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TREATMENT OF CHEMICAL AGENT CASUALTIES AND CONVENTIONAL MILITARY CHEMICAL INJURIES

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PREFACE

Purpose

This manual serves as a guide and a reference for trained members of the Armed Forces Medical Services and other medically qualified personnel on the recognition and treatment of chemical agent casualties and conventional military chemical injuries. Additionally, this manual provides information on first aid (self-aid, buddy aid, and combat lifesaver (CLS) aid) for these casualties.

Scope

a. This manual—

(1) Classifies and describes chemical agents and other hazardous chemicals associated with military operations.

(2) Describes how to diagnose and treat conventional military chemical injuries (that is, riot control agents, smokes, incendiary agents, and other inhaled noxious industrial-type chemicals).

(3) Describes procedures for recognizing chemical casualties (app A).

(4) Describes procedures for first aid, medical treatment, and medical management of chemical casualties.

(5) Describes measures for handling contaminated clothing and equipment (app B).

(6) Describes medical management and treatment in chemical operations (app C).

(7) Describes procedures for decontamination of the eyes and skin (app D).

(8) Describes procedures for administering the Nerve Agent Antidotes, MARK I (NAAK) and convulsant antidote for nerve agent (CANA) (app E).

b. The manual is divided into two parts:

(1) Part One, Chemical Agent Casualties, covers the recognition and treatment of nerve agents, incapacitating agents, blister agents (vesicants), lung-damaging agents (choking agents), and blood agents (cyanogens) casualties.

(2) Part Two, Conventional Military Chemical Injuries, covers the recognition and treatment of injuries caused by riot control agents, smokes, incendiary agents, and other noxious industrial-type chemicals.

c. The material in this manual is applicable to both the conventional battlefield and the integrated environment of the battlefield. (For the purpose of this manual, the "integrated environment" is intended to mean warfare and/or contingency operations where nuclear, biological, and chemical (NBC) weapons/agents are being employed or have a high probability of being employed in addition to conventional weapons.)

Definitions

a. Chemical Agent. This is a chemical substance which, because of its physiological, psychological, or pharmacological effects, is intended for use in military operations to kill, seriously injure, or incapacitate humans (or animals) through its toxicological effects. Excluded are riot control agents, chemical herbicides, and smoke and flame materials. Chemical agents may be nerve agents, incapacitating agents, blister agents (vesicants), lung-damaging agents, blood agents, and vomiting agents.

b. Chemical Contamination. This is the deposition of chemical agents on personnel, clothing, equipment, structures, or areas. Chemical contamination mainly consists of liquid, solid particles, and vapor hazards. (Vapor hazards are probably the most prevalent means of contaminating the environment.)

c. Chemical Decontamination. This is the process of sufficiently reducing the hazard caused by chemical agents in order to allow the mission to be continued. Decontamination can be done by individual service members, unit decontamination teams, or chemical units. Generally used methods for skin decontamination include removal and/or chemical neutralization of agent(s) and clothing removal for medical examination; for equipment, the methods used are removal, destruction, covering, weathering, and chemical neutralization.

d. Persistence. Chemical agents maybe divided into two main categories: persistent and nonpersistent.

(1) Persistent agents, in a solid or liquid state, continue to present a hazard for considerable periods after delivery. They remain as a contact hazard and/or an inhalation hazard by very slowly vaporizing.

(2) Nonpersistent agents dissipate or vaporize rapidly after release and present an immediate short duration hazard. These agents are released as aerosols, gases, vapors, liquids, or solids.

e. Physical Characteristics. Chemical agents cover the whole spectrum of physical properties. Their physical state may be aerosol, gaseous, liquid, or solid under normal conditions. Their vapor pressure (the force

exerted by the vapor when in equilibrium with the liquid or solid at a given temperature) may be high or negligible. Their vapor density varies from slightly lighter than air to considerably heavier than air. Their range of odors varies from none to highly pungent. They may be soluble or insoluble in water, fats, or organic solvents. The physical characteristics may give an indication of the field behavior of the agents with regard to vapor hazard, persistency, decontamination methods required, and personal and subsistence protection required.

f. Conventional Military Chemicals. These are chemical substances used within the military for day-to-day operations as well as in combat. Included in this group are chemical herbicides, insecticides, and smoke and incendiary materials.

g. Riot Control Agent. This is a chemical which produces transient effects that disappear within minutes of removal from exposure and very rarely require medical treatment. Riot control agents are effective in quelling civil disturbances and, in some military operations, to preclude unnecessary loss of life.

h. Noxious Chemicals. Included in this category are gases such as carbon monoxide (CO), oxides of nitrogen, chlorine vapor, hydrogen sulfide, and ammonia.

For a more comprehensive list of definitions, see Glossary, Section II, located in the back of this manual.

Use of Trade Names/Trademarks and Metric Measurements

a. Use of trade names or trademarks in this publication is for illustrative purposes only. Their use does not constitute endorsement by the Department of Defense.

b. Metric measurements used throughout this publication are approximate equivalents of the customary units of measure. They are provided for the convenience of the users of the manual.

User Comments

Users of this manual are encouraged to submit recommendations to improve the publication. Comments should be keyed to the page, paragraph, and line(s) of the text where the change is recommended. Reasons should be given for each comment to ensure understanding and complete evaluation. The proponent for this publication is the U.S. Army Medical Department Center and School (AMEDDC&S). Comments should be submitted on DA Form 2028 (Recommended Changes to Publications and Blank Forms) and forwarded to: Commander, AMEDDC&S, ATTN: MCCS-FCD, Fort Sam Houston, Texas 78234-6175.

References

References listed should be consulted for details beyond the scope of this manual.

PART ONE

CHEMICAL AGENT CASUALTIES

CHAPTER 1

INTRODUCTION

1-1. The Threat of Chemical Warfare Agents to U.S. Forces

a. Chemical warfare (CW) agents (also referred to as chemical agents) remain a continuing threat to U.S. forces through the 1990s. Delivery may be accomplished by multiple means, causing extensive injury and contamination. Traditionally, threat commanders have regarded chemical weapons as a part of their conventional arsenal. The Chemical Weapons Convention (CWC), signed by 130 countries in January 1993, will take many years to fully implement and to verify the destruction of known chemical weapons stockpiles. Some countries with offensive chemical warfare programs, like North Korea and Iraq, have not signed the CWC. In spite of the CWC and other diplomatic efforts, chemical weapons will be available to threat forces in regions where U.S. forces may be deployed.

b. In addition to established threat areas, many countries have shown that chemical weapons are readily obtainable. The ease of obtaining these weapons greatly increases the complexity and extent of the total threat. For example, nonmilitary organophosphate insecticide factories may also be used to produce nerve agents.

c. Some threat forces are well prepared to conduct military operations in a chemically contaminated environment. Many threat combat vehicles (including tanks, reconnaissance vehicles, and aircraft) have organic NBC protective systems. These systems provide personnel protection from the effects of NBC contamination on the battlefield.

d. Chemical weapons are most effectively employed against untrained or unprotected targets. Civilian fixed sites (airfields, depots, cities, and ports) are especially vulnerable. These sites may be targeted as part of the plan to defeat U.S. force projection.

1-2. Military Employment of Chemical Warfare Agents

a. Chemical agents dispersed by modern weapons can be tactically used anywhere within the range of current delivery systems.

b. Chemical agents can be used in conjunction with other weapons systems or by themselves. Chemical agents may produce temporary incapacitating effects, serious injury, or death. Chemical agents also have the potential for use by saboteurs and terrorists in rear areas against key targets and civilian populations. The scope of CW is broad since it aims at groups rather than individuals and could be directed against civilian populations. Vapors of chemical agents may penetrate vehicles, ships, aircraft, fortifications, and buildings. Special design of such equipment and/or structures can prevent chemical agent penetration.

c. The presence or threat of CW operations can create psychological or physiological problems, adversely affect morale, and reduce military or civilian efficiency.

d. Chemical fires may be employed with smoke. Therefore, friendly forces must be prepared for chemical attacks when the enemy is employing smoke munitions or production equipment.

e. All service members must take every precaution against becoming chemical casualties. Each service member must apply the principles of first aid and decontamination contained in this manual to increase their chances for survival and recovery. Medical personnel must apply the principles of first aid, treatment, and decontamination contained in this manual to increase their and their patients' chances of survival.

1-3. Routes of Entry

Chemical agents may enter the body by several routes. When inhaled, gases, vapors, and aerosols may be absorbed by any part of the respiratory tract. Absorption may occur through the mucosa of the nose and the mouth and/or the alveoli of the lungs. Liquid droplets and solid particles can be absorbed by the surface of the skin, eyes, and mucous membranes. Chemical agents that contaminate food and drink can be absorbed through the gastrointestinal tract. Finally, wounds or abrasions are presumed to be more susceptible to absorption than the intact skin.

1-4. Classification of Chemical Agents

Chemical agents are classified by either their physiological action or their military use.

a. Physiological Action.

(1) Nerve agents (anticholinesterase) (such as Tabun (GA), Sarin (GB), Soman (GD), GF, and V-agent (VX)) inhibit the cholinesterase enzymes. The cholinesterase enzymes are responsible for the hydrolysis of acetylcholine, a chemical neurotransmitter. This inhibition creates an accumulation of acetylcholine at a cholinergic synapse that disrupts the normal transmission of nerve impulses. Cholinergic synapses are located–

- In the central nervous system (CNS).
- In the neuromuscular endplates of the peripheral voluntary nervous system.
- At the parasympathetic endings and sympathetic presynaptic ganglia of the autonomic nervous system.

(2) Blister agents (vesicants) include sulfur mustard (H/HD) and nitrogen mustard (HN), arsenical (lewisite (L)), and phosgene oxime (CX). Blister agents produce pain and injury to the eyes, reddening and blistering of the skin, and when inhaled, damage to the mucous membranes and respiratory tract. Mustard may produce major destruction of the epidermal layer of the skin.

(3) Lung-damaging agents (choking agents) include phosgene (CG), diphosgene (DP), chlorine, and chloropicrin (PS). These agents produce injury to the lungs and irritation of the eyes and the respiratory tract. They may also cause intractable pulmonary edema and predispose to secondary pneumonia.

(4) Blood agents (cyanogens) include hydrogen cyanide (AC) and cyanogen chloride (CK). These agents are transported by the blood to all body tissues where the agent blocks the oxidative processes, preventing tissue cells from utilizing oxygen. The CNS is especially affected and leads to cessation of respiration followed by cardiovascular collapse.

b. Military Use.

(1) Toxic chemical agents produce serious injury or death. They include nerve agents, blister agents, lung-damaging agents (choking agents), and blood agents.

(2) Incapacitating agents produce temporary physical or mental effects, or both.

1-5. Means of Delivery of Chemical Agents

Chemical agents can be dispersed by explosive shells, rockets, missiles, aircraft bombs, mines, and spray devices. Also, water supplies have the potential for contamination by either water-soluble or miscible liquids or solids. The means of delivery does not in itself help in identifying chemical agents. A spray or cloud delivered from an aircraft or by shells and bombs indicates a chemical attack maybe taking place. It must be remembered that vapors delivered from aircraft may not be visible; also, vapors and sprays may be hidden by atmospheric conditions.

1-6. Diagnosis of Injury from Chemical Agents

a. Odor. Some agents have odors which may aid in their detection and identification (table 1-1), but many are essentially odorless. (Table 1-1 is located in the back of this manual.) The odor of a chemical agent delivered by an explosive shell may be concealed by the odor of the burning explosive. Vomiting agents may be mixed with more lethal agents to induce vomiting and irritation of the respiratory tract. This mixture forces the affected individuals to break the seal of their masks in order to vomit, exposing them to the more toxic agents in the environment. Detection of a chemical agent odor is one indication for immediately putting on the mask and wearing it until the "all clear" signal is given. However, odor alone must not be relied on for detection or identification of a chemical agent. Some chemical agents are not perceptible by smell even on initial exposure. Continued exposure dulls the sense of smell. Even harmful concentrations of an odor-producing chemical agent may become imperceptible. Standard detection devices are the most reliable means of identifying a chemical agent, but users should remember that detection devices indicate concentrations in their immediate area only. They may not cover large areas and should not be the sole means on which to base conclusions on the presence or absence of chemical agents.

b. Signs and Symptoms. A chemical agent that has produced signs and symptoms in exposed personnel can usually be identified from all of the following.

(1) A brief history bringing out the symptoms that have occurred and their progression.

(2) Physical examination of the eyes (pupils, conjunctival, lids) and skin.

(3) Observation of respiration, color of mucous membranes, and general behavior. However, if a mixture of agents has been used, identification of the agents may not be possible. Signs and symptoms are summarized in table 1-1. Full descriptions of the signs and symptoms produced by specific chemical agents are given in the chapters that follow.

1-7. Protective Measures and Handling of Casualties

a. Depending on the theater of operations, guidance issued may dictate the assumption of a minimum mission-oriented protective posture (MOPP) level. However, MOPP 4 (consisting of wearing the protective overgarment, mask with hood, gloves, and overboots) will be assumed immediately—

- When the local alarm or command is given.

- When entering an area known to be or suspected of being contaminated with an NBC agent.
- During any motor march, once CW has been initiated.
- When casualties are being received from an area where chemical agents have reportedly been used. Appendix A provides additional information on recognizing chemical agent casualties.

If individuals find themselves alone without adequate guidance, they must mask IMMEDIATELY and assume MOPP 4 under any of the following conditions:

(1) Their position is hit by artillery, mortar fire, rocket fire, or by aircraft bombs, and chemical agents have been used or the threat of their use is significant.

(2) Their position is under attack by aircraft spray.

(3) Smoke or mist of an unknown source is present or approaching.

(4) A suspicious odor, liquid, or solid is present.

(5) A chemical or biological attack is suspected.

(6) They have one or more of the following signs/symptoms:

(a) An unexplained sudden runny nose.

(b) A feeling of choking or tightness in the chest or throat.

(c) Blurring of vision and difficulty in focusing the eyes on close objects.

(d) Irritation of the eyes (could be caused by the presence of several chemical agents).

(e) Unexplained difficulty in breathing or increased rate of breathing.

(f) Sudden feeling of depression.

(g) Anxiety or restlessness.

(h) Dizziness or light-headedness.

(i) Slurred speech.

(7) Unexplained laughter or unusual behavior noted in others.

(8) Buddies suddenly collapsing without evident cause.

