ARMY TM 8-285
NAVY NAVMED P-5041
AIR FORCE AFM 160-12

TREATMENT OF CHEMICAL AGENT CASUALTIES AND CONVENTIONAL MILITARY CHEMICAL INJURIES

DEPARTMENTS OF THE ARMY, THE NAVY, AND THE AIR FORCE
MAY 1974

CHAPTER 2

NERVE AGENTS

Section I. INTRODUCTION

2-1. General

a. The nerve agents are a group of highly toxic organic esters of phosphoric acid derivatives which have physiological effects resembling those of physostigmine and neostigmine (protigmine), but which are more potent and longer acting.

b. The nerve agents are among the deadliest chemical agents. They include the G-agents and V-agents. Examples of G-agents are tabun (GA), sarin (GB), and soman (GD). The standard V-agent is VX. (Details on VX are found in FM 3-10B.)

c. Nerve agents can be dispersed by artillery shell, mortar shell, rocket, aircraft spray, and landmine.

d. Several related but somewhat less toxic compounds have proved to be useful in medicine and agriculture. Diisopropyl fluorophosphate (DFP) has been used in the treatment of abdominal distention, urinary retention, and glaucoma. Tetraethyl phrophosphate (TEPP) and octamethyl pyrophosphoramide (OMPA) have proved valuable in the management of some patients with myasthenia gravis. Parathion, hexaethyl tetraphosphate (HETP), TEPP, and malathion as well as carbamates have been used as insecticides. Their widespread use has caused many accidental poisonings, some fatal. The symptoms and treatment of poisoning by these compounds are similar to those of poisoning by nerve agents.

2-2. Physical and Chemical Properties

The nerve agents are colorless to light brown liquids. Some are volatile. Others are relatively nonvolatile at ordinary temperatures. Except for GA, they are usually odorless. GA has a faint, sweet, fruity odor which is not intense or distinctive enough for detection under field conditions. In toxic amounts, aqueous solutions of nerve agents are tasteless. These agents react slowly with water and rapidly with strong alkali. They range from nonpersistent to persistent.

2-3. Absorption of and Protection Against Nerve Agents

a. The nerve agents may be absorbed through any body surface. When dispersed as a vapor, spray, or aerosol, they are absorbed through the skin, the respiratory tract, or the eyes. If absorption is great enough, local effects are followed by generalized systemic effects. Liquid nerve agents may be absorbed through the skin, the eyes, the gastrointestinal tract, and the membranes of the nose and the mouth. Local effects may occur, particularly after ocular absorption. Absorption of enough nerve agent by any route results in generalized systemic effects. The respiratory tract is the most rapid and efficient route of absorption.

b. The protective mask protects the eyes, the mouth, and the respiratory tract against nerve agent spray, vapor, and aerosol. Nerve agent vapor in ordinary field concentrations is absorbed through the skin very slowly, so that proper masking protects against the effects of these vapor concentrations. To prevent inhalation of an incapacitating or lethal dose, it is essential that the mask be donned at the first warning of the presence of nerve agent vapor.

c. Liquid nerve agent penetrates ordinary clothing or impregnated permeable clothing rapidly. However, significant absorption through the skin requires a period of minutes, so that contaminated clothing may be removed and liquid nerve agent on the skin washed off or blotted away. Prompt decontamination of the skin is imperative. Decontamination of nerve agent undertaken within 1 minute after contamination is twice as effective as it would be if delayed 5 minutes. Nerve agent on the skin can be removed effectively by use of the skin decontaminating pad from the M13 kit. Plain water will remove V-agents from the skin but less effectively than by use of the skin decontami-

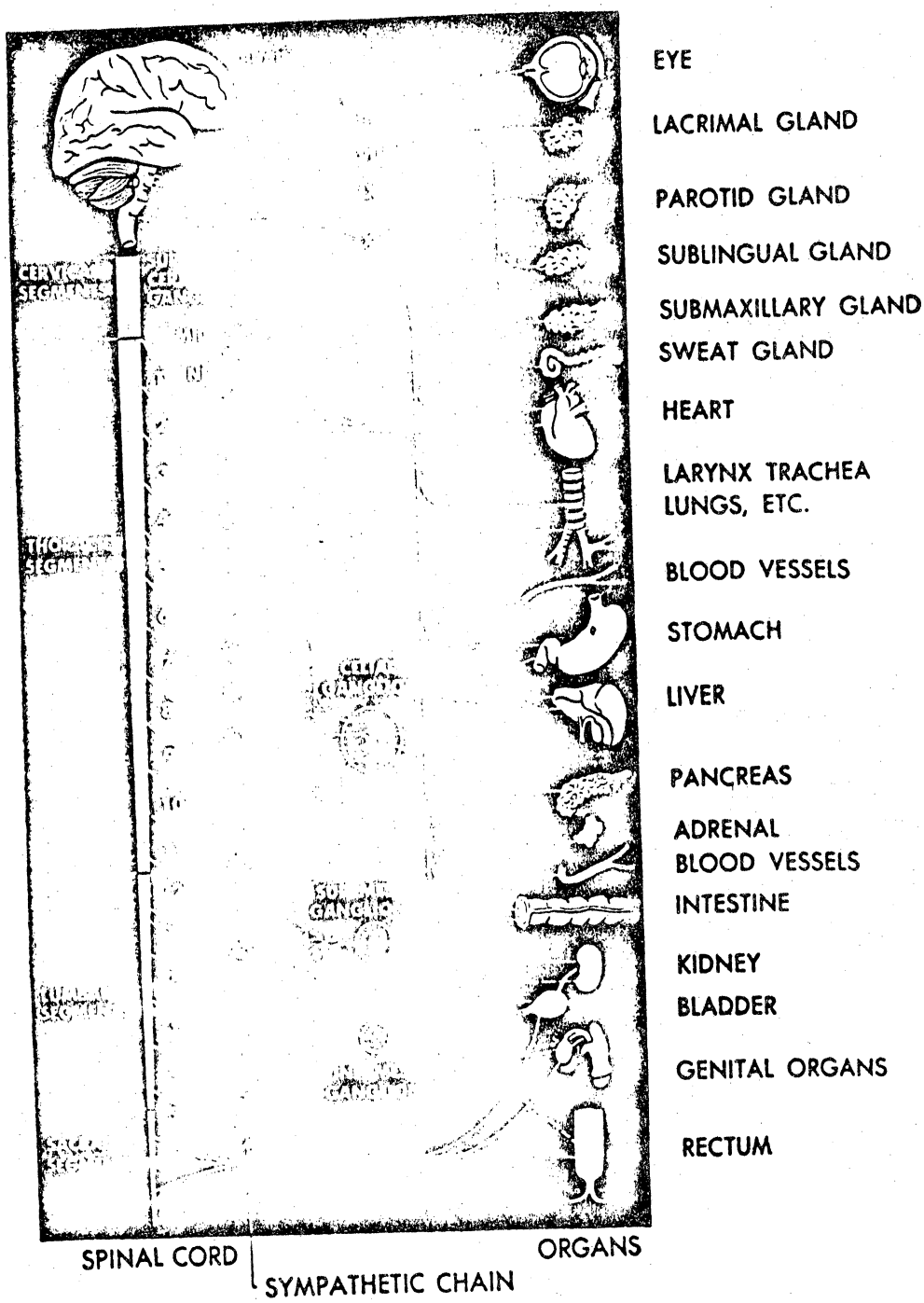


Figure 2–1. Autonomic nervous system.

in characteristic central nervous system symptoms (table 2–1). The inhibition of cholinesterase enzymes throughout the body by nerve agents is more or less irreversible, so that their effects are prolonged. Until the tissue cholinesterase enzymes are restored to normal activity, probably by very slow regeneration over a period of weeks or 2 or 3 months if the damage is severe, there is a period of increased susceptibility to the effects of another exposure to any nerve agent. During this period the effects of repeated exposures are cumulative.

b. Pathology. Aside from the decrease in the activity of cholinesterase anzymes throughout the body, which may be detected by chemical methods or by special staining, no specific lesions are detectable by ordinary gross or histological examination. At postmortem examination there is usually capillary dilatation, hyperemia, and

nating pad. (M5 ointment *will not* effectively neutralize G-agents.) Liquid nerve agent in the eye is absorbed faster than on the skin and is extremely dangerous, requiring immediate washing of the eye with copious amounts of water.

d. Impermeable protective clothing, individual protective covers, and impermeable protective gloves and aprons protect the skin against nerve agents in liquid form, aerosol, and vapor.

c. The nerve agents can poison food and water and contaminate their containers. Contamination by any material by liquid nerve agents will make it dangerous to handle without suitable protection or prior decontamination.

