3.1. The Clinical Picture

Phosgene intoxication progresses with typical phases. The following can be distinguished:

the reflector stage;

the latent stage;

the stage of toxic lung edema;

the regressive stage.

The reflector stage is poorly manifested for phosgene or may be entirely absent (it is more pronounced for diphosgene). Immediately upon inhalation of the agent there may occur an inflammation of the respiratory passages with sneezing and irritated coughing, as well as scratching in the throat. After 30 to 60 minutes, these symptoms completely disappear.

The subsequent latent stage lasts from 4 to 12 hours. Even for severe intoxications, there is complete freedom from complaint during this time. Only an accelerated breathing with simultaneous bradycardia can be detected.

The transition to the stage of toxic lung edema is ushered in by an impairment of the general condition, irritated coughing, vertigo, a bad taste in the mouth, and increasing tachypnoea.

As a result of the damage to the alveolar epithelia and lung capillaries there is a considerable accumulation of fluids in the alveoli. This may withdraw up to 30% of the circulating blood from the bloodstream.

The consequences of this change are:

disturbance of the oxygen absorption of the blood in the lungs and of the oxygen release in the cells;

increasing hypoxia with damage primarily to the organs with a large oxygen consumption (brain, heart, kidneys);

decrease in the circulating blood volume, coagulation of the blood;

right heart insufficiency, reduction of the heart time volume, drop in blood pressure, slowing of the blood flow.

The toxic lung edema reaches its climax at the second day of intoxication. The clinical picture is then characterized by

fever, considerable mental unrest;

large inspiratory deficit;

painful irritated coughing with copious expectoration of brownish foam and severe retrosternal/epigastral pain;

loud, gurgling, rattling noises over all the lung sections, audible as a distant noise even without stethoscope;

increasing cyanosis (depending on the appearance of the victim, one distinguishes between the so-called blue cyanosis with yet intact circulation and the subsequent so-called gray cyanosis with secondary circulatory failure).

The peak mortality of phosgene intoxication occurs during this period of toxic lung edema. If this stage is survived, there is an improvement of the general condition after 24 to 36 hours, lytic defevescence, and relief of the respiratory insufficiency. The regressive stage, commencing in this manner, requires 4 to 6 days for the uncomplicated form. But at any rate there often occur complications between the eighth and tenth day of intoxication, in particular:

bronchopneumonia,

thrombosis (especially of the lower limbs), cerebral embolism, coronary and pulmonary infarct

secondary heart insufficiency.

These complications may greatly prolong the period of recuperation or even cause death after recovery from the toxic lung edema.

Damage to the heart muscle, neuropathy and encephalopathy, and metabolic disorders have been observed as late complications.

5.3.3.2. Diagnostics

The minor initial inflammations (if at all present) can also be caused by irritant agents, or they may not always be noticed.

Even the tachypnoea and bradycardia may have other causes (disturbances of the vegetative nervous system, injury from nerve agents). They are not usually noticed.

There is a suspicion of phosgene intoxication if

mild inflammations of the respiratory organs occur immediately after the use of chemical agents, these symptoms diminishing after 30 to 60 minutes,

subsequently tachypnoea and bradycardia remain, despite general well-being, and if

symptoms of incipient lung edema occur after 4 to 12 hours.

By differential diagnosis, the following are to be separated from phosgene intoxication:

nerve agent injuries (no initial inflammation, myosis, gastro-intestinal spasms).

yperite injuries (typical epidermal symptoms after a period of latency).

injuries due to irritant agents (usually more pronounced inflammation, although a distinction is difficult, as high doses can also produce a toxic lung edema).

psychoreactive changes (no inflammation of the respiratory organs).

5.3.3.3. Therapy

The therapy for phosgene injury necessitates above all measures for the prophylaxis and treatment of toxic lung edema.

The immediate application of hexamethylene tetramine (3 g peroral or 20 ml of a 20% solution i.v.) can neutralize the intoxication (cannot be realized under combat conditions!).

Measures for prophylaxis of toxic lung edema:

absolute rest for the victim, even during the latent phase;

prevention of heat loss;

avoidance of fluids;

careful evacuation (recumbent with the upper body raised);

prednisolute injection (0.1 g i.v.).