(9) Animals or birds exhibiting unusual behavior and/or sudden unexplained death.

b. The mask should be worn until unmasking procedures indicate the air is free of chemical agent and the "all clear" signal is given (see FM 3-4 for unmasking procedures). If vomiting occurs, lift the mask momentarily and drain it (keep your eyes closed and hold your breath), then replace, clear, and seal the mask.

c. Chemically contaminated casualties present a hazard to unprotected personnel. Handlers must wear their individual protective equipment (IPE) while decontaminating these casualties. Casualty decontamination is conducted in an area equipped for casualty decontamination purposes (see para 1-11). The decontamination. area is located downwind (prevailing winds) of the medical treatment facility (MTF). Contaminated clothing and equipment are placed in plastic bags and removed to a designated dump site downwind from the MTF (see apps B and C).

d. When an MTF is expected to operate in a contaminated area, collective protective shelters (CPS) must be used (see app C).

e. Most chemical agents can poison food and water. Chemical contamination will make supplies and equipment dangerous to handle without protective equipment. Food and water packaged in sealed, airtight cans, bottles, or other impermeable containers can be decontaminated as described in FM 3-5 and FM 8-10-7. (Some plastics are permeable to chemical agents.) Exposed foods that are known to be contaminated or suspected of being contaminated should NOT be consumed unless approved by veterinary personnel. In the U.S. Army water purification is performed by quartermaster water purification units. Certification of water potability is the responsibility of medical personnel (preventive medicine personnel in the U.S. Army).

f. Military commanders, leaders, and medical personnel should be on the alert for the possibility of anxiety reactions (combat stress reactions (CSR)) among personnel during chemical agent attacks. All possible steps must be taken to prevent or control anxiety situations. Personnel in protective clothing are particularly susceptible to heat injury. Ambient temperature is considered when determining the degree of physical activity feasible in protective clothing. Wet bulb globe temperature (WBGT) index determinations (which indicate heat stress conditions in the environment) should be used with caution since the humidity within the protective ensemble will generally be higher than ambient humidity. At MOPP 4 add 10°F to the WBGT index. (See FM 3-4 for additional guidance on the degradation effects of the protective clothing.)

1-8. Chemical Agent Contamination Detection and Identification

Identification of chemical agents will greatly assist in the diagnosis and treatment of chemical injuries. The following are means of detecting and identifying chemical agent contamination:

a. Chemical agent detector paper or tape can be used to detect/identify liquid chemical agents.

(1) The VGH ABC-M8 Chemical Agent Detector Paper can be used to detect and identify liquid V- and G-type nerve agents and H-type blister agents. It does not detect chemical agent vapors. Some solvents and standard decontaminating (including the M258A1 kit) solutions cause false-positive reactions by the M8 paper.

(2) The M9E1 Chemical Agent Detector Paper (tape) (which can be worn on the uniform) detects the presence of liquid nerve agents (V and G) and blister agents (H/HD, HN, and L). The M9 paper does not distinguish between the types of agent involved—only that an agent or agents maybe present. Neither will it detect chemical agent vapors. Extremely high temperatures, scuffs, or certain types of organic liquids and decontaminating solution number 2 (DS2) cause false-positive reactions by the M9 paper.

b. Automatic chemical agent alarm systems and the chemical agent monitor (CAM) detect agent aerosol and vapor contamination consistent with their designed specifications and operational limitations.

c. Detector kits (such as the M256 Chemical Agent Detector Kit) detect and identify vapor concentrations of nerve, blister, and blood agents.

1-9. Medical Management

Medical management consists of those procedures for optimizing medical care to ensure the maximum return to duty (RTD) on the battlefield. This includes triage, basic survival treatment, decontamination, emergency forward treatment, evacuation, and continuing protection of chemical agent casualties. Although casualty decontamination is part of medical management, the physical decontamination of these personnel is the responsibility of the supported unit (app C).

1-10. Personal Decontamination

a. Eyes and Skin. Following contamination of the eyes or skin with vesicants or nerve agents, personal decontamination must be carried out IMMEDIATELY. These chemical agents are effective at very small concentrations. Within a very few minutes after exposure, decontamination is marginally effective for vesicants or nerve agents. Decontamination consists of either agent removal and/or neutralization. Decontamination after agent absorption occurs may serve little or no purpose. Service members will decontaminate themselves unless they are incapacitated. For those individuals who cannot decontaminate themselves, the nearest able person should assist them as the situation permits. Refer to appendix D for eye and skin decontamination procedures.

NOTE

In a cyanide or phosgene only environment, decontamination is not required.

(1) Decontaminate the eyes with copious amounts of water (see app D).

(2) Decontaminate the skin with the M291 or M258A 1 Skin Decontaminating Kit (see app C and app D (figs D-1 and D-2)).

(a) The M291 kit measures 4.4 by 9.3 by 0.7 inches and weighs 1.6 ounces. The kit can be folded to a measurement of 4.4 by 4.7 by 1.4 inches. Each kit contains six packets; enough to do three complete skin decontamination. Each packet contains an applicator bag filled with decontamination powder. The decontamination powder consists of 2.8 grams (gm) Ambergard™ XE-555 decontaminant resin. The pouch is discarded when all packets have been used. The kit will fit into the pocket on the outside rear of the M17 or M40 protective mask carrier or in the inside pocket of the carrier for the M24 and M25 series protective mask. The pouch can also be carried in a pocket of the battle dress uniform (BDU) or chemical protective overgarment.

WARNING

The M291 is for external use only. It may be irritating to the eyes. Keep the decontaminating powder out of the eyes. Use water to wash toxic agent out of eyes. You may also use a 0.5 percent chlorine solution to wash toxic agent out of cuts or wounds.

(b) The M258A1 kit measures 1¾ by 2⅝ by 4 inches and weighs 3.2 ounces. Each kit contains six packets: three DECON-1 packets and three DECON-2 packets. The DECON-1 packet contains a wipe pre-wetted with hydroxyethane 72 percent, phenol 10 percent, sodium hydroxide 5 percent, and ammonia 0.2 percent, and the remainder water. The DECON-2 packet contains a wipe impregnated with chloramine B and sealed glass ampules filled with hydroxyethane 45 percent, zinc chloride 5 percent, and the remainder water. The case fits into the pocket on the outside rear of the M17 or M40 protective mask carrier or in an inside pocket of the carrier for the M24 and M25 series protective mask. The case can also be attached to the web belt or on the D-ring of the protective mask carrier.

WARNING

The ingredients of the DECON-1 and DECON-2 packets of the M258A1 kit are poisonous and caustic and can permanently damage the eyes. The wipes must be kept out of the eyes, mouth, and open wounds.

b. Clothing and Equipment. When the M258A1 kit has been replaced with the M291 kit, the M258A1 will be used for decontamination of selected items of

individual clothing and equipment (there is insufficient capability to do more than emergency spot decontamination). The M258A1 kit is not used to decontaminate the protective overgarment. The protective overgarment does not require immediate decontamination since the charcoal layer is a chemical agent barrier; however, to enhance its protective capability, gross contamination should be removed. Exchange the protective overgarment as soon as the mission permits, using the procedures outlined in FM 3-5. The Decontamination Packet, Individual Equipment (DPIE), M295, is used to decontaminate individual equipment such as the weapon, helmet, and other individual gear. The M295 contains the same ingredients as the M291, except that the pads are much larger. However, the M295 is not Food and Drug Administration (FDA) approved for use on the skin.

1-11. Casualty Decontamination

Contaminated casualties entering the medical treatment system are decontaminated through a decentralized process. This is initially started through self-aid and buddy aid procedures. Later, units should further decontaminate the casualty before evacuation. Patient decontamination stations are established at the field MTF to decontaminate individuals as required (clothing removal and spot skin decontamination) prior to treatment and further evacuation. These stations are manned by nonmedical members of the supported units under medical supervision. Medical supervision is required to prevent further injury to the patient and to provide emergency medical treatment (EMT) during the decontamination process. There are insufficient medical personnel to both decontaminate and treat patients. Medical personnel will be fully employed providing treatment for the patients during and after decontamination by nonmedical personnel. Decontamination is accomplished as quickly as possible to facilitate medical treatment, prevent the patient from absorbing additional agent, and reduce the spread of chemical contamination. (For details on patient decontamination, see app C of FM 8-10-7 and chap 9 of FM 3-5.)

1-12. First Aid

First aid comprises either self-aid, buddy aid, or CLS.

a. Self-Aid. Self-aid consists of first-aid measures that service members can apply in helping themselves. These include individual decontamination and administration of chemical agent antidotes.

b. Buddy Aid. Buddy aid consists of emergency actions undertaken by individuals to restore or maintain vital body functions in a casualty. Exposure to chemical agents may produce effects which require immediate self-aid. However, mental confusion, muscular incoordination, physical collapse, unconsciousness, and cessation of breathing may occur so rapidly that the individual is incapable of providing self-aid. Therefore, the nearest individual may need to—

(1) Mask the casualty.
(2) Administer antidotes.
(3) Administer assisted ventilation, if required and if equipment is available.
(4) Decontaminate the casualty.
(5) Put selected items of protective clothing on the casualty to preclude further absorption of contamination through any exposed skin.
(6) Evacuate the casualty as soon as possible.

c. Combat Lifesaver. In addition to those actions taken as buddy aid, CLS aid also includes—

(1) Administering additional atropine.
(2) Administering additional CANA.
(3) Establishing an oropharyngeal airway.
(4) Starting intravenous infusions.

1-13. Medical Treatment

Medical treatment consists of those procedures undertaken to return soldiers to duty, to save life and limb, and to stabilize the patient for evacuation to the next level of medical care. Table 1-1 summarizes the treatment of chemical agent casualties. Specific chemical agent treatment procedures are described in the succeeding chapters.

Table 1-1. Summary of the Effects of Chemical Agents

Agent	Symbol	Odor	Mechanism of Action	Eyes			Nose and Throat		Respiratory Tract	Skin	GI Tract	Cardiovascular System	Genitourinary System	Central Nervous System	Other	Decontamination	Treatment	Agent
				Pupils	Conjunctival	Rest of Eye												
Tabun Sarin Soman GF VX	GA GB GD GF VX	None, or faint sweetishness, fruity or paint-like None	Anticholinesterase agents	Miosis	Redness	Pain (especially on focusing), dimness of vision, headache, lacrimation	Increased salivation, rhinorrhea		Tightness in chest, bronchoconstriction, occasional wheezing, increased bronchial secretion, cough, dyspnea substernal tightness	Sweating, pallor, then cyanosis	Salivation, anorexia, nausea, vomiting, abdominal cramps epigastric tightness, heartburn, eructation, diarrhea, tenesmus, involuntary defecation	Occasional early transient tachycardia and/or hypertension, followed by bradycardia, hypotension, and cardia arrhythmias	Frequent micturition, urinary incontinence	Apprehension, giddiness, insomnia, headache, drowsiness, difficulty concentrating, poor memory, confusion, slurred speech, ataxia, weakness, coma with areflexia, Cheyne-Stokes respiration, convulsions	Fasciculations, easy fatigue, cramps, weakness (including respiratory muscles), paralysis	Remove contaminated clothing. For skin use M291 Kit. For individual equipment use M295 Packet IAW established procedures	Pretreatment with pyridostigmine Post-exposure therapy: 1) Cholinergic blockade–atropine 2) Enzyme reactivation–oximes (2 PAM Cl) 3) Anticonvulsant–diazepam (CANA) 4) Assisted ventilation 5) Suction for respiratory secretions	GA GB GD GF VX
Mustard nitrogen mustard	H HD HN	Garlic or horseradish, irritating None or fishy, irritating	Vesicants. Bone marrow depressant. Alkylating agent, damages DNA		Redness, edema, irritation, gritty pain	Edema of lids, pain, blepharospasm, photophobia, lacrimation, corneal ulceration, and possibly scarring	Swelling, irritation, ulceration, discharge, occasional edema of larynx		Slowly developing irritation, hoarseness, aphonia, cough, tightness, dyspnea, rales. Pneumonia, fever, pulmonary edema in severe cases	No immediate signs. After minutes to hours, redness and burning. Several hours later blisters surrounded by redness and itching. Several days later necrosis, generally limited to epidermis. Delayed hyper- and hypo-pigmentation. Moist areas affected most. Risk of secondary infection	Pain, nausea, vomiting, diarrhea	Shock after severe exposure		Anxiety, depression	Late depression of bone marrow. Malaise and prostration	For liquid contamination of eyes, initially irrigate with copious amounts of water; then at the FMTF, with 2 sodium bicarbonate or saline eyewash. Remove contaminated clothing. For skin use M291 Kit. For individual equipment use M295 Packet	Eyes: antibiotics, cycloplegics and systemic analgesia Skin: local dressings and antibiotics for infection Antibiotics for respiratory infection. IV fluids	H HD HN
Lewisite and other arsenical vesicants	L	Fruity to geranium-like, very irritating	Vesicants. Arsenical poisons		Prompt redness, edema, irritation	Immediate burning sensation, iritis, corneal injury	Prompt irritation		Rapid irritation, hoarseness, aphonia, cough. Pneumonia, fever, pulmonary edema in severe cases, pleural effusion	Prompt burning. Redness within 30 minutes. Blisters on 1st or 2nd day. Pain worse and necrosis deeper than H	Diarrhea, nausea, vomiting, hepatic failure	Shock after severe exposure. Hemolytic anemia. Hemoconcentration	Renal failure	Anxiety, depression	Systemic arsenic poisoning	Like HD and HN	Like sulfur and nitrogen mustards. BAL in oil IM for systemic chelation, BAL ointment for eyes and skin	L
Mustard/ lewisite mixture	HL	Garlic-like	Like lewisite and mustard	Like HD, HN, and L													Like sulfur mustard, nitrogen mustard and lewisite	HL
Phosgene oxime	CX	Unpleasant and irritating	Powerful vesicant		Violently irritating, redness, edema	Lacrimation, corneal injury with blindness	Very irritating to mucous membranes		Rapid irritation, coughing. Later, pulmonary edema	Immediate severe irritation and intense pain. Within 1 minute the affected area turns white surrounded by erythema. Swollen within 1 hour, blisters after 24 hours, necrosis of skin. Long recovery (1 to 3 months)				Anxiety, depression		Wash with copious amounts of water or isotonic sodium bicarbonate	Apply dressings of sodium bicarbonate. Systemic analgesics. Treat as any other necrotic skin lesion	CX
Phosgene	CG	Green corn, grass, or new-mown hay	Lung-damaging agent		Irritation	Lacrimation (after respiratory symptoms)	Irritation		Coughing, choking, chest tightness on exposure. Latent period, then pulmonary edema, dyspnea, frothy sputum, rales, pneumonia, and fever	Possible cyanosis following pulmonary edema	Nausea, occasional vomiting (after respiratory symptoms)	Shock after severe exposure, hypertension and tachycardia		Anxiety, depression			Corticosteroids IV and by inhalation promptly may be lifesaving. Rest, oxygen, antibiotics	CG
Hydrogen cyanide	AC	Faint, bitter almonds	Interferes with oxygen utilization at cellular level						Deep respiration followed rapidly by dyspnea, gasping, then cessation of respiration	Initially pinker than usual; may change to cyanosis	Nausea	Profound hypertension		May have initial excitation; then depression, giddiness, headache, irrational behavior, ataxia, convulsions or coma			A. Drugs binding cyanide: 1) Methemoglobin formers; nitrites or DMAP 2) Scavengers; dicobalt edetate and hydroxocobalamin B. Provision of s-groups; thiosulfate C. Assisted ventilation D. Oxygen	AC
Cyanogen chloride	CK	Very irritating	Like hydrogen cyanide, lung irritant		Irritation	Lacrimation	Irritation		Irritation, cough, choking, dyspnea: pulmonary can be rapid	Like hydrogen cyanide (AC)							Like hydrogen cyanide and phosgene	CK
Vomiting agents	DM DA DC	Burning fireworks, very irritating	Local irritant, induces vomiting		Irritation	Lacrimation	Pain, rhinorrhea, tightness, sneezing	Tightness	Tightness and pain, uncontrollable coughing	Stinging (especially of face), occasional dermatitis	Salivation, nausea, vomiting			Severe headache, mental depression	May cause desire to remove protective mask		Wear mask in spite of symptoms. Spontaneous improvement	DM DA DC
Irritant agents	CN CA	Irritating	Local irritant		Redness, irritation	Pain, blepharospasm, profuse lacrimation, photophobia	Irritation, burning	Tightness, burning	Tightness and irritation if concentration is high	Stinging (especially of face), occasional dermatitis, may blister	Occasional vomiting			Headache		Wash eyes with copious amounts of water	Spontaneous improvement. Analgesic eye and nose drops if necessary	CN CA
	CS CR	Very irritating, pungent, pepper-like	Local irritant		Intense irritation	Pain, blepharospasm, profuse lacrimation, photophobia	Irritation, burning, tightness	Tightness, burning	Tightness in chest and difficulty breathing	Stinging, occasional dermatitis, may blister	Nausea and vomiting			Headache		Wash eyes with copious amounts of water	Symptoms disappear rapidly in fresh air	CS CR
Incapacitating agents	BZ	None	Anticholinergic	Mydriasis		Blurred vision	Extreme dryness	Extreme dryness		Dry, flushed	Constipation	Tachycardia, elevated blood pressure	Urgency, urinary retention	Headache, giddiness, drowsiness, disorientation, hallucinations and occasional maniacal behavior. Ataxia and/or lack of coordination		For contamination of skin, wash with soap and water	Restraint, cool environment. Physostigmine. Treatment may be required over several days	BZ
	LSD	None	Psychotomimetic	Mydriasis						Sweaty palms, cold extremities		Tachycardia		Mental excitation, poor concentration, tremor, indecisiveness, inability to act in a sustained or purposeful manner. Hallucinations	Pyrexia		Reassurance, restraint, prompt evacuation, diazepam	LSD