2–4. Effects of Nerve Agents

a. Mechanism of Action. The effects of the nerve agents (table 2–1) are due to their ability to inhibit cholinesterase enzymes throughout the body. Since the normal function of these enzymes to hydrolyze acetylcholine wherever it is liberated, such inhibition results in the accumulation of excessive concentrations of acetylcholine at its various sites of action. These include the endings of the parasympathetic nerves to the smooth muscle of the iris, ciliary body, bronchial tree, gastrointestinal tract, bladder, and blood vessels; to the secretory glands of the respiratory tract; and the cardiac muscle and the endings of sympathetic nerves to the sweat glands (fig 2–1). The accumulation of acetylcholine at these sites results in characteristic muscarine-like signs and symptoms (table 2–1). The accumulation of acetylcholine at the endings of motor nerves to voluntary muscles and in the autonomic ganglia results in nicotine-like signs and symptoms (table 2–1). Finally, the accumulation of excessive acetylcholine in the brain and spinal cord results

Table 2–1. Signs and Symptoms of Nerve Agent Poisoning

Site of Action	Signs and Symptoms
	Following Local Exposure
1. *Muscarine-like—*	
Pupils	Miosis, marked, usually maximal (pinpoint), sometimes unequal.
Ciliary body	Frontal headache, eye pain on focusing, slight dimness of vision, occasional nausea and vomiting.
Conjunctivae	Hyperemia.
Nasal mucous membranes	Rhinorrhea, hyperemia.
Bronchial tree	Tightness in chest, sometimes with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, cough.
	Following Systemic Absorption
Bronchial tree	Tightness in chest, with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, dyspnea, slight pain in chest, increased bronchial secretion, cough, pulmonary edema, cyanosis.
Gastrointestinal	Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness (cardiospasm) with "heartburn" and eructation, diarrhea, tenesmus, involuntary defecation.
Sweat glands	Increased sweating.
Salivary glands	Increased salivation.
Lacrimal glands	Increased lacrimation.
Heart	Slight bradycardia.
Pupils	Slight miosis, occasionally unequal, later maximal miosis (pinpoint).
Ciliary body	Blurring of vision.
Bladder	Frequency, involuntary micturition.
2. *Nicotine-like—*	
Striated muscle	Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness, including muscles of respiration, with dyspnea and cyanosis.
Sympathetic ganglia	Pallor, occasional elevation of blood pressure.
3. *Central Nervous System—*	Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, bursts of slow waves of elevated voltage in EEG, especially on overventilation, drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, ataxia, generalized weakness, coma, with absence of reflexes, Cheyne-Stokes respirations, convulsions, depression of respiratory and circulatory centers, with dyspnea cyanosis, and fall in blood pressure.

edema of the lungs, and there may be similar changes in the brain and the remaining organs.

c. Effects of Vapor. The lungs and the eyes absorb nerve agents rapidly. Local effects appear on the smooth muscle of the eye, resulting in miosis (contraction of the pupil), and on the smooth muscle and secretory glands of the bronchi, producing bronchial constriction and excessive secretion. If the concentration of vapor is great enough, the nerve agent is carried from the lungs throughout the circulatory system and widespread systemic effects may appear within 1 minute.

(1) *Local ocular and respiratory effects.* These effects begin within one to several minutes after exposure, before there is any evidence of systemic absorption. The earliest ocular effect, which follows minimal symptomatic exposure to vapor, is pupillary constriction. This is an invariable sign of ocular exposure to enough vapor to produce symptoms. It is also the last ocular manifestation to disappear. The pupillary constriction may be unequal. Within a few minutes after the onset of exposure, there also occurs redness of the eyes due to conjunctival hyperemia, and a sensation of pressure and heaviness in and behind the eyes. Vision is usually not grossly impaired, although there may be slight dimness especially in the peripheral fields and in dim or artificial light. The earliest effects on the respiratory tract, following minimal exposure, are a watery nasal discharge, nasal hyperemia, sensation of tightness in the chest, and occasionally prolonged wheezing expiration suggestive of bronchoconstriction or increased bronchial secretion. Exposure to about four times the minimal symptomatic dose results in extreme miosis; aching attributable to ciliary spasm in and behind eyes, especially on focusing; some difficulty of accommodation; and more severe rhinorrhea and frontal headache. The aching becomes worse when the subject tries to focus his eyes or looks at a bright light. Some twitching of the eyelids may occur. There is also intermittent more marked tightness in the chest and there may be some cough. Occasionally there is nausea and vomoting which, in the absence of systemic absorption, may be due to a reflex initiated by the ocular effects. These local effects may result in moderate discomfort and some loss of efficiency but they do not produce casualties. Following minimal symptomatic exposure, the miosis lasts from 24 to 72 hours and the rhinorrhea lasts a few hours. After exposure to four times the minimal symptomatic dose, miosis is well established within half an hour and soon becomes maximal. It remains marked during the first day after exposure and then diminishes gradually over 3 to 14 days. The conjunctival suffusion, eye pain, and headache may last from 2 to 15 days. The rhinorrhea usually lasts for several hours after minimal exposure and for about 1 day after more severe exposure. The respiratory symptoms are usually intermittent and of several hours duration after mild exposure, and may last for 1 or 2 days after more severe exposure.

(2) *Systemic effects.* At about six to eight times the minimal symptomatic exposure, enough nerve agent is absorbed to produce not only more severe local ocular and respiratory effects but also systemic effects. The resulting symptoms are severe enough to produce some casualties. At 15 to 20 times the minimal symptomatic exposure, the effects become alarming and all unprotected men will become casualties. The lethal exposure, without treatment, is about 30 to 50 times the minimal symptomatic exposure. The time interval between exposure and the onset of systemic effects depends upon the degree of exposure. Systemic symptoms may begin about a half hour after mild exposure, or within a few minutes to less than a minute after moderate or marked exposure. Following inhalation of a nerve agent, respiratory symptoms begin before the systemic effects, but other muscarine-like symptoms may develop accompanied by nicotine-like and central nervous system effects. High concentrations of vapor, especially of a nonpersistent nerve agent, are quite likely to occur in the field at and close downwind from the points of attack. The inhalation of one or two breaths of such concentrations may prove fatal without treatment. In these extreme exposures, mental confusion, incoordination, and initial convulsive movements of the extremities may occur within 30 seconds and progress to collapse and unconsciousness within a minute; and to flaccid paralysis and respiratory failure, which may supervene, within 2 to 3 minutes. Mild systemic effects generally last for several hours. Moderately severe symptoms remain at maximal severity for 6 to 24 hours and then diminish gradually in 1 to 6 days. During recovery, symptoms may recur intermittently, especially following exertion.

(*a*) *Muscarine-like effects.* The tightness in the chest, an early local symptom, increases as the nerve agent is absorbed into the syste-

mic circulation. After moderate or marked exposure, excessive bronchial secretion occurs and may become very profuse, causing coughing, airway obstruction, and respiratory distress. Audible wheezing may occur, with prolonged expiration and difficulty in moving air into and out of the lungs, due to the increased bronchial secretion or to bronchoconstriction, or both. Some pain may be referred to the lower thorax. Salivation increases. Bronchial secretion and salivation may be so profuse that watery secretions run out the sides of the mouth. If postural drainage or suction is not employed, airway obstruction may ensue. Laryngeal spasm may add to the respiratory difficulties. The subject may gasp for breath, froth at the mouth, and become cyanotic. If the upper airway becomes obstructed by secretions or laryngeal spasm, or if the bronchial tree becomes obstructed by secretions or, in some subjects, by bronchoconstriction, little ventilation may occur despite respiratory movements, and the subject may be seized with panic in his struggle for air. As his anoxemia and cyanosis increase, he may fall exhausted and unconscious. Following inhalation of nerve agent vapor, the respiratory manifestations predominate over the other muscarine-like effects; they are likely to be most severe in older subjects and in those with a history of respiratory disease, particularly bronchial asthma. However, if the exposure is not so overwhelming as to cause death within a few minutes, other muscarine-like effects appear. These include sweating, anorexia, nausea, and epigastric and substernal tightness (probably due to cardiospasm) with heartburn and eructation. If absorption of nerve agent has been great enough, whether due to a single exposure or to repeated smaller exposures, there may follow abdominal cramps, increased peristalsis, vomiting, diarrhea, tenesmus, increased lacrimation, urinary frequency, and occasional slight bradycardia. The subject becomes bathed in sweat and he may have involuntary defectation and urination.

(*b*) *Nicotine-like effects.* With the appearance of moderate muscarine-like systemic effects, the subject begins to have increased fatigability and mild generalized weakness which is increased by exertion. This is followed by involuntary muscular twitching, scattered muscular fasciculations, and occasional muscle cramps. The skin may be pale and blood pressure moderately elevated due to vasoconstriction, resulting from cholinergic stimulation of sympathetic ganglia and possibly from the release of epinephrine. If the exposure has been sufficiently marked, the fascicular twitchings, which usually appear first in the eyelids and in the facial and calf muscles, become generalized. Many rippling movements are seen under the skin and twitching movements appear in all parts of the body. This is followed by severe generalized muscular weakness, including the muscles of respiration. The respiratory movements become more labored, shallow, and rapid, then slow, and finally intermittent. The weakness of the respiratory muscles may become so profound that all respiratory efforts may cease, and the subject may die of anoxia within a few minutes unless artificial respiration is started promptly.