Measures during incipient or manifest toxic lung edema:

corticosteroid therapy (at once and after one hour, each time 0.1 to 0.2 g prednisolute i.v.);

pressurized respiration (expiration against a resistance counteracts the formation of edema);

oxygen insufflation (6 l/min) with frequent suctioning of secretions from the respiratory passages (respiration with pressure excess is counterindicated, due to the danger of additional damage to the alveoli);

intravenous infusion of a high-percentage sugar solution (100 ml of 40% sorbite solution and/or 20 ml of 20% calcium gluconate solution);

mental rest, e.g. by Faustan (0.01 g i.v.);

bloodless venesection (unbending of the legs from proximal to distal following deep rest), blood letting only as ultima ratio;

guarding against infectious complications, mainly pneumonia (penicillin, 1 to 2 million IE/day);

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support of the heart activity by a dose of strophanthin (0.00025 g/day).

The administration of medical aid under combat conditions is summarized in Tables 5.17 and 5.18.

An effective therapy for the toxic lung edema is only possible in medical facilities that have qualified or specialized internists.

Medical classification is based on the following principles:

The severity of the injury cannot be assessed prior to the commencement of the toxic lung edema and therefore every casualty of lung-damaging agents should be evacuated by first priority (recumbent with raised upper body), in order to reach a medical facility with qualified or specialized internists as early as possible before the commencement of the toxic lung edema.

All victims of lung-damaging agents require urgent measures during the medical first aid (prophylaxis or therapy for toxic lung edema).

If more than 15 hours have elapsed after exposure to phosgene without symptoms of toxic lung edema, there is no longer any danger.

Table 5.17. Self-Help and Mutual Aid, As Well As Medical First Aid, for Phosgene Injury

Important: A sanitary treatment of the victim is not necessary. There should be an earliest possible evacuation (before the commencement of toxic lung edema) to a medical facility with at least qualified internists.	
Measures	**Medication/Remedy**
- absolute body rest - raising of the upper body while lying down - prevention of heat losses - no intake of fluids	chemical thermal sack

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Table 5.18. Medical First Aid for Phosgene Injury

Measures	Medication/Remedy
Important: Earliest possible evacuation while observing the measures for prophylaxis of toxic lung edema.	
Urgent:	
Threatening lung edema:	
- corticosteroid prophylaxis	Prednisolute (0.1 g i.v.)
- general measures (self-help and mutual aid as well as medical first aid)	
Manifest lung edema:	
- corticosteroid therapy	Prednisolute (2x0.1 g i.v.) at an interval of 1 hour
- mental rest	Faustan (0.01 g i.m./i.v.)
- enhancement of the heart function	Strophanthin (0.00025 g i.v.)
- stimulation of diuresis	Disalunil (0.1 g i.v.)
- dehydration	Sorbitol 400, 100 ml as intravenous infusion
- general measures (cf. above)	
Contingent:	
- infection prophylaxis	Retacillin compositum (1.2 million IE i.m.
- alleviation of the irritated coughing	Eucopen (2 x 1 tablet/day)

5.3.4. Injuries Due to Tissue-Respiration Poisons

Among the tissue-respiration poisons are primarily hydrocyanic acid and halogen cyanogens. Hydrocyanic acid is important as a chemical agent. It can penetrate the organism through the respiratory or intestinal tracts, as well as through the intact skin [2,3,4].

When employed as a combat agent, intake is primarily by inhalation; when used as a sabotage poison, the intake is peroral.

The pathogenesis of this intoxication is relatively well known. Hydrocyanic acid in the organism forms a bond with the organic iron of hemin, thus blocking in this manner the so-called respiratory enzyme cytochromeoxydase. This disturbs

the transfer of oxygen from the erythrocytes to the tissue cells. Despite a sufficient supply of oxygen through the respiratory organs and the blood, there results a tissue hypoxia (so-called internal suffocation).

5.3.4.1. The Clinical Picture

Hydrocyanic acid intoxication has an extremely rapid course. The issue of the intoxication is decided in the first half hour.

We may distinguish four stages of intoxication:

The initial stage: Brief sensation of a metal taste, without latent period. Illusory sensations in the region of the tongue and palate. Increased salivation, nausea and vomiting. Incipient speech disorder.