CHAPTER 2

NERVE AGENTS

Section I. INTRODUCTION

2-1. General

a. Nerve agents are a group of highly toxic organic esters of phosphoric acid derivatives. These agents have physiological effects (inhibition of cholinesterase) resembling those of physostigmine and pyridostigmine. However, they are more potent, longer-acting, and tend to be irreversible after a time which varies with the agent.

b. Nerve agents are among the deadliest of chemical agents and may produce rapid symptoms. They include the G- and V-agents. Examples of G-agents are Tabun (GA), Sarin (GB), Soman (GD), and GF. A V-agent is VX. In some countries, "V" agents are known as "A" agents. (Detailed descriptions of nerve agents are found in FM 3-9.)

c. Nerve agents can be dispersed by artillery shell, mortar shell, rocket, land mine, missile, aircraft spray, and aircraft bomb or bomblet.

d. Several related but somewhat less toxic compounds have proven to be useful in medicine and agriculture, as indicated below. The symptoms and treatment of poisoning by these compounds are similar to those of poisoning by nerve agents.

(1) Anticholinesterase agents have been used in the treatment of abdominal distention, urinary retention, and glaucoma.

(2) Many of the insecticides currently in use are organophosphates and are chemically related to nerve agents. Although beneficial for arthropod control, their widespread use has caused many accidental poisonings—some fatal. Organophosphate insecticides may have a slower and longer lasting effect as compared to CW organophosphates.

2-2. Physical and Chemical Properties

Nerve agents are colorless to light brown liquids. Some are volatile, while others are relatively nonvolatile at room temperature. Most nerve agents are essentially odorless; however, some have a faint fruity odor. In toxic amounts, aqueous solutions of nerve agents are tasteless. The G-agents tend to be nonpersistent, whereas the V-agents are persistent. However, thickened nonpersistent agents may present a hazard for an extended period of time. These agents are moderately soluble in water with slow hydrolysis; are highly soluble in lipids; and are rapidly inactivated by strong alkalies and chlorinating compounds (strong alkalies and chlorinating compounds are used for decontaminating equipment; in diluted formulas, chlorinating compounds are used for patient decontamination).

2-3. Absorption of and Protection Against Nerve Agents

a. Nerve agents may be absorbed through any body surface. When dispersed as a spray or aerosol, droplets can be absorbed through the skin, eyes, and respiratory tract. When dispersed as a vapor at expected field concentrations, the vapor is primarily absorbed through the respiratory tract. If enough agent is absorbed, local effects are followed by generalized systemic effects. The rapidity with which effects occur is directly related to the amount of agent absorbed in a given period of time. Liquid nerve agents may be absorbed through the skin, eyes, mouth, and membranes of the nose. Nerve agents may also be absorbed through the gastrointestinal tract when ingested with food or water. Local effects after skin exposure are localized sweating and/or muscular twitching. Local effects after vapor or liquid exposure to the eye include miosis and often conjunctival hyperemia. Local effects of liquid on the mucous membrane include twitching or contracting of the underlying muscle and glandular secretions. Absorption of a nerve agent by any route may result in generalized systemic effects. The respiratory tract (inhalation) is the most rapid and effective route of absorption.

b. The protective mask and hood protect the face and neck, eyes, mouth, and respiratory tract against nerve agent spray, vapor, and aerosol. Nerve agent vapor (in expected field concentrations) is absorbed through the skin very slowly, if at all, so proper masking may protect against the effects of low vapor concentrations. To prevent inhaling an incapacitating or lethal dose, hold your breath and put on your mask within 9 seconds at the first warning of a nerve agent presence.

c. Liquid nerve agents penetrate ordinary clothing rapidly. However, significant absorption through the skin requires a period of minutes. The effects may be reduced by quickly removing contaminated clothing and neutralizing liquid nerve agent on the skin (washed off, blotted, or wiped away). Prompt decontamination

of the skin is imperative. Decontamination of nerve agents on the skin within 1 minute after contamination is perhaps ten times more effective than it would be if delayed 5 minutes. A nerve agent on the skin can be removed effectively by using the M291 Skin Decontaminating Kit or the M258A1 Skin Decontamination Kit (app D). The M291 Skin Decontaminating Kit is replacing the M258A1. Upon receipt of the M291, discontinue using the M258A1 on the skin. Liquid nerve agent in the eye is absorbed faster than on the skin and is extremely dangerous; immediately irrigate the eye with copious amounts of water.

d. The chemical protective overgarment, patient protective wrap (PPW), impermeable protective gloves, and overboots protect the skin against nerve agents in liquid, aerosol, and vapor forms.

2-4. Effects of Nerve Agents

a. Mechanism of Action. The effects of nerve agents (table 2-1) are due to their ability to inhibit cholinesterase enzymes throughout the body. Since the normal function of these enzymes is to hydrolyze acetylcholine wherever it is released, such inhibition results in the accumulation of excessive concentrations of acetylcholine at its various sites of action. These include the endings of the autonomic nerves to the smooth muscle of the iris, ciliary body, bronchial tree, gastrointestinal tract, bladder, and blood vessels; to the salivary glands and secretory glands of the gastrointestinal tract and respiratory tract; and to the cardiac muscle and endings of sympathetic nerves to the sweat glands (fig 2-l). The accumulation of acetylcholine at these sites results in characteristic muscarinic signs and symptoms (table 2-1). The accumulation of acetylcholine at the endings of motor nerves to voluntary muscles and in some autonomic ganglia results in nicotinic signs and symptoms (table 2-l). Finally, the accumulation of excessive acetylcholine in the brain and spinal cord results in characteristic CNS symptoms (table 2-1). The inhibition of cholinesterase enzymes throughout the body by nerve agents may be irreversible and their effects prolonged; therefore, treatment should begin promptly before irreversibility occurs. Until the tissue cholinesterase enzymes are restored to normal activity, there is a period of increased susceptibility to the effects of another exposure to any nerve agent. This period of increased susceptibility occurs during the enzyme regeneration phase which could last from weeks to several months, depending on the severity of the initial exposure. During this period the effects of repeated exposures are cumulative.

b. Pathology. Aside from the decrease in the activity of cholinesterase enzymes throughout the body (which may be analyzed by laboratory methods), no specific lesions are detectable by ordinary gross examination. At postmortem examination there is usually capillary dilation, hyperemia, and edema of the lungs; there may be similar changes in the brain and the remaining organs. Neuropathologic changes have been reported in animals following severe intoxication.

c. *Effects of Vapor.* The lungs and the eyes absorb nerve agents rapidly. Changes occur in the smooth muscle of the eye, resulting in miosis (contraction of the pupil); also in the smooth muscle and secretory glands of the bronchi, producing bronchial constriction and excessive secretions in the upper and lower airways. In high vapor concentrations, the nerve agent is carried from the lungs throughout the circulatory system; widespread systemic effects may appear in less than 1 minute.

(1) *Local ocular effects.* These effects begin within seconds or minutes after exposure and before there is any evidence of systemic absorption. The earliest ocular effect which follows minimal symptomatic exposure to vapor is miosis. 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 different in each eye. 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 with heaviness in and behind the eyes. Usually vision is not grossly impaired, although there may be a slight dimness especially in the peripheral fields or when in dim or artificial light. Exposure to a level of a nerve agent vapor slightly above the minimal symptomatic dose results in miosis; pain in and behind the eyes attributable to ciliary spasm, especially on focusing; some difficulty of accommodation; and frontal headache. The pain becomes worse when the casualty tries to focus the eyes or looks at a bright light. Some twitching of the eyelids may occur. Occasionally there is nausea and vomiting 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 may not necessarily produce casualties. Following minimal symptomatic exposure, the miosis lasts from 24 to 72 hours. After exposure to at least the minimal symptomatic dose, miosis is well established within half an hour. Miosis remains marked during the first day after exposure and then diminishes gradually over 2 to 3 days after **MODERATE** exposure, but may persist for as long as 14 days after severe exposure. The conjunctival erythema, eye pain, and headache may last from 2 to 15 days depending on the dose.

(2) *Local respiratory effects.* Following minimal exposure, the earliest effects on the respiratory tract are watery nasal discharge, nasal hyperemia, sensation of tightness in the chest, and occasionally, prolonged wheezing expiration

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

SITE OF ACTION	SIGNS AND SYMPTOMS
	Following Local Exposure
1. **Muscarinic**	
Pupils	Miosis, marked, usually maximal (pinpoint), sometimes unequal.
Ciliary body	Frontal headache, eye pain on focusing, blurring of vision.
Nasal mucous membranes	Rhinorrhea, hyperemia.
Bronchial tree	Tightness in chest, bronchoconstriction, increased secretion, cough.
Gastrointestinal	Occasional nausea and vomiting.
	Following Systemic Absorption (depending on dose)
Bronchial tree	Tightness in chest, with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, dyspnea, pain in chest, increased bronchial secretion, cough, cyanosis, pulmonary edema.
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	Bradycardia.
Pupils	Miosis, occasionally unequal, later maximal miosis (pinpoint).
Ciliary body	Blurring of vision, headache.
Bladder	Frequency, involuntary micturition.
2. **Nicotinic**	
Striated muscle	Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness/flaccid paralysis (including muscles of respiration) with dyspnea and cyanosis.
Sympathetic ganglia	Pallor, transitory elevation of blood pressure followed by hypotension.
3. **Central Nervous System**	Immediate (Acute) Effects: Generalized weakness, depression of respiratory and circulatory centers with dyspnea, cyanosis, and hypotension, convulsions, loss of consciousness, and coma. Delayed (Chronic) Effects: 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 hyperventilation, drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, ataxia.

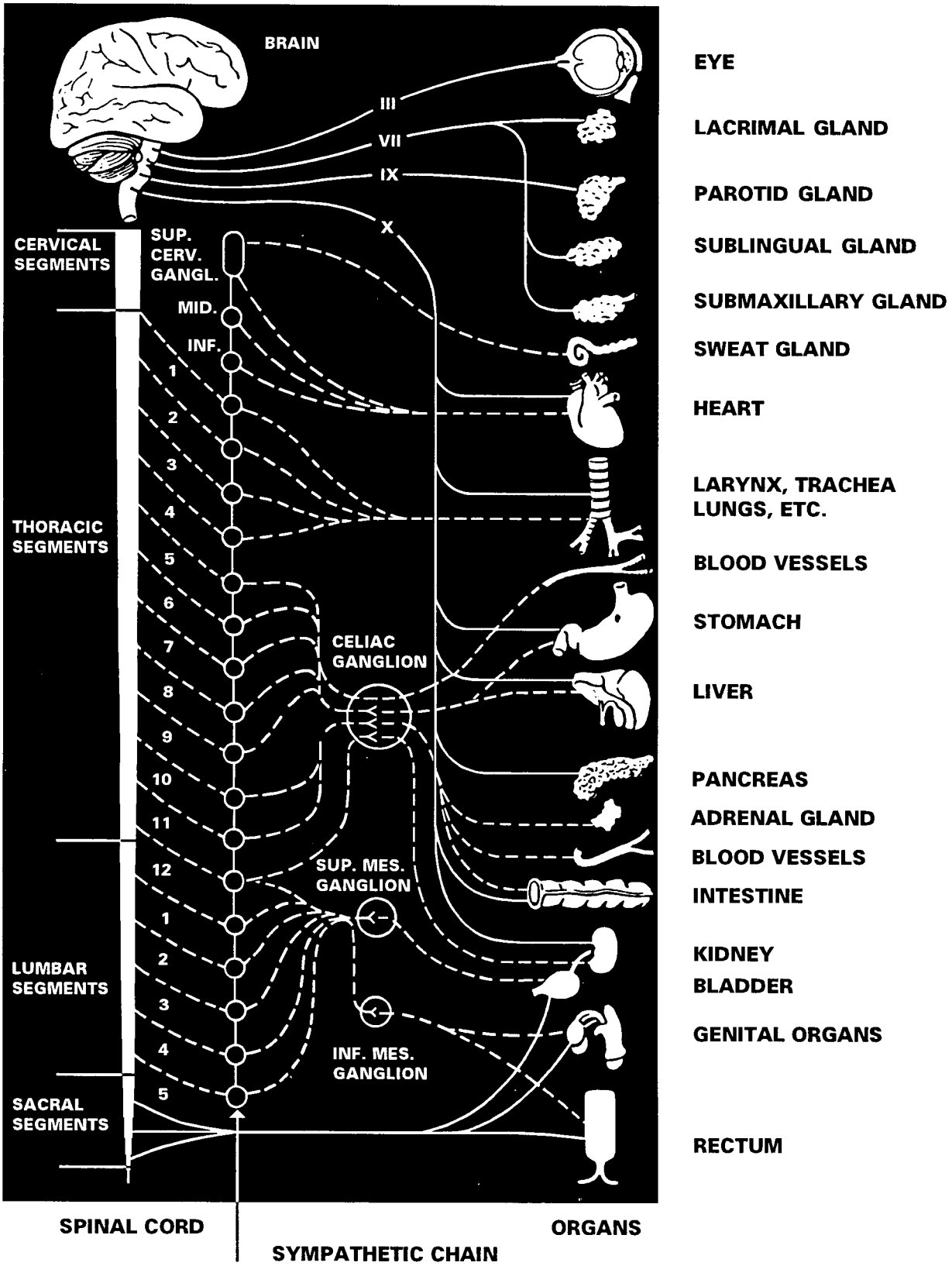


Figure 2-1. Autonomic nervous system.

suggestive of bronchoconstriction or increased bronchial secretion. 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 for several hours duration after **MILD** exposure; they may last for 1 or 2 days after more severe exposure.