(*c*) *Central nervous system effects.* In mild exposures the systemic manifestations of nerve agent poisoning usually include tension, anxiety, jitteriness, restlessness, emotional lability, and giddiness. There may be insomnia or excessive dreaming, occasionally with nightmares. If the exposure is more marked, the following symptoms may be evident: headache, tremor, drowsiness, difficulty in concentrating, impairment of memory with slow recall of recent events, and slowing of reactions. In some subjects there is apathy, withdrawal, and depression. With the appearance of moderate symptoms, there occur abnormalities of the electroencephalogram, characterized by irregularities in rhythm, variations in potential, and intermittent bursts of abnormally slow waves of elevated voltage similar to those seen in patients with epilepsy. These abnormal waves become more marked after one or more minutes of hyperventilation which, if prolonged, may occasionally precipitate a generalized convulsion. If absorption of nerve agent has been great enough, the subject becomes confused and ataxic. He may have changes in speech, consisting of slurring, difficulty in forming words, and multiple repetition of the last syllable. He may then become comatose, reflexes may disappear, and respiration may become Cheyne-Stokes in character. Finally, generalized convulsions may ensue. With the appearance of severe central nervous system symptoms, depression of respiration, central in origin, may occur, adding to the respiratory embarrassment that may already be present. If the respiratory depression is severe, flaccid paralysis develops and death will occur due to anoxemia unless artificial respiration is started. Depression of the circulatory centers also may occur, resulting in a fall of blood pressure some time before death.

d. Effects of Liquid Nerve Agent.

(1) *Local effects.* The local ocular effects are similar to the effects of nerve agent vapor. If the concentration of nerve agent in the liquid reaching the eye is high, the effects will be instantaneous and marked; and, if the exposures of the two eyes are unequal, the local manifestations will be unequal. There is no immediate local inflammatory reaction such as may occur following ocular exposure to more irritating substances (for example, mustard). Following the ingestion of solutions containing a nerve agent, which is tasteless, the initial symptoms, which begin about 30 minutes after ingestion, are usually gastrointestinal. Following cutaneous exposure there is no local irritant change, but local sweating at and near the site of exposure and, at times, localized muscular twitching and fasciculation may occur. However, these may not be noticed so that the absorption through the skin may go undetected until systemic symptoms begin.

(2) *Systemic effects.* The sequence of symptoms varies with the route of exposure. While respiratory symptoms are generally the first to appear after inhalation of nerve agent vapor, gastrointestinal symptoms are usually the first after ingestion of solutions containing a nerve agent and generalized sweating may be the first after cutaneous exposure. Following comparable degrees of exposure, respiratory manifestations are most severe after inhalation, and gastrointestinal symptoms may be most severe after ingestion. Otherwise, the systemic manifestations are, in general, similar after any exposure and severe nerve agent poisoning by any route is similar to that described following inhalation. If local ocular exposure has not occurred, the ocular manifestations, including miosis, may be absent or mild until severe systemic manifestation occurs.

e. Time Course of Effects of Nerve Agents. See table 2-2.

f. Cumulative Effects of Repeated Exposure. Daily exposure to concentrations of a nerve agent insufficient to produce symptoms following a single exposure may result in the onset of symptoms after several days. Continued daily exposure may be followed by increasingly severe effects. After symptoms subside, increased susceptibility persists for one to several days. The degree of exposure required to produce recurrence of symptoms and the severity of these symptoms depend on the extent of and time intervals since the last exposure. Increased susceptibility is not limited to the particular nerve agent initially absorbed.

g. Cause of Death. In the absence of treatment, death is caused by anoxia resulting from airway obstruction, weakness of the muscles of respiration, and central depression of respiration. Airway obstruction is due to bronchial secretion, often associated with pulmonary edema, and some degree of bronchoconstriction. Respiration is shallow, labored, and rapid and the subject may gasp and struggle for air. Cyanosis increases. Finally, respiration becomes slow and then intermittent. Unconsciousness ensues. The blood pressure, which previously may have been elevated, falls. Cardiac rhythm may become irregular, probably as a result of anoxia, and death ensues. If respiration is maintained artificially and bronchial secretions removed by postural draininge and suction and diminished by the administration of atropine, the subject usually will survive several lethal doses of a nerve agent. However, if the exposure has been overwhelming, amounting to many times the lethal dose, death may occur despite treatment, probably as a result of central depression of the circulatory center, with peripheral vascular collapse and fall in blood pressure. When overwhelming doses of the agent are absorbed quickly, death occurs rapidly without orderly progression of symptoms.

2-5. Diagnosis of Nerve Agent Poisoning

a. Signs and Symptoms.

(1) The protective mask must be put on *at once* if any of the following are noticed:

(*a*) A feeling of tightness or constriction in the chest.

(*b*) Unexplained runny nose.

(*c*) Difficulty in breathing, either on inhaling or on exhaling.

(*d*) Small, pinpoint-size pupils seen in a mirror or in the eyes of individuals in the vicinity.

Note. On exposure to vapor or aerosol, the pupils will become pinpointed immediately. However, if the nerve agent is absorbed through the skin only or by ingestion of contaminated food or water, the pinpointing of the pupils will be delayed or even absent.

(*e*) A drawing, slightly painful sensation in the eyes, or unexplained dimness of vision, occurring with pinpoint pupils.

(2) Nerve agent poisoning can be identi-

Table 2–2. Time Course of Effects of Nerve Agents

	Types of Effects	Route of Absorption	Description of Effects	When Effects Appear After Exposure	Duration of Effects After Mild Exposure	Duration of Effects After Severe Exposure
Vapor	Local	Lungs	Rhinorrhea, nasal hyperemia, tightness in chest, wheezing.	One to several minutes	A few hours	1 to 2 days.
Vapor	Local	Eyes	Miosis, conjunctival hyperemia, eye pain, frontal headache.	One to several minutes	Miosis—24 hours	3 to 14 days. 2 to 5 days.
Vapor	Systemic	Lungs or eyes	Muscarine-like, nicotine-like, and central nervous system effects (see table 2–1).	Less than 1 minute to a few minutes after moderate or marked exposure; about 30 minutes after mild exposure.	Several hours	8 days.
Liquid agent	Local	Eyes	Same as vapor effects	Instantly	Similar to effects of vapor.	
Liquid agent	Local	Ingestion	Gastrointestinal (see table 2–1)	About 30 minutes after ingestion.	3 days	5 days.
Liquid agent	Local	Skin	Local sweating and muscular twitching.	3 minutes to 2 hours	3 days	5 days.
Liquid agent	Systemic	Lungs	See table 2–1	Several minutes		1 to 5 days.
Liquid agent	Systemic	Eyes	Same as for vapor	Several minutes		2 to 4 days.
Liquid agent	Systemic	Skin	Generalized sweating	15 minutes to 2 hours		2 to 5 days.
Liquid agent	Systemic	Ingestion	Gastrointestinal (see table 2–1)	15 minutes to 2 hours		3 to 5 days.

After lethal or near lethal exposures to nerve agents, the time to onset of symptoms and to maximal severity of symptoms is shorter; it may be extremely brief after overwhelming exposure. Following exposure to lethal concentrations, the time interval to death depends upon the degree, route of exposure, and the agent. For example, GB is most toxic by inhalation. GA is more toxic through the skin than GB. If inhaled VX acts as fast as GB and is more toxic. Exposure to lethal concentration of GB results in death 5 minutes after appearance of symptoms if untreated.

fied from the characteristic signs and symptoms. If exposure to vapor has occurred, the pupils will be very small, usually pinpoint. If exposure has been cutaneous, or has followed ingestion of a nerve agent, the pupils may be normal or, in the presence of severe systemic symptoms, slightly to moderately reduced in size. In this event, the other manifestations of nerve agent poisoning must be relied on to establish the diagnosis. No other chemical agent produces muscular twitching and fasciculations, rapidly developing pinpoint pupils, or the characteristic train of muscarine-like, nicotine-like, and central nervous system manifestations.

b. Effect of Atropine. The effect of atropine administration on mild and moderate cases of nerve agent poisoning may help confirm the diagnosis. Atropine injection alleviates most of the muscarine-like manifestations. It has less effect on the central nervous system symptoms and no effect on the nicotine-like symptoms. If the subject has absorbed little or no nerve agent, the administration of a single dose of 2 mg of atropine by any route produces symptoms of mild atropinization in most subjects and repetition of this dose within 1 or 2 hours produces moderate symptoms of atropinization in almost all subjects. In contrast, a casualty with moderate manifestations of nerve agent poisoning will not develop symptoms of atropinization after 2 mg of atropine. A casualty with marked manifestations of nerve agent poisoning may tolerate, indeed may require, considerably more than 4 mg of atropine.

c. Determination of Plasma and Red Blood Cell Cholinesterase Activity. This is not feasible under most field conditions. When this determination can be made, it is useful in establishing whether or not absorption of a nerve agent has occurred. In most instances, when the clinical picture of nerve agent poisoning of man or animals is clear, this confirmation will not be needed. Yet there are situations when the nerve agent has been absorbed slowly without producing symptoms, the blood cholinesterase has been reduced greatly, perhaps to zero, and another exposure soon afterward, even to a mild dose, would incapacitate or kill. When systemic symptoms may be very mild, or the subject is seen shortly after recovery, or persons or animals are seen only after death, confirmation of the impression of nerve agent poisoning may be desired. Then, the determination of the cholinesterase activity of the plasma, red blood cells, serum, or whole blood will be helpful.