The "asthmatic" stage: Pronounced dispnoea, pain in the region of the thorax, exophthalmus, mydriasis, disturbances of coordination.

The convulsive stage: Tonic-clonic convulsions, simultaneously increasing respiratory deficit and fading consciousness.

The asphyctic (paralytic) stage: Cessation of breathing while convulsions still persist; heart activity may still be detected for several minutes.

The effects of a high dose lead to "reflex death" within several seconds. If the respiration is not basically disturbed after one hour, there is no longer danger to life [1].

5.3.4.2. Diagnostics

For severe injuries diagnosis is too late. Otherwise the diagnosis is based on the clinical condition. Typical are:

rapidly increasing dispnoea with pink coloration of the skin,

an odor of bitter almonds,

mydriasis.

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By differential diagnosis one should exclude:

nerve agent injury (myosis, bradycardia, cyanotic appearance with respiratory insufficiency)

intoxication by lung-damaging agents (latent period, symptoms of toxic lung edema, cyanosis).

5.3.4.3. Therapy

The rapid course of the intoxication demands an immediate commencement of therapy.

Cobalt EDTA (0.3 g i.v. several times for heavy metal bonding of the cyanogen ion) in combination with hemiglobin forming drugs is considered to be the most effective antidote. The portion of the hemoglobin not converted into hemiglobin binds the cyanogen ion at any rate. This complex is then neutralized by the formation of non-toxic thiocyanates from the cyanogen compounds. Hemiglobin formation:

4-DAMP (4-Dimethylaminophenol hydrochloride), 0.003 to 0.005 g/kg of body weight i.v. or

isoamyl nitrite, inhalation of the contents of 3 ampules for 30 seconds at intervals of two minutes.

Neutralization of the cyanogen/hemoglobin complex:

sodium thiosulfate (10%), 60 ml i.v. or

coloxide, 3 to 5 times, 25 to 30 ml i.v. at intervals of 10 minutes.

Oxygen doses (if possible by excess pressure respiration) and exchange transfusions may favorably influence the outcome. In the event of peroral intake of the agents, an immediate stomach rinsing with 0.2% $KMnO_4$ solution is essential.

The administration of medical aid under combat conditions

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Immediate antidote treatment is a matter of life and death.

Self-help and mutual aid, as well as medical first aid: administration of the antidote from the protective packet.

Medical first aid: urgent:

coloxide, 3 to 5 times, 25 to 35 ml i.v. at intervals of 10 minutes or sodium thiosulfate solution (10%), 1 to 2 times, 50 to 100 ml i.v.; oxygen insufflation.

The later treatment is done in a field hospital staffed with internists. An evacuation should ensue as soon as possible after alleviation of the acute phase.

5.3.5. Injuries from Psychochemical Agents

Psychochemical agents are divided into

psychomimetics and

dysregulators.

Typical representatives of the psychomimetics are LSD-25 and other derivatives of lysergic acid, piperidine glycolates and benzylates, e.g. Ditran and adrenaline derivatives. The characteristics of several psychomimetics are shown in Table 5.19.

Little is known concerning the dysregulators that can be used as chemical agents. They produce neuro-vegetative disorders, primarily responsible for bodily dysfunction. Prominent symptoms may be: indifference, apathy, narcolepsy, circulatory disorders, ataxia, disturbances of the temperature regulation, and paralysis [1].

5.3.5.1. The Clinical Picture

We may represent the typical course of psychomimetic intoxication by the example of LSD (a model psychosis). After approximately 30 minutes there occur

Table 5.19. Characteristics of Several Psychomimetics

Name	Toxic Features	Symptoms
LSD-25	PD 0.05 mg t_L 30-40 min W_{max} 2 hours W_e 8--10 hours	Intense hallucinations, space and time disorientation, reduced ability to concentrate
Ditran	PD 5--10 mg t_L up to 1 hour W_e 12--24 hours	Confusion, intense optic and acoustic hallucinations, weakness in speech and concentration, loss of contact with the surroundings
Adenochrome	PD 15 mg t_L 1 hour W_e up to 2 weeks	Extremely severe and persistent conditions resembling schizophrenia, hostile behavior, loss of contact with the surroundings, intense hallucinations

PD = psychosis-inducing dose
t_L = latent period
W_{max} = time before the climax of the intoxication
W_e = effective life

optical and/or auditory hallucinations, at times tactile, olfactory, and gustatory hallucinations. In the later course there is speech disorder and rambling speech. Intense excitement may alternate with deep depression and apathy. There is often persecution complex, groundless anxiety, and motor disturbances, as well as space and time disorientation.