(3) *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. 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 to nerve agent poisoning by any route. If local ocular exposure has not occurred, the ocular manifestations (including miosis) initially may be absent. The signs, symptoms, and their time course following exposure to nerve agent are given in table 2-2. The systemic effects may be considered to be nicotinic, muscarinic, or by any action at receptors within the CNS. The predominance of muscarinic, nicotinic, or CNS effects will influence the amount of atropine, oxime, or anticonvulsant which must be given as therapy. These effects will be considered separately.

(a) Muscarinic effects. The tightness in the chest is an early local symptom of respiratory exposure. This symptom progressively increases as the nerve agent is absorbed into the systemic circulation, whatever the route of exposure. After **MODERATE** or severe exposure, excessive bronchial and upper airway secretions occur 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 occur in the lower thorax and salivation increases. Bronchial secretion and salivation may be so profuse that watery secretions run out of the sides of the mouth. The secretions may be thick and tenacious. If postural drainage or suction is not employed, these secretions may add to the airway obstruction. Laryngeal spasm and collapse of the hypopharyngeal musculature may also obstruct the airway. The casualty may gasp for breath, froth at the mouth, and become cyanotic. If the upper airway becomes obstructed by secretions, laryngeal spasm, or hypopharyngeal musculature collapse, or if the bronchial tree becomes obstructed by secretions or bronchoconstriction, little ventilation may occur despite respiratory movements. As hypoxemia and cyanosis increase, the casualty will fall exhausted and become unconscious. Following inhalation of nerve agent vapor, the respiratory manifestations predominate over the other muscarinic effects; they are likely to be most severe. in older casualties 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 muscarinic effects appear. These include sweating, anorexia, nausea, and epigastric and substernal tightness with heartburn and eructation. If absorption of the nerve agent has been great enough (whether due to a single large exposure or to repeated smaller exposures), there may follow abdominal cramps, increased peristalsis, vomiting, diarrhea, tenesmus, increased lacrimation, and urinary frequency. Cardiovascular effects are occasional early bradycardia, transient tachycardia and/or hypertension followed by hypotension, and cardiac arrhythmias. The casualty perspires profusely, may have involuntary defecation and urination, and may go into cardiorespiratory arrest followed by death.

(b) Nicotinic effects. With the appearance of **MODERATE** muscarinic systemic effects, the casualty 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 due to vasoconstriction and blood pressure moderately elevated (transitory) together with tachycardia, resulting from epinephrine response to excess acetylcholine. If the exposure has been severe, the muscarinic cardiovascular symptoms will dominate and the fascicular twitching (which usually appear first in the eyelids and in the facial and calf muscles) becomes 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 they become slow and finally intermittent. Later, respiratory muscle weakness may become profound and contribute to respiratory depression. Central respiratory depression may be a major cause of respiratory failure.

(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 concentration, memory impairment with slow recall of recent events, and slowing of reactions. In some casualties, there is apathy, withdrawal, and depression. With the appearance of **MODERATE** symptoms, abnormalities of the electroencephalogram

occur, 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 1 or more minutes of hyperventilation which, if prolonged, may occasionally precipitate a generalized convulsion. If absorption of nerve agent has been great enough, the casualty becomes confused and ataxic. The casualty may have changes in speech (consisting of slurring, difficulty in forming words, and multiple repetition of the last syllable). The casualty 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 CNS symptoms, central respiratory depression will occur (adding to the respiratory embarrassment that may already be present) and may progress to respiratory arrest. However, after severe exposure, the casualty may lose consciousness and promptly convulse without other obvious symptoms. Death is usually due to respiratory arrest and anoxia. Prompt initiation of assisted ventilation may prevent death. Depression of the circulatory centers may also occur, resulting in a marked reduction in heart rate with a fall of blood pressure some time before death.

d. Effects of Liquid Nerve Agent.

(1) *Local ocular effects.* The local ocular effects are similar to the effects of nerve agent vapor. If the concentration of the liquid nerve agent contaminating the eye is high, the effects will be instantaneous and marked; and, if the exposure of the two eyes is unequal, the local manifestations may be unequal. Hyperemia may occur but there is no immediate local inflammatory reaction such as may occur following ocular exposure to more irritating substances (for example, lewisite).

(2) *Local skin effects.* Following cutaneous exposure, there is localized sweating at and near the site of exposure and localized muscular twitching and fasciculation. However, these may not be noticed causing the skin absorption to go undetected until systemic symptoms begin.

(3) *Local gastrointestinal effects.* Following the ingestion of substances containing a nerve agent (which is essentially tasteless), the initial symptoms include abdominal cramps, vomiting, and diarrhea.

(4) *Systemic effects.* The sequence of symptoms varies with the route of exposure. While respiratory symptoms are generally the first to appear after inhalation of a nerve agent vapor, gastrointestinal symptoms are usually the first after ingestion. 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 to nerve agent poisoning by any route. If local ocular exposure has not occurred, the ocular manifestations (including miosis) initially may be absent.

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 may persists for up to 3 months. The degree of exposure required to produce recurrence of symptoms and the severity of these symptoms depend on the dose received and the time interval 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 pharyngeal muscular collapse; upper airway and bronchial secretions; bronchial constriction and occasionally laryngospasm; and paralysis of the respiratory muscles. Respiration is shallow, labored, and rapid, and the casualty may gasp and struggle for air. Cyanosis increases. Finally, respiration becomes slow and then ceases resulting in unconsciousness. The blood pressure (which may have been transitorily elevated) falls. Cardiac rhythm may become irregular and death may ensue. The individual may survive several lethal doses of a nerve agent if assisted ventilation is initiated via cricothyroidotomy or endotracheal tube, if airway secretions are cleared by postural drainage and suction, and if secretions and bronchial constrictions are diminished by the vigorous administration of atropine. However, if the exposure has been overwhelming, amounting to many times the lethal dose, death may occur as a result of respiratory arrest and cardiac arrhythmia despite treatment. 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. Nerve agent poisoning may be identified from the characteristic signs and symptoms. If exposure to vapor has occurred, the pupils will be very small, usually pinpointed. If exposure has been cutaneous, or has followed ingestion of a nerve agent in contaminated food or water, 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 known chemical agent produces muscular twitching

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

AGENT DISPERSED AS	TYPES OF EFFECTS	ROUTE OF ABSORPTION	DESCRIPTION OF EFFECTS	WHEN EFFECTS APPEAR AFTER EXPOSURE	DURATION OF EFFECTS AFTER	
					MILD EXPOSURE	SEVERE EXPOSURE
Vapor	Local	Respiratory	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	2 to 3 days
Vapor	Systemic	Respiratory or eyes	Muscarinic, nicotinic, and central nervous system effects (see table 2-1)	Less than 1 minute to a few minutes after moderate or severe exposure; about 30 minutes after mild exposure	Several hours to a day	Acute effects: 2 to 3 days CNS effects: days to weeks
Liquid	Local	Eyes	Same as vapor effects	Instantly	Similar to effects of vapor	
Liquid	Local	Ingestion	Gastrointestinal (see table 2-1)	About 30 minutes after ingestion	Several hours to a day	2 to 5 days
Liquid	Local	Skin	Local sweating and muscular twitching	3 minutes to 2 hours	3 days	5 days
Liquid	Systemic	Bronchial tree	See table 2-1	Several minutes		1 to 5 days
Liquid	Systemic	Eyes	Same as for vapor	Several minutes		2 to 4 days
Liquid	Systemic	Skin	Generalized sweating	15 minutes to 2 hours		2 to 5 days
Liquid	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, the route of exposure, and the agent. If untreated, exposure to lethal concentrations of nerve agents can result in death 5 minutes after appearance of symptoms.

and fasciculations, rapidly developing pinpoint pupils, or the characteristic train of muscarinic, nicotinic, and CNS manifestations.

b. It is important that all service members know the following **MILD** and **SEVERE** signs and symptoms of nerve agent poisoning. Service members who have most or all of the symptoms listed below must **IMMEDIATELY** receive first aid (self-aid or buddy aid) (paras 2-11 *a* and *b*, respectively).

(1) **MILD** poisoning (self-aid). Casualties with **MILD** symptoms may experience most or all of the following:

(a) Unexplained runny nose.
(b) Unexplained sudden headache.
(c) Sudden drooling.
(d) Difficulty in seeing (dimness of vision and miosis).
(e) Tightness in the chest or difficulty in breathing.
(f) Wheezing and coughing.
(g) Localized sweating and muscular twitching in the area of the contaminated skin.
(h) Stomach cramps.
(i) Nausea with or without vomiting.
(j) Tachycardia followed by bradycardia.

(2) **SEVERE** *symptoms* (buddy aid). Casualties with **SEVERE** symptoms may experience most or all of the **MILD** symptoms, plus most or all of the following:

(a) Strange or confused behavior.
(b) Increased wheezing and increased dyspnea (difficulty in breathing).
(c) Severely pinpointed pupils.
(d) Red eyes with tearing.
(e) Vomiting.
(f) Severe muscular twitching and general weakness.
(g) Involuntary urination and defecation.
(h) Convulsions.
(i) Unconsciousness.
(j) Respiratory failure.
(k) Bradycardia.

Casualties with severe symptoms **WILL NOT** be able to treat themselves and MUST RECEIVE prompt buddy aid (para 2-11 *b),* CLS aid (paras 2-9 *e* and 2-12 *c),* and prompt follow-on medical treatment (paras 2-15 and 2-16) if they are to survive.

c. Casualties with **MODERATE** poisoning will experience an increase in the severity of most or all of the **MILD** symptoms. Especially prominent will be fatigue, weakness, and muscle fasciculations. The progress of symptoms from **MILD** to **MODERATE** indicates either inadequate treatment or continuing exposure to the agent.

Section II. PREVENTION AND TREATMENT OF NERVE AGENT POISONING

2-6. Essential Elements of Prevention and Treatment

The essential prevention and treatment elements of nerve agent poisoning are—

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

b. Administering the MARK I (para 2-11) as soon as any signs or symptoms are noted.

c. Administering the **CANA** to **MODERATE** to severely poisoned casualties (para 2-12).

NOTE

The U.S. Navy does not use the MARK I. Instead, the Navy issues three atropine and three pralidoxime chloride **(2 PAM C1)** auto injectors per person.

d. Removing or neutralizing any liquid contamination immediately.

e. Removing airway secretions if they are obstructing the airway. Airway suction may be needed.

f. Establishing a patent airway (for example, with a cricothyroidotomy or endotracheal tube) and administering assisted ventilation, if required. Oxygen is desired, if available.

2-7. Prevention of Poisoning

a. The respiratory tract absorbs nerve agent vapor very rapidly. The protective mask must be put on **IMMEDIATELY** when it is suspected that nerve agent vapor is present in the air. Hold the breath until the mask is on, cleared, and checked. If the nerve agent concentration in the air is high, a few breaths may result in the inhalation of enough nerve agent to be incapacitating or even lethal. When 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 urgent once symptoms have begun. Protective masks should be worn until the "all clear" signal is given.

b. **DO NOT** give nerve agent antidotes for preventive purposes **BEFORE** contemplated exposure to a nerve agent. To do so may enhance respiratory absorption of nerve agents by inhibiting bronchoconstriction and bronchial secretion. Atropine will degrade performance when taken in doses of more than 2 milligram (mg) without nerve agent exposure, especially when maximal visual acuity is required. Also, atropine will degrade an individual's ability to

perform duties in a hot environment. Atropine is rapidly used up in the treatment of nerve agent poisoning. A person incapacitated by nerve agent poisoning will likely remain incapacitated since atropine will not reverse all the signs and symptoms of poisoning, even in large doses.

c. Nerve agents (liquid or vapor) can poison food and water. For details on management and decontamination of food and water, see FM 8-10-7.

2-8. Effects of Nerve Agent Antidotes

a. General.

(1) *Atropine.* Atropine sulfate remains an essential drug in the treatment of nerve agent poisoning. It acts by blocking the effects of acetylcholine at muscarinic receptors and produces relief from many of the symptoms previously listed. If given in large doses, some therapeutic effects are also produced within the CNS although atropine does not readily penetrate the blood-brain barrier as does diazepam (para 2-8 *a* (3)), and central muscarinic receptors are thought not to be identical with those in the periphery. It is thought to counteract the respiratory depression in the medulla oblongata. Used alone, it has little influence on the mortality rate in the potentially fatal apneic cases for which assisted ventilation is many times more effective. However, the combination of adequate atropinization *plus* assisted ventilation is several times more effective in saving lives than assisted ventilation alone.

(2) *2 PM Cl.* **2 PAM Cl** is an oxime which increases the effectiveness of drug therapy in poisoning by some—but not all—cholinesterase inhibitors. Unlike atropine, **2 PAM Cl** acts by blocking the nerve agent inhibition of cholinesterase and/or reactivating the inhibited acetylcholinesterase clinically at muscarinic sites. Thus **2 PAM Cl** relieves the skeletal neuromuscular block, as well as reactivating the acetylcholinesterase clinically at muscarinic sites. The role of **2 PAM Cl** is to block and reverse the bonding of the nerve agent to the acetylcholinesterase. Oximes must be given early in the poisoning; after a short period of time (different for each type of nerve agent), they may no longer be effective.

NOTE

2 PAM Cl varies in its effectiveness against nerve agents. It is least effective against **GD.**

(3) *Diazepam.* Diazepam readily crosses the blood-brain barrier to block the effects of acetylcholine on the CNS, in contrast to the partial protection of atropine at best. Diazepam antagonizes the convulsive action of nerve agents. The addition of diazepam to the basic antidotes prevents or ameliorates convulsions in **MODERATE** to severe nerve agent poisoning.

b. Rate of Absorption.

(1) *Atropine.* A 2-mg intramuscular (IM) injection will reach peak effectiveness in 3 to 10 minutes, then blood concentrations will decline. If the system is unchallenged by a nerve agent, a 2-mg IM injection will cause atropine effects for several hours. In the presence of a nerve agent challenge, the effectiveness of atropine is markedly reduced and the duration of the agent is significantly shortened. More frequent doses of atropine will be required to achieve and maintain atropinization.