(1) The signs and symptoms produced by nerve agents are due to inhibition of the cholinesterase enzymes of the nervous system, muscle, and secretory glands, and not to the coincident inhibition of the cholinesterase enzymes of the plasma and red blood cells. Determination of the latter, however, may be a guide of some value in detecting systemic absorption of a nerve agent and persistence of its effects. Following repeated exposure there is no predictable correlation between the onset of symptoms and the precise level of cholinesterase activity of the plasma or red cells, except that the level is depressed below normal. Cholinesterase activity of the red blood cells may be gradually depressed in near zero by repeated exposure over several days without systemic symptoms necessarily ensuing, or without any relation to the severity of the symptoms that occur. The red blood cell cholinesterase and, to a lesser extent, the plasma cholinesterase remain at a low level of activity long after symptoms disappear.

(2) No correlation exists between the plasma or red blood cell cholinesterase activity and the occurrence of local ocular or local respiratory effects. These may occur after local exposure without any depression of blood cholinesterase. In trying to evaluate the significance of determinations of plasma and red blood cell cholinesterase activity, one should keep in mind that the range of *normal activity* is fairly wide. In addition, plasma cholinesterase activity may be reduced moderately by many acute or chronic illnesses, particularly those affecting liver function. Red blood cell cholinesterase may be reduced also by relatively uncommon diseases of the blood such as pernicious anemia and leukemia.

2-6. Prevention of Poisoning

a. Since the respiratory tract absorbs nerve agent vapor very rapidly, the protective mask must be put on *immediately* when its presence in the air is suspected. Stop breathing until the mask is on, the facepiece cleared and checked. If the concentration of the nerve agent in the air is high, the inhalation of a few breaths may result in the absorption of enough to be incapacitating or even lethal. If the concentration in the air is low, a longer exposure may precede the onset of symptoms and the detection of nerve agent poisoning. Since the effects of a nerve agent are progressive and cumulative, the prevention of further absorption is particularly urgent once symptoms have begun.

Protective masks should be worn as long as approved test procedures indicate the presence of a nerve agent in the air and until the "all clear" signal is given.

b. Atropine should *not* be given for preventive purposes *before* contemplated exposure to a nerve agent, as this may increase respiratory absorption of the agent by inhibiting bronchoconstriction and bronchial secretion.

c. As liquid or vapor, nerve agents can poison food and water and may make other supplies or equipment dangerous to handle without protective mask and impermeable protecting gloves. Water and food supplies suspected of contamination or water from open sources in any area where nerve agent attack has occurred, should be examined by chemical tests and approved before consumption (app C and D). If necessary, water supplies should be decontaminated chemically (app C). Contaminated food may be reclaimed (app D) and materiel decontaminated (app B).

d. Approval of food and water for human consumption is the responsibility of the Medical Department.

Section II. TREATMENT OF NERVE AGENT POISONING

2–7. Principles of Treatment

The essential elements of treatment of nerve agent poisoning are—

a. Immediate donning of the protective mask and hood at the first indication of a nerve agent attack.

b. Immediate removal of any liquid contamination.

c. Administration of atropine as soon as any local or systemic symptoms due to a nerve agent are noted. As an adjunct to atropine, a therapeutic oxime, pralidoxime chloride (2-PAM Cl) may be administered.

d. Removal of bronchial secretions if they are obstructing the airway. Artificial airway with aspirator may be needed.

e. Administration of artificial respiration, if necessary, and oxygen, if necessary and available.

f. Administration of anticonvulsant medication if convulsions are not controlled by atropine and are endangering life.

2–8. Effects of Atropine

a. General. Atropine inhibits the action of the excess acetylcholine at all of its many sites of action except in most voluntary muscles and at the preganglionic synapses. As a result, atropine has a beneficial central nervous system effect on the respiratory depression, a marked inhibitory effect on the peripheral muscarine blockade, and no effect on the peripheral neuromuscular paralysis. Hence, atropine *alone* is suitable only to treat the mild to moderately severe cases which do not suffer failure of respiration. Used alone, it has little influence on the mortality rate in the potentially fatal apneic cases for which artificial respiration is many times more effective. However, the combination of adequate atropinization *plus* artificial respiration is several times as effective in saving life as artificial respiration alone.

b. Rate of Absorption. The effects of 2 mg of atropine sulfate or tartrate intramuscularly begin about 8 minutes after injection and are maximal in about 35 minutes. The effects of the same dose injected intravenously begin within 1 minute after injection and are maximal within 6 minutes. When this dose is given orally the effects begin about 20 minutes after administration and are maximal after about 50 minutes. While the rates of absorption of atropine administered in the same dose by these routes differ, the effects of the drug are, in general, the same after absorption has occurred.

c. Symptoms Produced by Atropine.

(1) The administration of a single dose of 2 mg (1 syrette or automatic injector) of atropine by any of the above routes *to an individual who has absorbed little or no nerve agent* produces mild symptoms, including dryness of the mouth and throat, with slight difficulty in swallowing. He may have a feeling of warmth, slight flushing, rapid pulse, some hesitancy of urination, and an occasional desire to belch. The pupils may be dilated slightly but react to light. In some individuals there may be mild drowsiness and slowness of memory and ability to recall. The recipient may have the feeling that his body movements are slow and his near vision is blurred, particularly after the intravenous administration of atropine. Some individuals may be mild-

CHAPTER 4

BLISTER AGENTS (VESICANTS)

Section I. INTRODUCTION

4-1. General

a. Examples of blister agents (vesicants) are mustard (HD), nitrogen mustards (HN), lewisite (L) and other arsenicals, mixtures of mustards and arsenicals, and phosgene oxime (CX).

b. Vesicants act on the eyes, the lungs, and the skin, and burn and blister the skin or any other part of the body they touch. They damage the respiratory tract when inhaled and cause vomiting and diarrhea when absorbed. The nitrogen mustards and the arsenicals are the most dangerous in causing vomiting and diarrhea.

c. Some vesicants have a faint odor; others are odorless. They often have a more serious effect than is immediately apparent. Most vesicants are insidious in action and there is little or no pain at the time of exposure, except with lewisite and phosgene oxime, which cause immediate pain on contact. Thus, in some cases, signs of injury may not appear for several hours.

d. Vesicants poison food and water and make other supplies dangerous to handle.

e. Vesicants can be disseminated by artillery shell, mortar shell, rocket, aircraft spray, and bomb.

f. The severity of a chemical burn is directly related to the concentration of the agent and the duration of contact with the skin.

4-2. Self-Aid

a. Wear the protective mask, hood, and clothing whenever liquid or vaporized vesicant agents are known to be present.

b. Liquid vesicants in the eyes or on the skin require immediate decontamination procedures as outlined in paragraph 1-11.

4-3. Precautions in Receiving Casualties

a. Casualties contaminated with vesicants endanger unprotected attendants. Individuals in contact with these casualties should wear, at least, protective masks and impermeable aprons and gloves. Exposed areas of the skin should be covered with M5 Protective Ointment. The ointment will protect against liquid vesicants indefinitely unless it is wiped off, worn thin, or otherwise depleted.

b. Special precautions must be taken in receiving contaminated casualties to prevent injury to others. Contaminated casualties should be undressed outside the medical treatment facility to prevent vapor accumulation indoors. They should be kept separated from uncontaminated casualties until decontamination is complete. Contaminated litters, blankets, and equipment should be left outdoors. Decontamination is necessary for equipment and any vehicles, small craft, or aircraft that have been used to transport contaminated casualties. For details on decontamination, see appendix B.

4-4. Protective Devices

a. Any protective mask protects only the face, eyes, and respiratory tract. The mask protects against both liquid and vapor forms of vesicants.

b. Chemical protective clothing, boots treated with Vesicant Gas Resistant Leather Dressing, and M5 Protective Ointment help to prevent the vesicant from reaching the skin. They are effective against vapor, but large drops of liquid vesicant will exhaust the impregnite locally in the clothing, boots, or ointment and allow penetration to the skin.

4-5. Disposition of Casualties

See chapter 5 for disposition of casualties with blister agent burns.

Section II. MUSTARD (HD)

4-6. Properties

a. Physical. Mustard is an oily liquid ranging from colorless, when pure, to dark brown when plant-run. Mustard is heavier than water, but small droplets float on water surfaces and thus represent a special hazard in contaminated areas. It smells like garlic or horseradish. Mustard freezes at 57° F. (14° C.) and boils at 442° F. (228° C.). It is only slightly soluble in water, which gradually destroys it, but undissolved mustard may persist in water for long periods. It is most soluble in fats and oils and freely soluble in gasoline, kerosene, acetone, carbon tetrachloride, and alcohol. These solvents do not destroy mustard. Mustard disappears from contaminated ground or materials through evaporation or through hydrolysis. It can be destroyed rapidly by decontaminating chemicals or by boiling in water. The primary use of mustard is to cause delayed casualties by liquid and vapor effect on the skin and the eyes and by vapor effect through the respiratory system.

b. Persistence. The persistence of hazard from mustard vapor or liquid depends on the degree of contamination by the liquid, type of mustard, nature of the terrain and soil, type of munition used, and weather conditions. Mustard may persist much longer in wooded areas than in the open. Mustard persists two to five times longer in winter than in summer. The hazard from the vapor is many times greater under hot than under cool conditions. Standard chemical agent detector kits should be used to detect the presence of hazardous concentrations of mustard vapor in the field.

c. Cumulative Effect. Even very small repeated exposures to mustard are cumulative in effect. *For example,* repeated exposures to vapors from spilled mustard can kill or produce 100-percent disability by irritating the lungs and causing a chronic cough and pain in the chest.