There is almost always a pronounced delay, reduced precision, and decreased ability to concentrate in the solving of problems, so that effective actions are hardly possible.

The climax of the symptoms is reached after roughly 2 hours. For slight intoxication the effects abate after 6 to 8 hours, or 10 to 12 hours for heavy intoxication.

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5.3.5.2. Diagnostics

Damage from psychochemical agents is indicated by the simultaneous occurrence of the above symptoms in several or numerous persons.

The differential diagnosis should preclude psychoreactive disorders (no psychotic symptoms). The symptoms of an intoxication by dysregulators can only be distinguished from the particular situation.

5.3.5.3. Therapy

Usually no therapy is needed!

Important measures at the early stages of the medical evacuation are:

1. Assure the victims that the effects are only temporary (if they are still capable of understanding).

2. Take away their weapons to guard against emotional outbreaks.

3. Keep them in guarded rooms under constant observation.

In special cases of severe forms with sensory disorders in the foreground, a sedation with sinophenin (1 to 3 times, 0.1 to 0.2 g/day i.m./i.v.) may be necessary, as well as artificial respiration if there is a threat of respiratory paralysis.

Medical evacuation to provide qualified or specialized medical aid is restricted to special cases. The majority of patients are ready for combat after 12 to 18 hours.

5.3.6. Injuries From Irritants

Irritants are divided into

nose-throat irritants (phenarsazine chloride, diphenylarsinic chloride, diphenylarsinic cyanide),

eye irritants (chloraztophenone, bromacetone, ethyl bromoacetate) and

general irritants, which affect all the mucosa (O-chlorobenzylmalodinitrile).

The effects of the irritants proceed from an irritation of the sensitive nerve endings in the mucosa. The symptoms occur at once with no latent period. For the most part they rapidly abate after leaving the contaminated area. But toxic lung edema can occur at higher concentrations in the breathing air and for a longer exposure [1]. Table 5.20 provides a survey of the symptoms.

Table 5.20. Characteristic Effects of Irritant Agents

Symptoms	NATO Symbol for the CA	Irritation Threshold in mg/m^3	Limits of Toleration in mg/m^3
nose-throat irritation - intense secretion - coughing - sneezing - headache - labored breathing - nausea - vomiting	DM CS	0.1 0.5	0.4 5.0
eye irritation - intense flow of tears - convulsion of the eyelids - sensation of foreign objects	CN	0.3	4.5

Note: The limits of toleration refer to an exposure time of 1 minute.

5.4. Questions For Review

1. Describe the most important chemical agents.

2. What are the degrees of severity of nerve agent injury and by what criteria are they determined?

3. What dysfunctions are of special importance for nerve agent casualties?

4. What measures should be carried out and in what sequence for the self-help and mutual aid on the battlefield when nerve agents are used?

5. What is the basis for the medical classification of nerve agent casualties?

6. What are the degrees of severity of yperite casualties and by what criteria are they determined?

7. By what measures can the further effects of yperite on the organism be hindered?

8. What prophylactic or therapeutic measures are essential in the event of threatening or manifest lung edema caused by lung-damaging agents?

9. What is the significance of psychochemical agents?

5.5. Further Reading

[1] Handbuch für Militärmedizin, Bd. Innere Militärmedizin ("Manual for Military Medicine, Vol: Internal Military Medicine"), Berlin, 1973.

[2] Lehrbuch der Militärchemie, Bd. 1 ("A Textbook for Military Chemistry"), Vol. 1, Berlin, 1977.

[3] Lehrbuch der Militärchemie, Bd. 2 ("A Textbook for Military Chemistry"), Vol. 2, Berlin, 1977.

[4] Lohs, K., Synthetische Gifte ("Synthetic Poisons"), Berlin, 1974.