(2) *2 PAM Cl.* Depending on the degree of intoxication, a 600-mg IM will be effective in 6 to 8 minutes and will maintain peak effectiveness for 1 hour or more. If the system is unchallenged by a nerve agent, a 600-mg IM will remain in the circulatory system for several hours without apparent effect.

(3) *Diazepam.* A 10-mg IM injection in the thigh ordinarily produces significant plasma levels in 10 minutes; peak plasma concentrations are obtained in about 1 hour. The concentrations will then decline over a prolonged period. Rapid administration of diazepam by IM autoinjector after nerve agent exposure may more effectively prevent or ameliorate convulsions. **SEVERE** nerve agent toxicity may require multiple 10-mg doses given at about 10 minute intervals for a maximum of three (3) injections (a total of 30 mg diazepam) to control convulsions.

c. Symptoms Produced by the Antidotes.

(1) *Atropine.*

(a) The administration of a single dose of 2 mg (one autoinjector) of atropine to *an individual who has absorbed minimal or no nerve agent* produces **MILD** symptoms, including dryness of the skin, mouth, and throat, with slight difficulty in swallowing. The individual may have a feeling of warmth, slight flushing, rapid pulse, some hesitancy of urination, and an occasional desire to belch. The pupils may be slightly dilated but react to light. In some individuals, there may be **MILD** drowsiness and slowness of memory and ability to recall. Recipients of atropine may have the feeling that their movements are slow and their near vision is blurred. Some individuals may be mildly relaxed. These symptoms should not interfere with ordinary activity, except in the occasional individual who proves to be unusually reactive to the "sensation" effects of atropine (particularly the feeling of drowsiness). However, mental reaction may be slightly slowed down (for this reason, aviators must not fly an aircraft after taking atropine until cleared by the flight surgeon). If the administration of 2 mg of atropine is repeated within an hour without nerve agent challenge, the symptoms become **MODERATE.** In most of these individuals, there will be some CNS symptoms (such as drowsiness, fatigue, slowness of memory and ability to recall, the

feeling that body movements are slow, and blurred near vision); but they can continue ordinary activity with some loss of efficiency. Near vision may be impaired for as long as 24 hours. After repeated injections of atropine, heat-stressed individuals will become casualties *((b)* below). A third 2-mg dose of atropine (again without nerve agent challenge) administered within an hour will result in severe symptoms which will not permit ordinary activity—in fact, most individuals will be incapacitated. **SEVERE** incapacitating symptoms of overatropinization (nerve agent antidote poisoning) are a very dry mouth; swelling of the tongue and oral mucous membranes; difficulty in swallowing; thirst; hoarseness; dry and flushed skin; dilated pupils; blurred near vision; tachycardia (rapid pulse); urinary retention (in older individuals); constipation; slowing of mental and physical activity; restlessness; headache; disorientation; hallucinations; depression; increased drowsiness; extreme fatigue; rapid respiratory panting; and respiratory distress. Abnormal behavior may require restraint. The effects of atropine without nerve agent challenge are fairly prolonged, lasting 3 to 5 hours after one or two injections and 12 to 24 hours after marked overatropinization. Overatropinization may be incapacitating but presents little danger to life in a temperate environment for the nonheat-stressed individual. A single dose of 10 mg of atropine has been administered intravenously to *normal young adults* without endangering life—even in the absence of any prior absorption of a nerve agent—although it has produced very marked signs of overdose.

NOTE

While an unchallenged dose of atropine may allow individuals to continue normal duties, they must be closely monitored for possible heat injury. This is especially important when at **MOPP 4** and the individuals' ability to perspire is reduced due to atropine.

(b) In hot, desert, or tropical environments or in heat-stressed individuals, doses of atropine tolerated well in temperate climates may be seriously incapacitating by interference with the sweating mechanism. This can sharply reduce the combat effectiveness of troops who have suffered little or no exposure to a nerve agent. In hot climates or in heat-stressed individuals, one dose (2 mg) of atropine can reduce efficiency. Two doses (4 mg) will sharply reduce combat efficiency, and 6 mg will incapacitate troops for several hours. In hot, humid climates, individuals who have inadvertently taken an overdose of atropine and are exhibiting signs of atropine intoxication should have their activity restricted. In addition, these casualties must be kept as cool as possible for 6 to 8 hours after injection to avoid serious incapacitation. Usually, the casualties will recover fully in 24 hours or less from a significant overdose of atropine.

(c) Experience in chemical operations has shown that when troops become alarmed, some believe they have been exposed to more chemical agents than they actually have been. Hence, it is important that service members **NOT** give themselves more than one atropine injection (2 mg). Casualties who are able to ambulate and know who they are and where they are **WILL NOT** need any more atropine injections. If the symptoms do recur additional atropine, up to two more injections for a total of three (3), can be administered to these casualties. A service member must consult with a buddy to determine if he or she needs additional injections of atropine. If an individual's heart rate is above 90, breathing appears normal, bronchial secretions have diminished, and the skin is dry, the individual does not need anymore atropine at this time. Additional atropine is given by a **buddy** since casualties requiring more will be unable to administer additional injections to themselves. The additional administration of atropine to a service member with only **MILD** symptoms must be approached cautiously with at least 10 to 15 minutes elapsing between successive injections. If the signs of nerve agent poisoning (para 2-5) disappear, or if signs of atropinization, such as a heart rate above 90, diminished bronchial secretions, and dry skin, appear during one of these 10- to 15-minute periods, no further injections should be administered. These casualties should remain under observation without further injections of atropine unless signs of nerve agent intoxication reappear.

NOTE

Although one means of determining the casualty's need for additional atropine is the heart rate, assessing his or her respiratory effort is important in the evaluation. Labored breathing, including coughing, noisy breathing, wheezing, and gasping for air, indicates the need for administering additional atropine. When the heart rate is not obtainable, the need for additional atropine may be based on the degree of respiratory impairment. When adequate atropine has been given, labored breathing efforts will be relieved. This assessment must be performed without compromising the protective posture of **MOPP.**

(d) Patients with severe symptoms due to systemic absorption of a nerve agent have increased tolerance for atropine. Multiple doses maybe required before signs of atropinization appear, such as heart

rate above 90, diminished bronchial secretions, and dry skin. Large doses are required to ameliorate the muscarinic effects of nerve agent poisoning. The absence of increased tolerance for atropine indicates that nerve agent poisoning probably is not present or is **MILD.** In the presence of severe nerve agent poisoning, as much as 50 mg of atropine may be required for treatment in a 24-hour period. More than three injections of atropine will be administered only by the CLS or medical personnel.

(2) *2 PAM Cl.* **MILD** visual changes maybe a side effect of **2 PAM Cl.** After the administration of three injections of **2 PAM Cl,** generally no further oxime benefit is attained by additional injections of **2 PAM Cl.**

(3) *Diazepam.* The administration of a single dose of 10 mg (one autoinjector of **CANA)** to an individual who has absorbed minimal or no nerve agent produces significant performance decrements for about 2 to 5 hours. The individual will have impaired vision and decision-making functions over this time period. Overall alertness may be impaired. There could also be breathing difficulty. For this reason, casualties should be lying on their side until they are alert again. There may be transient irritation, as well as pain, at the injection sites.

Section III. SELF-AID, BUDDY AID, COMBAT LIFESAVER PROCEDURES, AND COMBAT MEDIC/CORPSMAN TREATMENT

2-9. Principles of Self-Aid and Buddy Aid

a. The protective mask and hood must be put on **IMMEDIATELY** at the first signs of a chemical attack. (The protective overgarment should have already been put on prior to the use of chemicals on the battlefield.) Stop breathing, put on your mask, clear and seal the mask, and resume breathing. Secure the mask hood. The mask and protective clothing are worn continually until the "all clear" signal is given.

b. **IMMEDIATELY** mask any casualty that does not have a mask on if the atmosphere is still contaminated.

c. The appearance of nerve agent poisoning symptoms calls for the immediate IM injection of the nerve agent antidote (paras 2-11 and 2-12). Since inhalation will be the most common route of exposure, the most likely initial symptom will be rhinorrhea (runny nose), then miosis (dim vision), followed by a feeling of tightness or constriction in the chest. After ocular (eyes) splash, there will be immediate miosis. After cutaneous (skin) splash, the initial systemic symptoms may be localized sweating and localized muscular twitching, followed by nausea and abdominal cramps. After ingestion, the first symptoms are likely to be nausea and vomiting. In any case, use the nerve agent antidotes as directed (paras 2-11 and 2- 12).

d. Promptly remove any liquid nerve agent on the skin, on the clothing, or in the eyes.

(1) If a liquid nerve agent gets on the skin, decontamination must be accomplished within 1 minute (see app D). Then continue the mission. Examine the contaminated area occasionally for local sweating and muscular twitching. If these occur, the nerve agent antidote should be administered. Combat duties should be continued, as systemic symptoms of nerve agent poisoning may not occur or may be **MILD** if the decontamination was done immediately and successfully.

(2) If a drop or splash of liquid nerve agent gets into the eye, instant action is necessary to avoid serious effects. Irrigate the eye immediately with water as described in appendix D. During the next minute, the pupil of the contaminated eye should be observed by a buddy. If the pupil rapidly gets smaller, a nerve agent antidote should be administered. If the pupil does not get smaller, the ocular contamination was not caused by a nerve agent and atropine is not needed.

e. If good relief is obtained from one set of MARK I injections and breathing is normal, carry on with combat duties. Dryness of the mouth is a good sign—it means enough atropine has been taken to overcome the dangerous effects of the nerve agent. If symptoms of the nerve agent are not relieved, the service member should be administered two more sets of the MARK I injections plus one injection of **CANA** by a buddy, in accordance with the provisions of paragraph 2-11. If symptoms still persist and the pulse (heart rate) drops below 90 per minute, bronchial secretions persist, or the skin remains moist, then the service member can be administered additional atropine injections by the CLS or medical personnel (who carry additional atropine for the treatment of nerve agent casualties) to maintain adequate atropinization. The CLS and combat medic/corpsman also carry extra **CANA** for administration to nerve agent casualties (para 2-12). The CLS or combat medic/corpsman can administer additional **CANA** up to a maximum of three before evacuating the casualty. Evacuate the service member to a field MTF as soon as the combat situation permits.

f. Atropine and **2 PAM Cl** by injection do not relieve the local effects of nerve agent vapor on the eyes. Although the eyes may hurt and there may be difficulty in focusing and a headache, the service members should carry on with their duties to the best of their ability. These symptoms are annoying but not dangerous.

g. Exposure to high concentrations of a nerve agent may bring on incoordination, mental confusion, and/or collapse so rapidly that the casualty cannot perform self-aid. If this happens, the nearest able service member must render buddy aid.

h. **SEVERE** nerve agent exposure may rapidly cause unconsciousness, muscular paralysis, and the cessation of breathing. When this occurs, antidote alone will not save life. **IMMEDIATELY** after a buddy administers three sets of the MARK I and **CANA,** assisted ventilation must be started by medical personnel, if a resuscitation device is available. Assisted ventilation should be continued until normal breathing is restored.

2-10. The Nerve Agent Antidote Kit, MARK I

The Nerve Agent Antidote Kit, MARK I (fig E-1), is an antidote used by the Army and the Air Force in the treatment of nerve agent poisoning.

a. Description. The MARK I kit consists of four separate components: the atropine autoinjector, the **2 PAM Cl** autoinjector, the plastic clip, and the foam carrying case.

(1) The atropine autoinjector consist of a hard plastic tube containing 2 mg (0.7 milliliter (ml)) of atropine in solution. It has a pressure activated coiled spring mechanism which triggers the needle for injection of the antidote solution. The container is white plastic with yellow lettering on green identification and directions labels. The safety cap is yellow plastic attached to the clip at the rear of the container. The needle end is a green plastic cap which, when pressure is applied, activates the spring mechanism.

(2) The **2 PAM Cl** autoinjector is a hard plastic tube which dispenses 600 mg/2 ml of **2 PAM Cl** (300 mg/ml) solution when activated. It has a pressure activated coiled spring mechanism identical to that in the atropine autoinjector. The container is clear plastic with black lettering on a brown identification label. Directions are in black lettering on a white background. The safety cap is gray plastic attached to the clip at the rear of the container. The needle end is black plastic.

(3) The clip is made of clear hard plastic constructed to hold the pair of autoinjectors together while attached to their safety caps. The safety caps are held flush to the bottom of the plastic clip by a movable metal retaining flange. The clip container recesses are labeled with black numbers: "1" for the atropine and "2" for the **2 PAM Cl** autoinjector.

(4) The foam envelope is a charcoal gray form-fitting case with pressed seams and is designed to carry both autoinjectors. The envelope is used for shipping purposes only and is removed by service members prior to putting the MARK I kits in their mask carrier.

b. Issue to Service Members. In the U.S. Army and the U.S. Air Force, each person is authorized to carry three sets of the MARK I kit for the treatment of nerve agent poisoning. The U.S. Navy, however, does not use the MARK I but, rather, its antidote components are issued as three atropine and three **2 PAM Cl** autoinjectors per person.

c. *Protection Against Freezing.* The atropine and the **2 PAM Cl** solutions freeze at about 30°F (1°C). Therefore, when the temperature is below freezing, the MARK I injectors should be protected against freezing. Autoinjectors issued to the individual service member are normally carried in the protective mask carrier. During cold weather when the temperature is below freezing, the injectors should be carried in an inside pocket close to the body. (Should the MARK I injectors become frozen, they can be thawed and used.)

2-11. Principles in the Use of the Nerve Agent Antidote Kit, MARK I

The following are principles to be followed in the administration of the MARK I (fig E-1).

a. Self-Aid. If you experience most or all of the **MILD** symptoms of nerve agent poisoning (para 2-5), you should **IMMEDIATELY** hold your breath **(DO NOT INHALE)** and put on your protective mask. Then administer *one* set of MARK I injections into your lateral thigh muscle (or buttocks). (Self-aid procedure for administering the autoinjectors is found in app E.)

(1) Wait 10 to 15 minutes after giving yourself the *first* set of injections since it takes that long for the antidote to take effect. If you are able to ambulate, know who you are, and where you are, you **WILL NOT** need a second set of MARK I injections.

WARNING

Giving yourself a second set of injections may create a nerve agent antidote overdose, which could result in incapacitation.

(2) If symptoms of nerve agent poisoning are not relieved after administering one set of MARK I injections, seek someone else to check your symptoms. A buddy must administer the second and third sets of injections, if needed.

b. Buddy Aid. If you encounter a service member suffering from **SEVERE** signs of nerve agent poisoning (para 2-5), render the following aid:

(1) Mask the casualty, if necessary. Do not fasten the hood.

(2) Administer, in rapid succession, three sets of the MARK I.

Follow administration procedures outlined in appendix E.