4-7. Effects on the Eye

a. Pathology, Symptoms, and Prognosis (fig 4-1 and 4-2). In a single exposure, the eye is more vulnerable to mustard than either the respiratory tract or the skin. Conjunctivitis follows exposure of about 1 hour to a concentration barely perceptible by odor (0.001 mg per liter). This exposure does not affect the respiratory tract or the skin significantly. A latent period of 4 to 12 hours follows mild exposure, after which there is lacrimation and a sensation of grit in the eyes. The conjunctivae and the lids become red and edematous. Heavy exposure irritates the eye after 1 to 3 hours and produces some severe lesions. Mustard burns of the eye may be divided as follows:

(1) Mild conjunctivitis (75 percent of cases in World War I). Recovery, 1 to 2 weeks.

(2) Severe conjunctivitis with minimal corneal involvement (15 percent of cases). Blepharospasm and edema of lids and conjunctivae. Orange-peel roughening of the cornea. Recovery, 2 to 6 weeks.

(3) Mild corneal involvement (10 percent of cases). Areas of corneal erosion stain green with fluorescein dyes. Superficial corneal scarring and vascularization. Iritis. Temporary relapses. Convalescence, 2 to 3 months. Casualties of this type require hospital care.

(4) Severe corneal involvement (about 0.1 percent of cases). Ischemic necrosis of conjunctivae. Dense corneal opacification with deep ulceration and vascularization. Convalescence, several months. Predisposition to late relapses.

b. Treatment.

(1) *Self-aid.*

(*a*) The risk of leaving liquid vesicant in the eye is so much greater than the risk from exposure of the eyes to vesicant vapors, during the short period of decontamination, that decontamination must be done despite the presence of vapor.

(*b*) Speed in decontaminating the eye is absolutely essential. The self-aid procedure is very effective for mustard within the first few seconds after exposure but is of little value after 2 minutes. Decontamination is done the same as for other vesicants (para 1-11*a*).

(2) *Treatment of mustard conjunctivitis.*

(*a*) Mild lesions require little treatment. Although the lesions become infected, a steroid antibiotic eye ointment such as Dexamesthasone phosphate-neomycin opthalmic ointment can be applied. Five percent boric acid ointment will provide lubrication and a mild antibacterial effect.

(*b*) More severe injuries will cause enough edema of the lids, photophobia, and blepharospasm to obstruct vision. This alarms the patient. To allay his fears, the lids may be gently forced open to assure him that he is not blind.

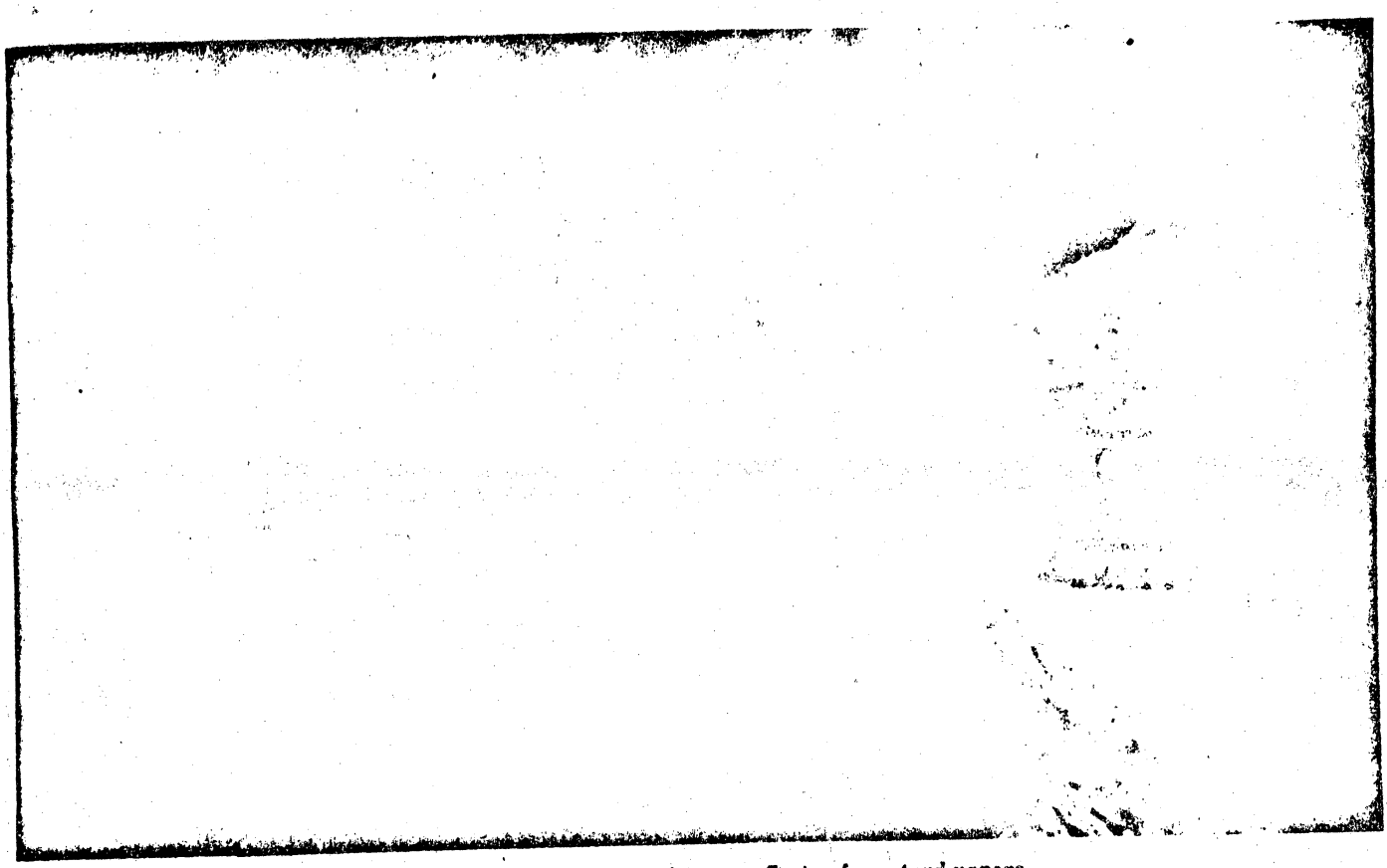
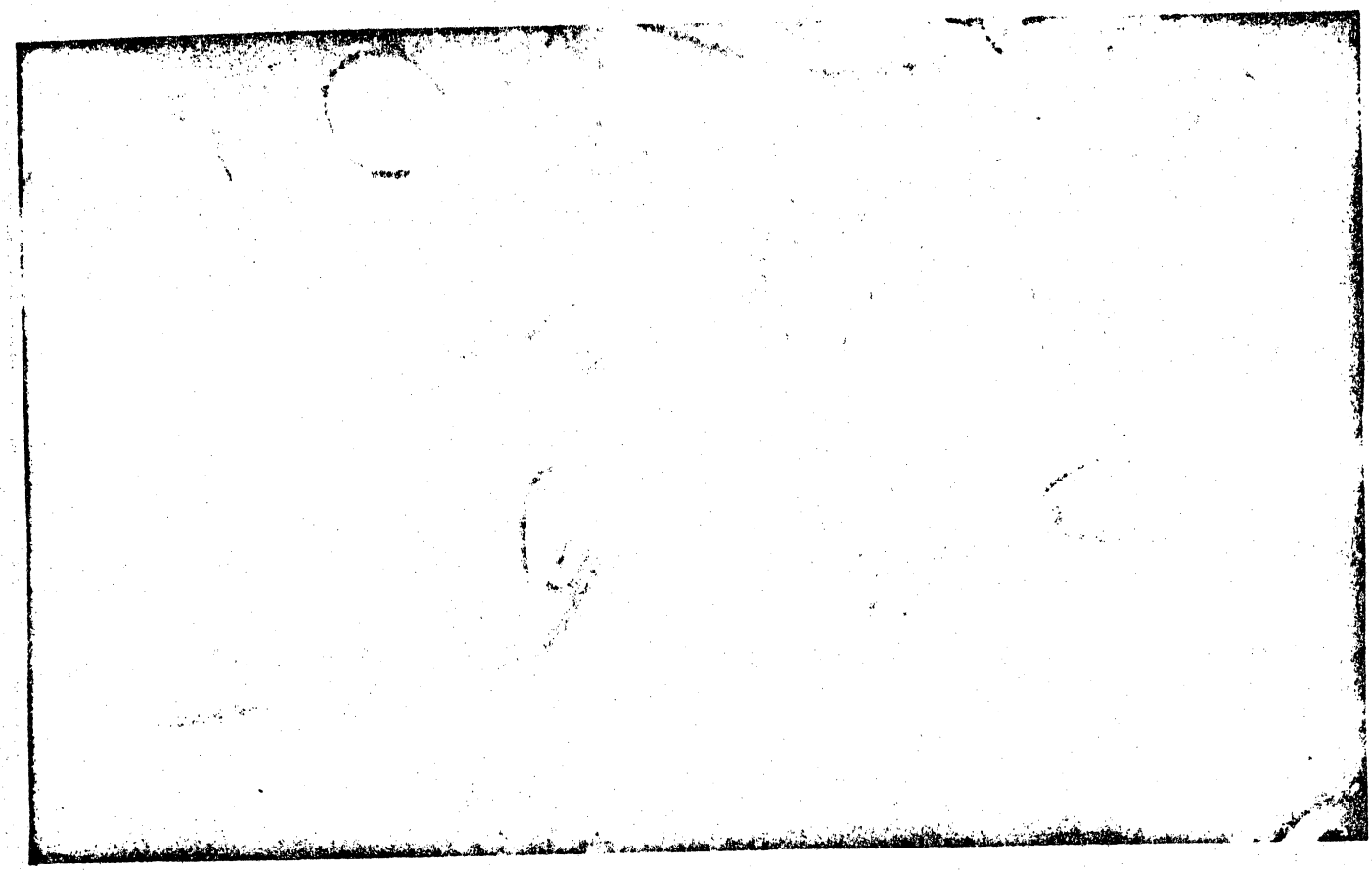


Figure 4-1. Casualty showing eye effects of mustard vapors.



Fiyure 4-2. Casualty with eye effects of mustard vapor.

The pain is controlled best by morphine and other systemic medication. Patients with severe photophobia and blepharospasm should have one drop of atropine sulfate solution (1 percent) instilled in the eye three times a day. To prevent infection, a few drops of 15-percent solution of

Sodium Sulfacetamide should be instilled very 4 hours. Other antibacterial ophthalmic preparations may be substituted for Sodium Sulfacetamide. The eye must not be bandaged or the lids allowed to stick together. Sealing of the lids may be prevented as described (*a*) above. The accumulation of secretions in the conjunctival sac or pressure on the eye, predisposes to corneal ulceration. To prevent complications, the patient should be evacuated to an ophthalmologist as soon as possible. When possible, the patient should be kept in a darkened room. Dark glasses or an eyeshade may be worn for photophobia.

(3) *Treatment of infected mustard burns of eye.* Secondary infection is a serious complication and increases the amount of permanent scarring of the cornea. If infection develops, initial treatment should be carried out with several drops of a 15-percent solution of Sodium Sulfacetamide every 2 hours. After appropriate cultures, specific antibacterial preparations may be applied. Irrigation should be gentle and employed only to remove accumulated exudate. Pain is controlled as described in (2)(*b*) above. Patients with secondary infection or other complications should be referred to an ophthalmologist. Local anesthesia should not be used unless necessary and then no more often than every 6 hours.

c. Classification of Eye Lesions. See paragraph 5–8.

4–8. Effect on the Skin

a. Pathology. The severity of the lesions and the rapidity with which they develop are greatly influenced by the weather conditions as well as by the degree of the exposure. Hot, humid weather strikingly increases the action of mustard. Even under temperate conditions, the warm, moist skin of the perineum, external genitalia, axillae, antecubital fossae, and neck are particularly susceptible.

(1) *Latent period.* Exposure is followed by a latent period which varies with the degree of exposure. It may be as short as an hour after liquid contamination, when the weather is hot and humid, or as long as several days after mild vapor exposures. With most vapor exposures in temperate weather, the latent periods is usually 6 to 12 hours.

(2) *Erythema.* Erythema gradually disappears and becomes brighter, resembling sunburn. Microscopically there is capillary hyperemia in the corium, accompanied by a variable degree of dermal edema. In severe burns the latter may be considerable so as to limit motion of the limb.

(3) *Vesication.* Except with mild vapor burns, erythema is followed by vesication. This is caused by progressive development of liquefaction necrosis of the cells in the lower layers of the epidermis. Exudation of tissue fluid into the spaces so formed results in an intraepidermal vesicle. Clinically multiple pinpoint lesions may arise within the erythematous skin; these enlarge and coalesce to form the typical blister, which is usually large, domed, thin-walled, superficial, translucent, yellowish, and surrounded by erythema. The blister fluid is clear, at first thin and straw-colored, later yellowish and tending to coagulate. The fluid may be quite irritating. Liquid contamination of the skin usually results in a ring of vesicles surrounding a gray-white area of skin which, although necrotic, does not vesicate.

(4) *Resorption.* If the blister does not rupture, resorption takes place in about a week. The roof forms a crust beneath which re-epidermization takes place. However, because of their thinness and tenseness, the blisters are fragile and usually break. If the roof becomes ragged, the burn may be considered an open wound. Such lesions may become secondarily infected.

(5) *Healing.* Since the damage to the corium usually is relatively superficial, healing occurs with little or no scar tissue formation except in very severe or infected burns.

(6) *Pigmentation.* Mustard burns usually are followed by a persistent brown pigmentation except at the site of actual vesication, where there may be a temporary depigmentation due to exfoliation of the pigmented layers of the skin.

(7) *Hypersensitivity.* Repeated burns may lead to hypersensitivity of the skin to mustard.

b. Symptoms and Prognosis.

(1) An outstanding characteristic of the action of mustard is its insidiousness. Exposures to mustard are not accompanied by symptoms, nor do any local manifestations occur until erythema develops. At this time there may be itching and mild burning. This pruritis may last several days and persist after healing. The blisters may be painful.

(2) Mustard erythema heals at about the same rate as sunburn of like severity. Areas of multiple pinpoint vesication usually heal, with desquamation, in 1 to 2 weeks. Mustard blisters

bacterial drugs may be given either locally or systemically as indicated.

4-9. Effect on the Respiratory Tract

a. Pathology.

(1) Inhalation of mustard vapor causes damage primarily to the laryngeal and tracheobronchial mucosa. The lesion develops slowly after exposure. A single exposure to a small amount of mustard vapor ordinarily does not produce significant injury. Repeated or chronic exposure to low concentrations of mustard vapor may lead to progressive pulmonary fibrosis, chronic bronchitis, and bronchiectasis. Moderate exposures result in hyperemia of the respiratory mucous membrane and necrosis of the lining epithelium. With severe exposures, the necrotizing action is accompanied by exudation resulting in a diphtheritic-like pseudomembrane, which may form a cast of the tracheobronchial tree.

(2) In the more severe cases, the pulmonary parenchyma shows congestion, mild patchy edema, moderate acute emphysema, and focal atelectasis. Altogether, these changes are insufficient to cause hypoxia, but, they are frequently complicated by bacterial infection of the lungs, which results in suppurative bronchitis and bronchopneumonia. The latter is responsible for almost all deaths following vapor exposures. The mortality from mustard among American troops in World War I, slightly more than 2 percent was due almost entirely to such complications following inhalation of vapor.

b. Symptoms and Prognosis. Respiratory tract lesions develop slowly and do not reach maximal severity for several days. Symptoms begin with hoarseness, which may progress to aphonia. A cough, worse at night, appears early and later becomes productive. Fever, dyspnea, and moist rales may develop. The incidence of bronchopneumonia is high. Convalescence is slow and cough may persist a month or longer. Milder symptoms, like hoarseness, last only 1 or 2 weeks.

c. Treatment of Respiratory Tract Injury Due to Mustard. Mild respiratory tract injury, with hoarseness and sore throat only, usually requires no treatment. Cough may be relieved by codeine. Laryngitis and tracheitis may be treated symptomatically with steam inhalations. If more severe respiratory tract injury is suspected hospitalization may be advisable. Secondary bronchopneumonia must be treated initially with broad spectrum antibiotics. After the isolation of specific organisms with their antibiotic sensitivities, antibiotic therapy can be limited to specific agents.

4-10. Systemic and Gastrointestinal Effects

a. Pathology.

(1) Ingestion of mustard produces vacuolation and nuclear swelling of the epithelial cells of the gastrointestinal tract, with eventual necrosis and desquamation with hemorrhage. Absorption of the mustard from the intestinal lumen results in the damage to blood-forming organs mentioned in (2) below.

(2) With ordinary skin or respiratory exposures to mustard, no apparent systemic lesions develop. With amounts approaching a lethal dose, injury to the hematopoietic tissues (bone marrow, lymph nodes, and spleen) may result. Such hematopoietic damage is reflected in the peripheral blood by leucopenia and thrombocytopenia. Lymphoid tissue is involved also, with consequent lymphocytopenia.

b. Symptoms.

(1) Ingestion of food or water contaminated by liquid mustard produces nausea and vomiting, pain, diarrhea, and prostration. Mustard vapor does not significantly contaminate food or water.