NOTE

Use the casualty's own antidote autoinjectors when providing aid. Do not use your injectors on a casualty. If you do, you may not have any antidote available when needed for self-aid.

c. Combat Lifesaver. The CLS must check to verify if the individual has received three sets of the MARK I. If not, the CLS performs first aid as described for buddy aid above. If the individual has received the initial three sets of MARK I, then the CLS may administer additional atropine injections at approximately 15 minute intervals until atropinization is achieved (that is a heart rate above 90 beats per minute; reduced bronchial secretions; and reduced salivation). Administer additional atropine at intervals of 30 minutes to 4 hours to maintain atropinization or until the casualty is placed under the care of medical personnel. Check the heart rate by lifting the casualty's mask hood and feeling for a pulse at the carotid artery. Request medical assistance as soon as the tactical situation permits.

d. Combat Medic/Corpsman. A casualty has received three sets of MARK I; however, atropinization has not been achieved. Administer additional atropine at approximately 15 minute intervals until atropinization is achieved (that is a heart rate above 90 beats per minute; reduced bronchial secretions and reduced salivation). Administer additional atropine at intervals of 30 minutes to 4 hours to maintain atropinization or until the casualty is evacuated to an MTF. Check the heart rate by lifting the casualty's mask hood and feeling for a pulse at the carotid artery. Provide assisted ventilation for severely poisoned casualties, if equipment is available. Monitor the patient for development of heat stress.

2-12. Principles in the Use of Convulsant Antidote for Nerve Agents

The following are principles to be followed in the administration of **CANA** (fig E-1).

a. Self-Aid. The **CANA** is **NOT** for use as self-aid. If you know who you are, where you are, and what you are doing, you do not need **CANA.** If symptoms do not subside after self-administering one MARK I, seek assistance from a buddy.

b. Buddy Aid. In addition to administering the MARK I antidotes for nerve agents as buddy aid, also administer the **CANA.**

(1) Mask the casualty, if necessary. Do not fasten the hood.

(2) Administer the **CANA** with the third MARK I to prevent convulsions. **DO NOT** administer more than one **CANA.** Follow administration procedures outlined in appendix E.

NOTE

DO NOT use your own **CANA** on the casualty. You may not have any antidote for your own treatment, if needed.

c. *Combat Lifesaver and Medic/Corpsman.* The CLS or medic/corpsman should administer additional **CANA** to casualties suffering convulsions. Administer a second, and if needed, a third **CANA** at 5 to 10 minute intervals for a maximum of three injections (30 mg diazepam). Follow the steps and procedures described in buddy aid for administering the **CANA. DO NOT** give more than two additional injections for a total of three (one buddy aid plus two by CLS or medic/corpsman).

2-13. Effects 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 muscarinic manifestations. It has little effect on the CNS symptoms and no effect on the nicotinic symptoms. If the casualty has absorbed little or no nerve agent, the administration of a single dose of 2 mg of atropine produces symptoms of mild atropinization (tachycardia, dry mouth) in most individuals and repetition of this dose within 1 or 2 hours produces **MODERATE** symptoms of atropinization in almost all individuals. In contrast, a casualty with **MODERATE** symptoms of nerve agent poisoning will not develop symptoms of atropinization after administration of 2 mg of atropine. A casualty with severe symptoms of nerve agent poisoning may tolerate—indeed may require—considerably more than 4 mg of atropine (as much as 50 mg in 24 hours).

2-14. Effects of Convulsant Antidote for Nerve Agents

Diazepam **(CANA)** is intended to prevent or ameliorate convulsions in **MODERATE** to severe nerve agent poisoning. A casualty with severe nerve agent poisoning may require multiple doses of **CANA** (30 mg or more).

Section IV. TREATMENT IN THE FIELD (MEDICAL TREATMENT FACILITY)

2-15. Administration of the Nerve Agent Antidotes

Upon arrival at the MTF a casualty is still presenting signs/symptoms of nerve agent poisoning (para 2-5). The casualty has received self-aid, buddy aid, CLS care, or treatment by the combat medic/corpsman, or other medical personnel in the field before and during evacuation. Additional injections of the nerve agent antidote(s) must be administered at the field MTF.

a. Atropine. Atropinization should have been achieved before the casualty is evacuated to an MTF; if not, then atropine is administered as follows:

(1) **MILD** symptoms should be treated by administering 2 mg of the atropine every 15 minutes until signs of atropinization (dry mouth and skin, with cleared pulmonary secretions) are achieved. Maintain atropinization until muscarinic signs disappear.

(2) **MODERATE** symptoms should be treated by administering 2 mg of the atropine every 10 to 15 minutes until atropinization is achieved. Maintain atropinization by injecting 2 mg of atropine as often and long as needed.

(3) **SEVERE** symptoms should be treated by administering 2 mg of atropine as frequently as required until atropinization is achieved. Maintain atropinization by injecting 2 mg of atropine every 10 to 30 minutes as long as needed.

NOTE

Smoking should be prohibited until the symptoms of nerve agent poisoning have subsided.

b. 2 *PAM Cl.* Specifically as an adjunct to atropine, **2 PAM Cl** may be used to increase the effectiveness of therapy in poisoning by some, but not all nerve agents. An important facet of the activity of **2 PAM Cl** in such therapy is the reduced duration of required assisted ventilation.

(1) **MILD** symptoms should have been treated by administering at least one 600-mg **IM** injection of **2 PAM Cl.**

(2) **MODERATE** symptoms should have been treated by administering *one* or more 600-mg **IM** injections of **2 PAM Cl.**

(3) **SEVERE** symptoms should have been treated by administering *three* 600-mg **IM** injections of **2 PAM Cl.** Repeat the dose at least every hour if respiration has not improved. Generally, no increased oxime benefit is obtained after three injections of **2 PAM Cl.**

c. Diazepam. Diazepam is used specifically as a prevention of or treatment for convulsions in nerve agent poisoned casualties. If brain damage is to be prevented in **MODERATE** to severe nerve agent poisoned casualties, **CANA** must be administered early. Seizures should be anticipated in all **MODERATE** to severe cases and treated with the **CANA** and repeated as necessary.

2-16. Administration of Follow-on Medical Treatment

The following medical treatment may also be administered in a CPS or a clean (uncontaminated) environment, depending on the patient's needs. Modifications of these procedures may be used in a contaminated environment although an increase in exposure will occur. The alternative of not performing these procedures is death of the patient.

a. Administration of Additional Atropine. For patients who are in severe respiratory distress or are convulsing, all three sets of their MARK I autoinjectors should have been given. (Convulsions are treated with diazepam, as described in *c* below.) If relief does not occur and bronchial secretions and salivation do not decrease, administer additional atropine as often as needed. In severe nerve agent poisoning, the effect of each 2-mg atropine injection may be transient, lasting only 5 to 15 minutes. Therefore, these patients must be closely observed and atropine repeated at intervals that relieve (or counteract) the muscarinic effects of the nerve agent and maintain mild atropinization for as long as necessary.

b. Management of Bronchial Secretions and Salivation. Patients having excessive airway secretions and salivation (an indication for additional atropine) should be lying on their side, with the foot of the litter or bed elevated, if possible, to promote drainage. If airway obstruction is occurring, the collar should be loosened, the tongue pulled out, and the saliva and mucus cleared periodically from the mouth and pharynx by suction. Then an oropharyngeal airway may be inserted and suction carried out intermittently, as needed (through and around the airway). If, despite concentrated efforts to carry out assisted ventilation, the upper airway remains obstructed and adequate exchange of air does not occur, insert an endotracheal tube. High airway resistance because of bronchial constrictions and secretions may be decreased with the administration of additional atropine.

c. Management of Convulsions. Severely poisoned casualties that develop convulsions usually progress rapidly to unconsciousness and generalized muscular weakness or flaccid paralysis, at which point external evidences of convulsions cease. Seizures should be anticipated in all **MODERATE** to severe

cases and expectantly treated with **CANA/diazepam** and repeated as necessary. Seizing is a prominent feature of nerve agent poisoning, especially GD. Administer **CANA** until seizures are controlled.

d. Treatment of Ocular Symptoms. Ocular symptoms produced by local absorption of a nerve agent do not respond to the systemic administration of atropine. However, minimal pain relief may be obtained by the local instillation of atropine sulfate ophthalmic ointment (1 percent), repeated as needed at intervals of several hours for 1 to 3 days. If local ocular effects of a nerve agent are present, the size of the pupils cannot be used as an indicator of the systemic effects of the nerve agent or the atropine.

e. Gastric Lavage. If water or food contaminated with a nerve agent has been ingested, colicky abdominal pains, substernal tightness, increased salivation, and perhaps nausea and vomiting will occur 1/2 hour or later. If ingestion is known to have occurred, early gastric lavage with water should be done.

f. Removal of Liquid Nerve Agent. Any liquid nerve agent on the skin or in the eyes should be removed immediately.

g. Assisted Ventilation. If respiration is severely impaired or if it ceases after administration of atropine, cyanosis will ensue and death will occur within minutes unless immediate effective assisted ventilation is begun and maintained until spontaneous respiration is resumed. Far forward in the field, a cricothyroidotomy is the most practical means of providing an airway for assisted ventilation, using a hand-powered ventilator equipped with an NBC filter. When a casualty reaches an MTF where oxygen and a positive pressure ventilator is available, these should be employed continuously until adequate spontaneous respiration is resumed. An endotracheal tube will most likely be required.

NOTE

Treatment outlined in paragraphs 2-15 and 2-16 is based on the U.S. Army doctrine on the use of the MARK I and CANA (diazepam). These procedures do not address the uniqueness of other environments (such as the threat in naval operations) where alternatives may be more constrained, requiring modification in the procedures. Procedures to address these variations should be issued by the services concerned in accordance with their specific needs.

Section V. NERVE AGENT PYRIDOSTIGMINE PRETREATMENT

2-17. Purpose

a. This section prescribes the use of nerve agent pyridostigmine pretreatment as an adjunct to the MARK I. Studies in many different types of animals indicate that when pyridostigmine is used in conjunction with the MARK I (para 2-10 and app E), the survivability of nerve agent poisoned casualties may be enhanced. Also covered in this section are the individual, unit, and command responsibilities for the pretreatment regimen.

b. Animal data suggest that any potential benefits that may be derived from use of this pretreatment regimen will be realized only in nerve agent poisoned casualties who have been treated with the Mark I at the time of nerve agent exposure, and who have taken their pretreatment medication within 8 hours prior to nerve agent exposure.

c. Minimal detrimental effects are expected at the recommended dosages. Adverse effects and contraindications are described in paragraph 2-22 below.

2-18. The Nerve Agent Pyridostigmine Pretreatment Tablet Set

a. The Nerve Agent Pyridostigmine Pretreatment (NAPP) Tablet Set (fig 2-2) contains the pretreatment medication to be taken within 8 hours prior to exposure to nerve agents at which time the MARK I is used. The NAPP consists of a blister pack containing 21 tablets. Each tablet consists of 30-mg pyridostigmine bromide. Each blister pack (NAPP) contains enough tablets for 7 days (1 taken every 8 hours).

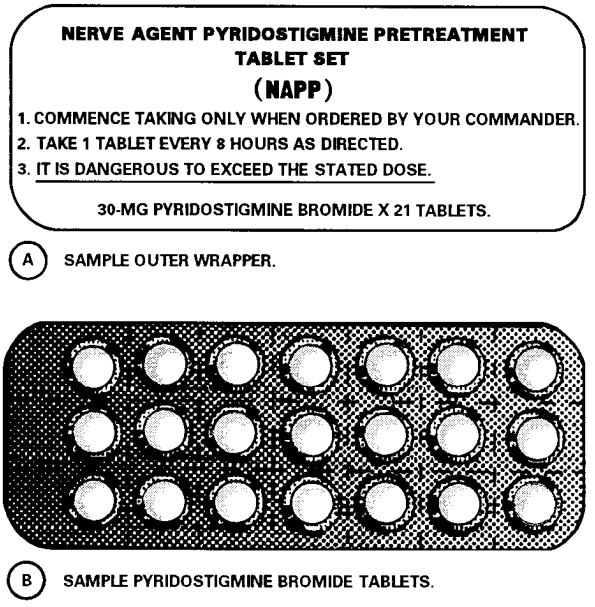


Figure 2-2. Nerve Agent Pyridostigmine Pretreatment Tablet Set.

b. Service members are initially issued one **NAPP** when the chemical protective ensemble is expected to be opened for use. They are responsible for carrying the **NAPP** and safeguarding it against loss. Service members will secure the blister pack in the sleeve or breast pocket of the chemical protective ensemble (or in another part of the ensemble, as directed by local standing operating procedure (SOP)).

NOTE

In conjunction with the **NAPP,** service members should be issued an additional M291 Skin Decontaminating Kit (fig E-l). The M291 kit will be carried in the protective mask carrier or as specified in unit SOP.

c. Orders to start taking the **NAPP** will be issued by the proper authority within the chain of command.

d. Resupply will be provided by combat, combat support, and combat service support units.

2-19. Effects of Pyridostigmine Bromide

a. Pyridostigmine bromide protects an enzyme (known as acetylcholinesterase) in the body from the action of nerve agents. Muscles function as a result of nerve impulses and the release of specific chemical substances. A chemical transmitter, acetylcholine, acts at the neuromuscular junction (where the nerve interfaces with the muscle) (fig 2-3). When a nerve impulse reaches the neuromuscular junction, acetylcholine is released, thereby causing the muscle to contract. The enzyme, acetylcholinesterase, stops the action of acetylcholine on the muscle after the muscle has contracted. Nerve agents block the acetylcholinesterase; there is an accumulation of excessive acetylcholine at the neuromuscular junction resulting in nerve agent poisoning and its accompanying symptoms. Pyridostigmine protects acetylcholinesterase against nerve agents, thus preventing the accumulation of excessive acetylcholine when the MARK I is administrated.

b. Pyridostigmine is not a "true" pretreatment. A true pretreatment would, by itself, provide some protection against chemical agents. Pyridostigmine is an *antidote enhancer.* Though **NOT** providing protection by itself, pyridostigmine significantly **ENHANCES** the efficacy of the MARK I within 1 to 3 hours after taking the first tablet. Maximal benefit develops with time and is reached when a tablet is taken every 8 hours.

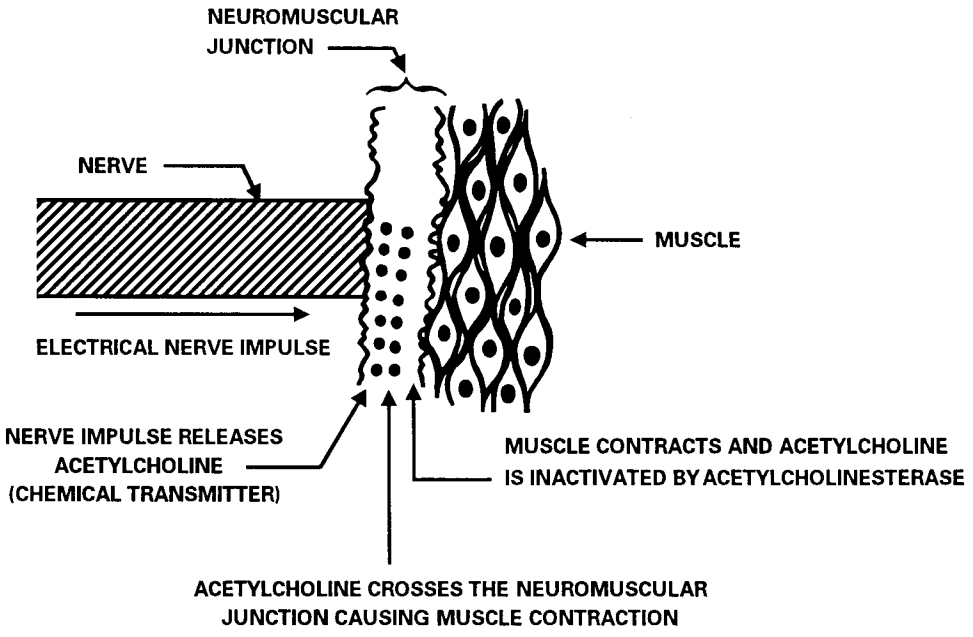


Figure 2-3. Schematic neuromuscular junction (not to scale).

2-20. Principles in the Use of the Nerve Agent Pyridostigmine Pretreatment Tablet Set

a. To be maximally effective, one pyridostigmine bromide tablet should be taken every 8 hours on a continuous basis prior to exposure to a nerve agent until all 21 tablets in the blister pack have been taken, or the individual has been directed to discontinue taking the medication. If pyridostigmine is to be continued, another blister pack of the medication must be issued. This regimen maintains an effective blood level of the medication. If a tablet is not taken every 8 hours, the beneficial effect of pyridostigmine as a pretreatment significantly diminishes after 8 hours from the last tablet.

b. The use of the pyridostigmine pretreatment medication does not change the administration of MARK I.

NOTE

Do not attempt to give a NAPP tablet to a casualty with nerve agent symptoms.

c. At times a commander may have to make a decision to defer administration of the **NAPP** on schedule. Examples of this would be when service members—

(1) Have experienced sleep deprivation. The commander would have to decide whether the service members should be allowed to sleep or be awakened to take the pretreatment.

(2) Are in a contaminated environment. The commander would have to decide whether or not to delay administration of the medication until the unit is safely out of the contaminated area (para *d* below). In any case, the benefits versus the risks should be carefully weighed before a decision is reached.

d. When the order to take pyridostigmine has been given, it should be taken as directed (para 2-21). As long as the environment is contaminated, it is desirable to continue the pretreatment. The pretreatment should continue regardless of MOPP level since the protective posture could be breached at any time. Command guidelines should be developed for situations such as—

(1) Providing collective protection or rest and relief shelters so that personnel can remove their protective mask and take the tablets, or relocate small groups to an uncontaminated area, if possible.

(2) Taking the tablets while in MOPP 4 would be hazardous. (Examples: Troops are operating at night without lights or are in a chemical agent vapor environment.) In either case it would be more appropriate to delay taking the medication for a few hours until the tablets can be taken in a less hazardous environment.

e. The NAPP should not be taken during pregnancy.

2-21. Administration of Pyridostigmine Pretreatment in an Uncontaminated Environment

One 30-mg tablet is to be taken by mouth, with sufficient water to assist in swallowing the medication, every 8 hours as directed by your commander. *If a dose is missed, do not make it up. Do not take 2 tablets at once because of a missed dose—merely start again with 1 tablet every 8 hours.* Taking 2 tablets at once could result in adverse side effects. Taking more than 1 tablet at a time **DOES NOT** provide additional protection—in fact, it maybe more hazardous if there is exposure to a nerve agent.

a. When the order to take pyridostigmine pretreatment has been given, it should be taken as directed, even though the protective mask is worn.

b. During hours of darkness while in an uncontaminated environment, the **NAPP** will be administered using the above schedule.

2-22. Signs and Symptoms of Pyridostigmine Bromide Overdose, Adverse Reactions, and Contraindications

Although no detrimental effects are expected at the recommended dosage, depending on the length of time and the amount of medication taken, as well as individual physiologic variations, some individuals may have contraindications for taking pyridostigmine bromide while others may experience adverse reactions.

a. Signs and symptoms of overdose, adverse reactions, or side effects are—

(1) Abdominal cramps.
(2) Nausea and vomiting.
(3) Diarrhea.
(4) Blurring of vision, miosis.
(5) Increased bronchial secretions.
(6) Cardiac arrhythmias, hypertension.
(7) Weakness, muscle cramps, and muscular twitching.
(8) Skin rash.

b. Since pyridostigmine bromide may increase bronchial secretions and aggravate bronchiolar constriction, caution should be used in its administration to personnel with bronchial asthma.

c. Pyridostigmine bromide may cause urinary obstruction.

d. Additional contraindications include hyperthyroidism, sensitivity to bromide, peptic ulcer disease, and low serum acetylcholinesterase.

e. If any of the above signs/symptoms occur, the service member should consult unit medical personnel as soon as possible.

2-23. Emergency Medical Treatment for Pyridostigmine Adverse Side Effects, Allergic Reactions, and Overdose

Ordinarily, discontinuing pyridostigmine should be adequate to alleviate the signs and symptoms of adverse side effects, allergic reactions, and overdose. Pyridostigmine may persist in the blood for as long as 24 hours; however, after the blood level peaks in about 4 hours, the effects of the medication diminish gradually.

a. Emergency treatment for an overdose of pyridostigmine requires the administration of atropine in adequate doses to overcome the cholinergic crisis. Initially, the 2-mg atropine autoinjector found in the MARK I kit should be used. In most cases, this will be sufficient. Further administration of atropine may be necessary to control the cholinergic effects of pyridostigmine. If additional atropine is required, 2 mg should be administered by medical personnel every 15 to 20 minutes, thereby permitting the previous injection of atropine to exert its anticholinergic effect prior to the next injection.

b. **SEVERE** cases may require assisted ventilation because of weakness, but would be unusual when the pretreatment medication was administered every 8 hours as directed.

c. When stabilized, the patient should be evacuated for further observation and treatment.

2-24. Responsibilities

a. The corps / division / wing commander will–

(1) Decide whether to begin, continue, or discontinue the administration of **NAPP** based on the threat. The intelligence officer, chemical officer, and the surgeon act as advisors to the commander in making his decision if a chemical nerve agent threat exists (for example, the enemy having nerve agents in the combat zone or the probability of their use). After 3 days of self-administration of **NAPP** by the service member, combat conditions should be reevaluated by the commander and his staff to determinate whether to continue the medication or not. However, orders to discontinue the pretreatment **CAN** and **SHOULD** be made at any time, depending on the situation. If the pretreatment is to be continued, then a second blister pack must be ordered while the service member completes the administration of the 7 days (21 tablets) and is issued the second pack on the 7th day. *Administration of the medication beyond 14 days is not recommended without a thorough evaluation of the situation and recommendation of the medical authority. However, the magnitude of the threat may outweigh any possible adverse side effects and indicate continuance of the pretreatment.*

(2) Train the service members to faithfully take the **NAPP** as directed to enhance their survivability if they are exposed to a nerve agent. Service members must be trained to take the **NAPP** during the day, at night, and while in MOPP 4, should these procedures become necessary.

(3) Issue unit **SOPs** for the retention and decontamination of the **NAPP** blister pack during personnel decontamination and overgarment exchange.

b. Units will–

(1) Obtain the supplies of **NAPP** through medical supply channels.

(2) Maintain at least a 2-week supply of **NAPP** per member of the unit. One **NAPP** is issued to each member of the unit. An additional week's supply of **NAPP** for each individual in the unit will be maintained in the unit area. Authorized quantities will be commensurate with the latest doctrine for its use.

(3) Store the **NAPP** for individual issue and request replacements as the items are issued, or as they exceed their labeled shelf life. The **NAPP** should be stored (refrigerated) in temperatures ranging from 350 to 46°F (2° to 8°C). If the medication is removed from refrigeration for a total of 6 months, it should be assumed that it has lost its potency and should not be used.

(4) Issue the **NAPP** to the service members at the time the chemical protective ensemble is expected to be opened for use.

c. Unit medical personnel will—

(1) Recognize the signs and symptoms of pyridostigmine overdose, adverse reactions, and side effects (para 2-22 above) for determining, on an individual basis, whether or not a service member is to continue the **NAPP** based on any adverse reaction to the medication.

(2) Advise the commander if any serious problems occur.

d. The individual service member will—

(1) Take the **NAPP** as directed and in accordance with the provisions of paragraph 2-20 above.

(2) Secure the **NAPP** against loss.

CHAPTER 3
INCAPACITATING AGENTS

3-1. General

a. An incapacitating agent is a chemical agent which produces temporary disabling conditions. The disabling conditions persist for hours to days after exposure to the agent (unlike that produced by riot control agents, which usually are momentary or fleeting in action). Medical treatment, while not essential, may facilitate more rapid recovery. In the narrower sense, the term "incapacitating agents" has come to mean those agents that are—

(1) Highly potent (an extremely low dose is effective) and logistically feasible.

(2) Able to produce their effects mainly by altering the higher regulatory activity of the CNS.

(3) Temporary in duration of action lasting hours or days, rather than of a momentary or fleeting action.

(4) Not likely to produce permanent injury in concentrations which are militarily effective.

b. Incapacitating agents are not considered to include the following:

(1) Lethal agents, such as nerve agents which are incapacitating at sublethal doses.

(2) Substances which cause permanent or long-lasting injury, such as blister agents, choking agents, and those injuring the eyes.

(3) Common pharmaceutical substances with strong CNS actions, such as the belladonna alkaloids, tranquilizers, and many hallucinogens. These drugs, although effective and relatively safe, are logistically infeasible for large-scale use because of the large amounts required.

(4) Agents which are transiently effective by producing reflex responses interfering with duty performance. These include vomiting and irritant agents.

(5) Agents which disrupt basic life-sustaining systems and prevent physical activity. Examples include agents which lower the blood pressure, paralyzing agents (such as curare), respiratory depressants, and agents that interfere with oxygen transport. Although theoretically effective, such agents almost invariably have a low margin of safety between the effective dose and possible lethal dose. Therefore, these agents defeat the basic purpose of an incapacitating agent: to reduce military effectiveness without endangering life.

c. Despite restrictions imposed by the above definition, a great variety of mechanisms remain by which CNS regulation and maintenance of performance could theoretically be disrupted. In reality, however, only two general types of chemical agents are likely to be encountered in military use.

(1) *Central nervous system depressants.*

(a) These compounds produce their effects by interfering with information transmission across central synapses. An example of this type of agent is BZ (table 1-1) which blocks the muscarinic action of acetylcholine, both peripherally and centrally. The CNS anticholinergic compounds disrupt the high integrative functions of memory, problem solving, attention, and comprehension. A relatively high dose produces toxic delirium, destroying the individual's ability to perform any military task.

(b) Cannabinols and phenothiazine-type compounds are potential incapacitating agents which seem to act as CNS depressants. The primary effects of these agents are to sedate and destroy motivation rather than disrupt the ability to think.

(2) *Central nervous system stimulants.* These agents cause excessive nervous activity by facilitating transmission of impulses. The effect is to flood the cortex and other higher regulatory centers with too much information. This flooding makes concentration difficult and causes indecisiveness and an inability to act in a sustained, purposeful manner. A well-known drug which appears to act in this manner is d-lysergic acid diethylamide (LSD); similar effects are sometimes produced by large doses of amphetamines.

3-2. Diagnosis

Current, field laboratory methods do not permit isolation and identification of specific agents in the environment or in samples of body fluid (for example, blood, urine, cerebrospinal fluid). Therefore, diagnosis rests almost entirely upon chemical acumen, combined with whatever field intelligence or detector system data that may be available. Following a suspected incapacitating agent attack, the medical officer should take the steps listed below.

a. Instruct evacuation teams to transport casualties to an uncontaminated area. After initial treatment, resistant or disoriented individuals should be restrained in the triage area.

b. Once the diagnosis of a nerve agent or other lethal substance has been ruled out, the principal signs and symptoms to consider are those shown in table 3-1.

Table 3-1. Signs and Symptoms Produced by Incapacitating Agents

SIGNS AND SYMPTOMS	POSSIBLE ETIOLOGY
Restlessness, dizziness, or giddiness; failure to obey orders, confusion, erratic behavior; stumbling or staggering; vomiting.	Anticholinergics (e.g., BZ), indoles (e.g., LSD), cannabinols (e.g., marihuana), anxiety reaction, other intoxications (e.g., alcohol, bromides, barbiturates, lead).
Dryness of mouth, tachycardia at rest, elevated temperature, flushing of face; blurred vision, pupillary dilation; slurred or nonsensical speech, hallucinatory behavior, disrobing, mumbling and picking behavior, stupor and coma.	Anticholinergics
Inappropriate smiling or laughter, irrational fear, distractibility, difficulty expressing self, perceptual distortions; labile increase in pupil size, heart rate, blood pressure. Stomach cramps and vomiting may occur.	Indoles (Schizophrenic psychosis may mimic in some respects.)
Euphoric, relaxed, unconcerned daydreaming attitude, easy laughter; hypotension and dizziness on sudden standing.	Cannabinols
Tremor, clinging or pleading, crying; clear answers, decrease in disturbance with reassurance; history of nervousness or immaturity, phobias.	Anxiety reaction

c. In a large-scale attack, the diagnosis will be simplified by the epidemiological distribution of the casualties. Look for characteristics common to all or most casualties, rather than atypical features. For example, some anticholinergics cause marked disorientation, incoherence, confusion, and hallucinations (the pathognomonic features of delirium) with very little, if any, evidence of peripheral autonomic effect (such as tachycardia and dilated pupils). This should not dissuade the medical officer from considering the likelihood of a centrally predominant anticholinergic being the causative agent. Very few other pharmaceutical classes can produce delirium in militarily effective doses. The disturbance produced by indoles (such as LSD) or the cannabinols (such as marihuana extracts) is not really delirium. Indole casualties remain receptive to their environment and can comprehend quite well, even though they may have great difficulty reacting appropriately.

3-3. Protection, Decontamination, and First Aid

a. Protection. It is likely that such agents will be dispersed by smoke-producing munitions or aerosols and use the respiratory tract as the portal of entry. The use of the protective mask is essential to prevent inhaling the agent. With some agents, the percutaneous route may be used, thus MOPP 4 will be required.

b. Decontamination. Complete cleansing of the skin with soap and water should be accomplished at the earliest opportunity. The M291 Skin Decontaminating Kit can be used (app D) if washing is impossible. Symptoms may appear as late as 36 hours after percutaneous exposure, even if the skin is washed within an hour. In fact, a delay in onset of several hours is typical. This time should be used to prepare for the possibility of an epidemic outbreak 6 to 24 hours after the attack.

c. First Aid. The most important considerations are the following:

(1) If the casualty is stuporous or comatose, be sure that respiration is unobstructed; then turn the casualty onto one side to avoid aspiration in case vomiting should occur.