(2) Exposure of only the skin to mustard may cause systemic symptoms such as malaise, vomiting, and fever, coming on about the time of onset of the erythema. With severe exposures, particularly by extensive liquid contamination of the skin, these symptoms may be so marked as to result in prostration. Exceptional cases of severe systemic mustard poisoning may also present central nervous symptoms such as cerebral depression and parasympathomimetic effects such as bradycardia and cardiac irregularities. In animals, cerebral excitation and salivation have been observed, as well as bloody diarrhea with excessive loss of fluid and electrolytes. Hemoconcentration and shock may occur. It must be emphasized that such severe systemic effects do not occur with ordinary mustard exposures, but only if sufficient agent has been absorbed systemically.

c. Self-Protection. Never drink water which has been subjected to chemical attack until it has been certified as fit to drink by the Medical Department. Never eat foods which have been exposed to liquid vesicants, unless in sealed cans,

vary widely in healing time with both severity and anatomical location. In general, blisters of the face heal rapidly in 5 to 8 days. Blisters located in other areas may take slightly longer to heal, but if protected from infection will heal in 2 to 4 weeks. If cutaneous injury results in full-thickness coagulation necrosis, skin grafting may ultimately be necessary.

(3) Moderate contamination of mustard skin lesions with saprophytic bacteria, which cause no appreciable inflammatory reaction, does not seem to delay the healing of mustard burns. Active infection, with inflammation and purulent exudation, may increase the severity of the lesions and delay healing greatly.

c. Diagnosis of Skin Lesions Due to Mustard. Similar skin burns are produced by mustard and the nitrogen mustards. Mustard burns are also similar in appearance to those due to arsenical vesicants. Differentiation of mustard lesions from those produced by arsenicals is based upon—

(1) History of exposure to mustard.

(2) Absence of pain or discomfort at time of contamination (lewisite is irritating or painful immediately).

(3) Wide zone of erythema surrounding blisters (not prominent with arsenicals).

It should be remembered that vesicular lesions, much like mild mustard burns, may be produced in sensitive individuals by a variety of substances, notably plant poisons such as poison ivy or poison oak.

d. Decontamination of Casualties. Casualties having experienced liquid mustard contamination of the skin or clothing will seldom be received by the Medical Service in time to prevent subsequent blistering. Nevertheless, if erythema has not appeared, known or likely skin areas of contamination should be decontaminated as described in paragraph 1–11*b* and *c*. Cut away and discard hair contaminated with liquid mustard. Decontaminate the exposed scalp with decontaminating powder or protective ointment. If short of these substances, use bleach slurry (one part of bleach, chlorinated lime, in two to three parts of water) for decontamination of skin and hair. Wash off the bleach within 3 or 4 minutes to prevent skin irritation, taking care that none of the bleach washes into the eyes. If erythema of the skin has appeared, soap and water is the best decontaminant. Contaminated clothing should be removed promptly from casualties outside the treatment facility to prevent more severe burns and to lessen the vapor hazard to patients and attendants.

e. Treatment of Mustard Erythema. Mustard erythema in mild cases requires no treatment. If annoying itching is present, considerable relief may be obtained with Compound Calamine Lotion (containing 1 percent each of phenol and menthol). Severe erythema around the genitalia may become quite painful and associated weeping and maceration may ensue. Often treatment with exposure of the area is desirable and care must be taken so that secondary infection of tissue does not occur.

f. Treatment of Mustard Blister.

(1) All blisters should be opened and fluid drained with care, for the fluid itself may be irritating and cause secondary erythema and blister formation. Cleansing of the area with tap water or saline with the application of sterile petrolatum gauze when the areas are small is recommended. Dressings should be changed and the wound inspected every 3 or 4 days. Small blisters on the face are opened and best left uncovered. Large blisters may best be treated by open methods with the application of 10 percent Sulfamylon burn cream. Application of about one-eighth of an inch.

(2) If the dressing sticks to the wound, care will be necessary to avoid pulling off the top of the blister. It is good practice to trim the edges of adherent gauze, leave it in place, and put a fresh dressing over it. If the wound needs to be examined, the dressing may be soaked off with sterile saline.

g. Treatment of Denuded Areas.

(1) Contamination of mustard burns with saprophytic bacteria is common and unless careful wound care is given serious infection may result. If there is no inflammatory reaction, the treatment is the same as for uncontaminated burns.

(2) Wounds which become infected must be treated with appropriate antibiotics after adequate cultures have been obtained. The attending physician must evaluate the infection and make the appropriate decision regarding further care.

h. Specific Antibacterial Therapy. Routine wound inspection aids in the early detection and institution of appropriate therapy for any complicating bacterial infections. Appropriate anti-

until examined by the Medical Department and certified as safe to eat.

d. Treatment of Systemic Mustard Poisoning.

(1) In the treatment of systemic symptoms, atropine subcutaneously (0.4 to 0.8 mg; not the 2-mg syrette or automatic injector) may prove useful in reducing the gastrointestinal activity. General discomfort and restlessness may be treated with sedatives but may also be a manifestation of hypovolemic shock from severe systemic injury. In the exceptional cases of severe systemic poisoning with vomiting and diarrhea, leucopenia, hemoconcentration, and shock, every effort should be made to maintain an adequate nutritional status and to replace the loss of fluid and electrolytes.

(2) Injury due to the ingestion of liquid mustard in food or water may require morphine and atropine for relief of pain and shock therapy for collapse.

e. Prognosis.

(1) With ordinary field exposures to mustard vapor, it is not anticipated that deaths will occur from the systemic effects of the absorbed mustard. However, such deaths may occur from prolonged exposures to high concentions of mustard vapor or, in instances of extensive liquid contamination of the skin, where decontamination is neglected or unduly delayed. The occurrence of shock or pronounced leucopenia in these cases may be regarded as bad prognostic signs.

(2) Severe injury from ingestion of mustard is rare.

Section III. ARSENICAL VESICANTS

4-11. Properties

a. These agents are organic dichloroarsines. The main ones are phenyldichloroarsine and chlorovinyldichloroarsine (lewisite). Ethyldichloroarsine and methyldichloroarsine also have been used.

b. All arsenical vesicants are colorless to brown liquids, soluble in most organic solvents but poorly soluble in water. In general, they are more volatile than mustard and have fruity to geranium-like odors. They react rapidly with water to yield the corresponding solid arsinoxides, with concurrent loss of volatility and most of their vesicant properties. As liquids they gradually penetrate rubber and most impermeable fabrics.

c. They are much more dangerous as liquids than as vapors. The liquids will cause severe burns of the eyes and skin, while field concentrations of the vapors are unlikely to cause significant injuries. Immediate decontamination is required to remove the liquid agents in time to prevent severe burns, but decontamination is not required for vapor exposure unless pain is experienced. When inhaled, the vapors act as sternutators and may produce mild to moderate irritation of the upper respiratory tract. More significant respiratory injury is unlikely from ordinary field concentrations of vapor.

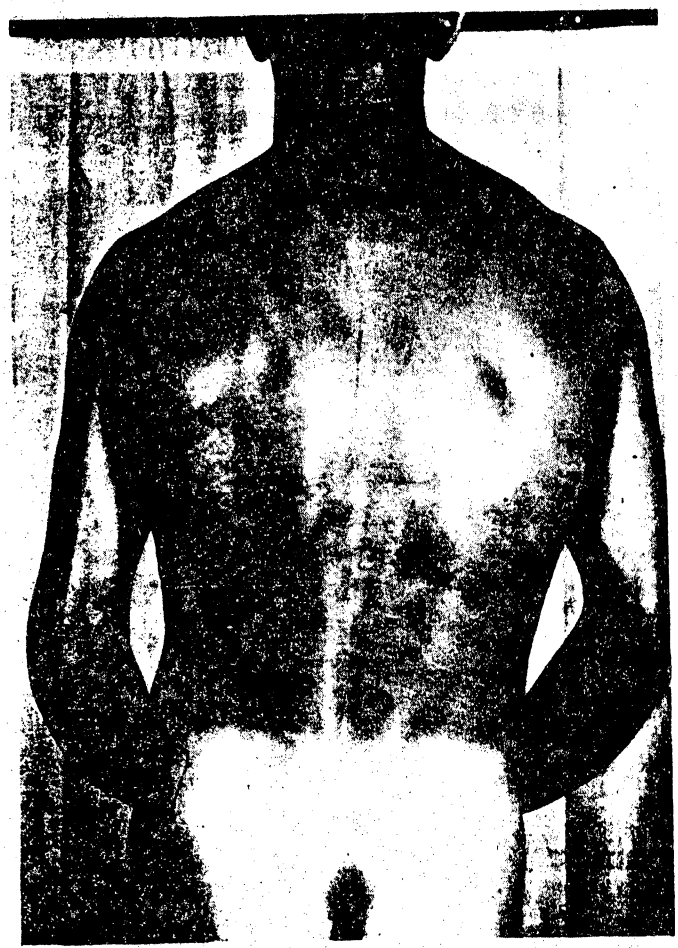
4-12. Effects on the Eye

a. Pathology, Symptoms, and Prognosis. Liquid arsenical vesicants cause severe damage to the eye. On contact, pain and blepharospasm occur instantly. Edema of the conjunctivae and lids follow rapidly and close the eye within an hour. Inflammation of the iris usually is evident by this time. After a few hours the edema of the lids begins to subside, while haziness of the cornea develops and iritis increases. The corneal injury, which varies with the severity of the exposure, may heal without residuals, induce pannus formation, or progress to massive necrosis. The iritis may subside without permanent impairment of vision, if the exposure was mild. After heavy exposure, hypopyon may ensue, terminating in necrosis, depigmentation of the iris, and synechia formation. Liquid arsenical vesicants instantly produce a gray scarring of the cornea, like an acid burn, at the point of contact. Necrosis and sloughing of both bulbar and palpebral conjunctivae may follow very heavy exposure. All injured eyes are susceptible to secondary infection. Mild conjunctivitis in man, due to arsenical vesicants, heals in a few days without specific treatment. Severe exposure may cause permanent injury or blindness.

b. Treatment. Treatment is largely symptomatic. In severe cases, the systemic use of morphine may be necessary. When the conjunctival edema subsides enough to permit ophthalmic examination, the cornea should be stained with fluorescein to detect erosions, and the iris should be examined for iritis. Atropine sulfate ointment should be instilled to obtain and maintain good mydriasis in all cases with corneal erosions, iritis, cyclitis, or with marked photophobia or miosis. Sodium Sulfacetamide solution may be used

5-3. World War II Experience

The effects of blister agent burns on the ability of troops to carry out usual military duties were investigated during World War II. In the United States, Canada, Great Britain, and Australia, volunteers ranging from recruits to troops with combat experience were exposed to blister agents. The degree of disability was evaluated on assault courses, route marches, or in simulated combat exercises lasting several days. These observations defined the limitations of casualty production according to type of lesion, and are the basis for this guidance. It should not be considered an adequate substitute for clinical observations of blister agent burns in the orientation of medical personnel.



Twenty-four hours before this photograph was made this man, wearing a protective mask and protective shorts, was exposed to a high dosage of mustard vapor in a warm climate.

Within 6 hours he became a casualty due to nausea and vomiting and generalized erythema involving the upper part of the limbs and trunk. The gastric symptoms persisted for 2 days.

During the ensuing 2 days the erythema continued to increase in severity, deepen in color, and break out in pinpoint vesication at scattered areas. The axillae and flexures of the elbows were especially affected.

Such cases are classified as casualties soon after exposure.

Figure 5-3. Casualty with generalized erythema and systemic intoxications.

Vesication of this extent is of casualty severity. Frequently a rise in temperature occurs. Nausea and vomiting may be present and add to the incapacitation caused by the burns.

These blisters were produced by heavy liquid mustard which contaminated the uniform covering the back. No decontamination measures were applied. The uniform was worn 4 hours after exposure to the agent.

Figure 5-4. Casualty with severe vesication.

5-4. Types of Blister Agent Burns

a. Two broad types of blister agent patients will not offer a problem in disposition.

(1) The first type consists of the totally disabled who are incapable of either offensive or defensive activity regardless of the urgency of the military situation. These individuals will be class as casualties and evacuated. Examples of injuries causing total disability and blindness, vesication of extensive areas of the trunk, or vesication of an entire limb.

(2) The second type consists of those who have sustained burns too minor to impair military effectiveness significantly. These individuals will be classed as noncasualties and returned to their units with or without treatment.

b. The intermediate types are partially disabled individuals who can perform only certain kinds of military duties but not others. The disposition of such cases is likely to constitute the main problem. This chapter is confined to typical injuries within this group. In disposing of these cases, the medical officer will be influenced by not only the severity of the lesions, but also the nature of the military situation and the general physical and mental condition of the individual under consideration.

5–5. Differentiation Among Injuries According to Agent

For simplicity, no effort is made here to differentiate among the several blister agents that may be used by an enemy. While there are differences between the typical mustard and arsenical vesicant lesions, it is not recommended that the medical officer in the field try to dispose of such cases separately. The diagnostic features of the various blister agent lesions and the therapy peculiar to each are described in chapter 4.

Widespread vesication caused by mustard extending from the shoulders to the buttocks. The skin between the vesicles was erythematous and edematous, and in many areas showed pinpoint vesication.
Severe discomfort, caused by stretching of the skin, accompanies lesions of this type. In addition, increase in temperature, nausea, and vomiting are common, especially in tropical climates.
Burns of this severity, uncomplicated by secondary infection, require hospital treatment for at least 3 to 4 weeks followed by a period of convalescence.

Figure 5–5. Casualty.

Section II. EYE INJURIES

5–6. Disposition

The correct disposition of personnel with eye lesions caused by blister agents is less of a problem to the medical officer than those with lesions involving the trunk and limbs. An accurate estimate of the degree of impairment of vision resulting from eye lesions can be made by simple inspection.

5–7. Sensitivity to Mustard

The eye is more sensitive and more vulnerable to the action of mustard than any other part of the body. About 86 percent of the mustard casualties in World War I had eye lesions to some degree. Exposure for 2 hours to a concentration of mustard, barely perceptible by odor, will produce eye lesions but may not affect the respiratory tract or the skin. There is no immediate symptomatic or local reaction to the absorbed agent. A latent period, that varies with the degree of exposure, proceeds the onset of symptoms. This period varies from 4 to 12 hours after mild exposure, and may be reduced to 1 to 3 hours after severe exposure.

5–8. Classification of Lesions of the Eyes

Eye lesions produced by mustard are divided into the following types:

a. Mild. Of all cases in World War I, 75 percent had mild burns of the eyes. The early symptoms include itching, lacrimation, and a sensation of grit in the eye, followed by burning and some-

APPENDIX C

DETECTION AND PURIFICATION OF CONTAMINATED WATER

C-1. General

Contamination of water supplies is to be expected areas attacked with chemical agents or in surface waters supplied by runoff from such areas. This contamination of water, whether intentional or inadvertent, may reach concentrations which can produce many casualties. By special methods of analysis, the presence of dangerous amounts of chemical agents can be determined. The purification of contaminated water is difficult, expensive, time-consuming, and requires chemicals and equipment not regularly issued to troops. Purification of a contaminated water supply should be undertaken only when an uncontaminated supply is not available.

C-2. Detection of Contaminated Water

The Chemical Agents Water Testing Kit is designed to provide a simple, rapid field test to determine whether a raw water supply is dangerously contaminated with chemical agents. Tests must be done on the raw water before chlorination since the presence of residual chlorine has an adverse effect on the accuracy of the kit. If the raw water is found to be free of chemical contamination it may be used after purification from bacterial contamination by chlorination or iodination. The Chemical Agents Water Testing Kit is so designed that negative results indicate the water is suitable for disinfection with chlorine or iodine and may thereafter be consumed by troops.

C-3. Procedure on Discovery of Contaminated Water

When a positive test is obtained with the Chemical Agents Water Testing Kit, the water will be considered contaminated and the following actions taken:

a. The commanding officer will be notified the water source is contaminated and unfit for drinking.

b. The commanding officer will establish the necessary safeguards to prevent personnel from drinking the contaminated water.

c. An alternative source of uncontaminated water should be sought and, if found, used. Consideration should be given to using ground water sources which are the least susceptible to chemical attacks and subsequent contamination.

d. Consideration should be given to moving to a different location or to transporting purified water to the area, if a source of uncontaminated water cannot be found.

e. Contaminated water should not be used until it has been decontaminated and disinfected.

f. Contamination discovered in otherwise suitable water should be reported promptly to the senior headquarters of the area for necessary action by the appropriate Medical Service personnel and the officer responsible for purification.

C-4. Purification of Contaminated Water

It potable water is not available, treat the contaminated water as follows:

a. Treatment of Large Volumes. Large volumes of contaminated water require additional equipment and supplies for treatment before purification. This pretreatment will be done by Engineer personnel as described in TM 5-700, or NAVFACSYSCOM TP-PL-2 or NAVDOCKS MO 218 or BUSHIPS Manual, chapter 9770.

b. Treatment in Lyster Bags.

(1) When the mobile water purification unit is not available, small volumes of water can be treated by using a Lyster bag and Set No. 1 (FSN 4610-256-4198, Filter Unit, Water Purification, Knapsack, Self-priming, Diaphragm Pump, ¼ gpm, Hand Operated). Persons handling contaminated water must wear adequate protective clothing.

(2) Fill the Lyster bag with the contaminated water (about 36 gallons).

CAMBRIDGE CITY COUNCIL

CITY HALL, CAMBRIDGE, MASSACHUSETTS 02139

(617) 498-9094

April 5, 1984

The Honorable; the City Council:

At the request of Councillor Francis H. Duehay, attached please find a booklet entitled "Treatment of Chemical Agent Casualties and Conventional Military Chemical Injuries" for your information.

Sincerely,

Susan Cruickshank

Susan Cruickshank
Assistant to the City Council

Attachment

S-242

Comm. from Susan Cruickshank, Asst. to the City Council, transmitting for informational purposes a copy of a booklet entitled "Treatment of Chemical Agent Casualties and Conventional Military Chemical Injuries".

In City Council,

April 9, 1984

4/9/84

Placed on File -