(2) If the body temperature is elevated above 102°F (39°C) and mucous membranes are dry, immediate and vigorous cooling (as for heatstroke) is indicated. (Methods that can be used to cool the skin are spraying with 72 to 75°F (22 to 24°C) water and air circulation (fanning); applying alcohol-soaked cloths and air circulation; and providing maximum exposure to air in a shaded area, along with maximum air circulation. **DO NOT USE ICE FOR SKIN COOLING.)** Such cases are usually the result of anticholinergic intoxication. Rapid evacuation should be accomplished since treatment with appropriate medication may be lifesaving.

(3) Reassurance and a firm, but friendly, attitude by personnel administering first aid will be beneficial if the casualty appears to comprehend what is being said. Conversation is a waste of time if the individual is incoherent or cannot understand what is being said. In such cases, the less said, the better it is—these patients will benefit more from prompt and vigorous restraint and evacuation to an MTF.

(4) Although anticholinergic poisoning may produce alarming dryness and coating of the lips and tongue, there is usually no danger of immediate dehydration. In such cases, fluids should be given sparingly—if at all—because of the danger of vomiting and the likelihood of temporary urinary retention. (Temporary urinary retention is due to paralysis of the bladder smooth muscle.) Cleansing the mouth with an astringent swab may be comforting and will reduce the foul breath associated with membrane parching.

(5) Weapons and other potentially harmful items should be removed from the possession of individuals who are suspected of being casualties. This includes cigarettes, matches, medications, and small items which might be accidentally ingested. Delirious casualties have been known to attempt to eat items bearing only a superficial resemblance to food.

3-4. Treatment

General treatment consists of close observation, restraint and confinement (as required), supportive care with fluids, and appropriate clothing. Underlying medical problems should be identified and treated as they would be ordinarily.

a. Anticholinergics. Certain cholinesterase inhibitors (such as physostigmine) are highly active antagonists of the centrally active anticholinergics. Neostigmine and pyridostigmine are ineffective because they lack the tertiary nitrogen required to enable them to pass the blood-brain barrier. Treatment with 2 to 3 mg of physostigmine salicylate IM will be required to alleviate the condition. Repeated injections at intervals of approximately 15 minutes to 1 hour may be required to build up a sufficient level. Once a desirable effect is achieved, it should be maintained by slow intravenous (IV) injection or infusion. Doses of 2 to 4 mg every 1 to 2 hours may be required. The dose should be titrated against symptoms with gradual tapering of the dose as the effect of the poisoning runs its course. This may vary from a few hours to several weeks. Oral dosing should replace IV therapy as soon as possible (2 to 5 mg every 1 to 2 hours).

NOTE

1. Phenothiazines and other sedatives (such as chloral hydrate) will potentate the effects of these depressant compounds and are specifically contraindicated.

2. An overdose of physostigmine can result in cholinergic toxicity up to and including temporary apnea. If apnea occurs, assisted ventilation is indicated. Small doses (0.5 mg) of atropine given intravenously may be used to control less severe symptoms of overdose. Since the half-life of physostigmine is only about 30 minutes, overtreatment usually does not require any additional therapy for spontaneous recovery to occur. Then treatment can be resumed, using a slightly smaller and less frequent dosage.

b. Indoles. No true antagonist to the indoles is as yet known. The best treatment known at present for LSD intoxication is the administration of diazepam 10 to 20 mg IV or IM to sedate the patient until spontaneous recovery occurs. Chlorpromazine 50 to 100 mg IM injection has been suggested but does not appear to have any advantage over these drugs.

c. Cannabinols. Stimulants such as d-amphetamine (15 mg) can antagonize the sedation and indifference induced by marihuana-like substances. Although amphetamine may slightly potentate the effects of LSD (if given to such individuals in error), this is not a contraindication to its use if cannabinol intoxication is suspected.

d. Other Agents. Unfamiliar agents or mixtures of agents may be encountered on future battlefields. In such instances, the general principles of restraint, close observation, and supportive medical care apply. No medication should be given until an etiological diagnosis can be made with reasonable certainty—unless circumstances require it (for example, concomitant wounds, burns, or fractures requiring major surgical intervention). The judgment of the medical officer remains the only useful guide to action in these complex and unforeseeable circumstances.

CHAPTER 4

BLISTER AGENTS (VESICANTS)

Section I. INTRODUCTION

4-1. General

a. Blister agents (vesicants) are likely to be used to produce casualties and to force opposing troops to wear full protective equipment. Blister agents are used to degrade fighting efficiency rather than to kill, although exposure to such agents can be fatal. Thickened blister agents will contaminate terrain, ships, aircraft, vehicles, or equipment and present a persistent hazard. Vesicants include sulphur mustard (H and HD), nitrogen mustards (HN), lewisite (L) (this may be used in mixture with HD), and halogenated oximes (example, phosgene oxime (CX)). Halogenated oximes properties and effects are very different from those of the other vesicants.

b. Vesicants burn and blister the skin or any other part of the body they contact. They may act on the eyes, mucous membranes, lungs, and skin; mustards may act on blood-forming organs. They damage the respiratory tract when inhaled and cause vomiting and diarrhea when ingested.

c. Some vesicants have a faint odor; others are odorless. They often have more serious effects than is immediately apparent. Both L and CX cause immediate pain on contact. The mustards are insidious in action, with little or no pain at the time of exposure. 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 blister agent burn is directly related to the concentration of the agent and the duration of contact with the skin.

4-2. Self-Aid

a. Assume MOPP 4 whenever liquid or vaporized agents are known to be present.

b. Liquid vesicants in the eyes or on the skin require immediate decontamination procedures as outlined in appendix D.

4-3. Precautions in Receiving Casualties

a. Casualties contaminated with vesicants endanger unprotected attendants. Individuals in contact with these casualties must be at MOPP 4, plus wear a butyl rubber apron.

b. Special precautions must be taken in receiving contaminated casualties to prevent injury to others. Contaminated casualties are decontaminated outside the field MTF to prevent vapor accumulation indoors. They are kept separated from clean (uncontaminated) casualties until decontamination is completed. Contaminated litters, blankets, and equipment must be left outdoors. Decontamination is necessary for equipment, vehicles, watercraft, and aircraft that have been used to transport contaminated casualties. Appendix B contains further information on decontamination.

c. Unhydrolyzed mustard on patients' skin surface can present a hazard to individuals receiving or treating these patients even after several hours. As mustard reacts with skin and subcutaneous tissue, it is hydrolyzed; however, the destroyed tissue becomes a barrier for complete hydrolyzation of excess mustard on the surface.

4-4. Protective Devices

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

b. Chemical protective overgarments help prevent the vesicant from reaching the skin.

4-5. Disposition of Casualties

See section V for disposition of casualties with blister agent burns.

Section II. MUSTARDS

4-6. Mustard (H and HD)

a. Physical Properties. Mustard is an oily liquid ranging from colorless, when pure (neat), to dark brown when plant-run (unpurified form when first produced). Mustard is heavier than water, but small droplets float on water surfaces and present a special hazard in contaminated areas. It smells like garlic or horseradish. Distilled HD, the most common form of 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. It is freely soluble in acetone, carbon tetrachloride, alcohol and liquid fuels (gasoline, kerosene, and diesel); however, these solvents do not destroy mustard. Mustard disappears from contaminated ground or materials through evaporation or through hydrolysis. It is rapidly destroyed by decontaminating chemicals or by boiling in water. The primary use of mustard is to cause delayed casualties by the liquid and vapor effects on the skin and the eyes and by the vapor effects through the respiratory system.

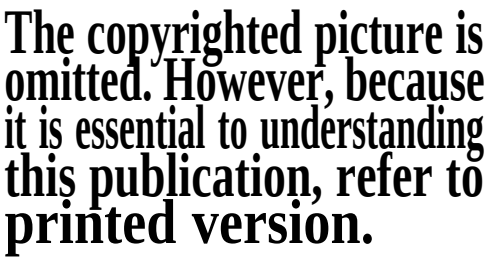
b. Persistence. The persistence of hazard from mustard vapor or liquid depends on the degree of contamination by the liquid, type of mustard, nature of the terrain and soil, type of munition used, and weather conditions. Mustard may persist much longer in wooded areas than in the open. Mustard persists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

c. Cumulative Efect. 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 of HD on the Eyes

a. Pathology, Symptoms, and Prognosis. In a single exposure, the eyes are more susceptible to mustard than either the respiratory tract or the skin. Figures 4-1 through 4-4 show effects of mustard on the eyes. Conjunctivitis follows an exposure time of about 1 hour to a concentration barely perceptible by odor. 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 conjunctival and the lids become red and edematous. Heavy exposure irritates the eyes after 1 to 3 hours and produces some severe lesions. Although temporary blindness may occur, permanent blindness is very

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rare. Casualties should therefore be reassured and a positive attitude taken. Care must be exercised to avoid transferring liquid agent from the hands to the eyes. Mustard burns of the eyes may be divided as follows:

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

(2) Severe conjunctivitis with minimal corneal involvement (15 percent of the cases in World War I). Blepharospasm, edema of the lids, and conjunctival occur, as may orange-peel roughening of the cornea. Recovery takes 2 to 5 weeks.

(3) Mild corneal involvement (10 percent of the cases in World War I). Areas of corneal erosion stain green with fluorescein. Superficial corneal scarring and vascularization occurs as does iritis. Temporary relapses occur and convalescence may take 2 to 3 months. Hospital care is indicated for casualties of this type.

(4) Severe corneal involvement (about 0.1 percent of mustard casualties in World War I). Ischemic necrosis of conjunctival may be seen. Dense corneal opacification with deep ulceration and vascularization occurs. Convalescence may take several months. Patients may be predisposed to late relapses.

b. Treatment.

(1) *Self-aid.*

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

(b) Speed in decontaminating the eyes is absolutely essential. This self-aid procedure is very effective for mustard within the first few seconds after exposure but is of less value after 2 minutes. Decontamination is done the same as for other vesicants (app D).

(2) *Treatment of mustard conjunctivitis.*

(a) Mild lesions require little treatment. Although the lesions may become infected, a steroid antibiotic eye ointment, such as dexamethasone sodium phosphate-neomycin ophthalmic ointment, can be applied. Ophthalmic ointments, such as 5 percent boric acid ointment, will provide lubrication and minimal antibacterial effects. The application of sterile petroleum jelly between the eyelids will provide additional lubrication and prevent sealing of the eyelids.

(b) More severe injuries will cause enough edema of the lids, photophobia, and blepharospasm to obstruct vision. This obstruction of vision alarms patients. To allay their fears, the lids may be gently forced open to assure them that they are not blind.

(c) The pain is controlled best by systemic narcotic analgesics. 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 every 4 hours. Other antibacterial ophthalmic preparations may be substituted for sodium sulfacetamide.

(d) The eye must not be bandaged or the lids allowed to stick together. Sealing of the lids may be prevented as described in *a* above. The accumulation of secretions in the conjunctival sac or pressure on the eye predisposes to corneal ulceration.

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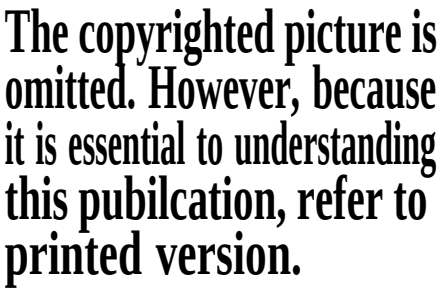

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Figure 4-3. Casualty showing eye effects of mustard vapor.



Figure 4-4. Casualty showing effects of mustard conjunctivitis.

To prevent complications, the patient should be treated by an ophthalmologist as soon as possible. When possible, the patient should be kept in a darkened room, given dark sunglasses, or given an eyeshade to help his photophobia.

(3) *Treatment of infected mustard bums of the 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) *(c)* above. Patients with secondary infection or other complications should be referred to an ophthalmologist. Local anesthetics should not be used.

c. Classification of Eye Lesions. See paragraph 4-29b.

4-8. Effects of HD on the Skin

a. Pathology. The severity of the lesions and the rapidity with which they develop are greatly influenced by weather conditions as well as by the degree of 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 period is usually 6 to 12 hours.

(2) *Erythema.* Erythema gradually appears (2 to 48 hours postexposure) and becomes brighter, resembling sunburn (figs 4-5 and 4-6). Slight edema of the skin may occur. In severe burns, the edema may limit motion of the limb. Itching is common and may be intense. As the erythema fades, areas of increased pigmentation are left (this sequence is reminiscent of that seen in sunburn).

(3) *Vesication.* Except with mild vapor burns, erythema is followed by vesication (figs 4-7, 4-8, 4-9, and 4-10). 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 intra-epidermal vesicle. Clinically, multiple pinpoint lesions may arise within the erythematous skin; these enlarge and coalesce to form the typical blister (which is unusually large, domed, thin-walled, yellowish, and may be surrounded by erythema). The blister is filled with a clear or slightly yellow liquid that tends to coagulate. The blister fluid does not contain mustard and is not a vesicant. 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. As noted in paragraph 4-3 *c* above, unhydrolyzed vesicant on contaminated patients may pose a hazard to other individuals coming in contact with them.

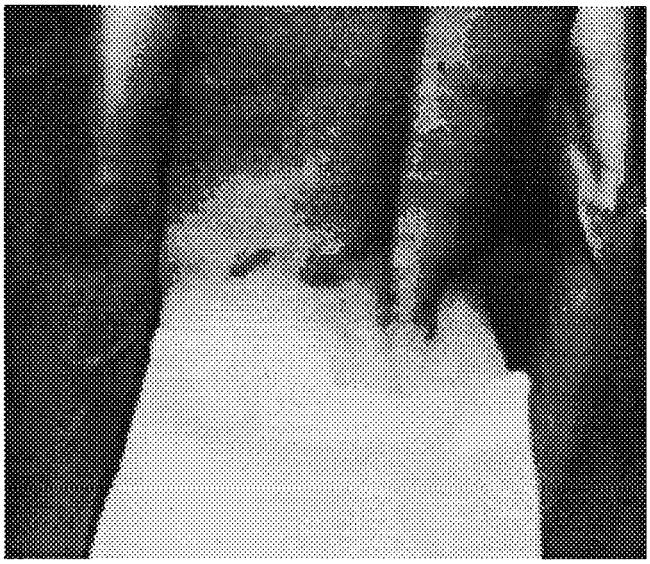


Figure 4-5. Noncasualty with erythema.

(4) *Resorption.* If the blister does not rupture, resorption takes place in about a week. The roof forms a crust beneath which reepidermization 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. Once the blister has broken, it is best to remove its ragged roof to decrease the possibility of secondary infection.

(5) *Healing.* Since the damage to the corium is relatively superficial, healing occurs with little scar tissue formation, except in more extensive or infected burns where scarring is more severe.

(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 (figs 4-11 and 4-12).

(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 immediate symptoms, nor do any local manifestations occur until erythema develops. At this time there may be itching and mild burning. This pruritus may last several days and persist after healing. The blisters may be painful.

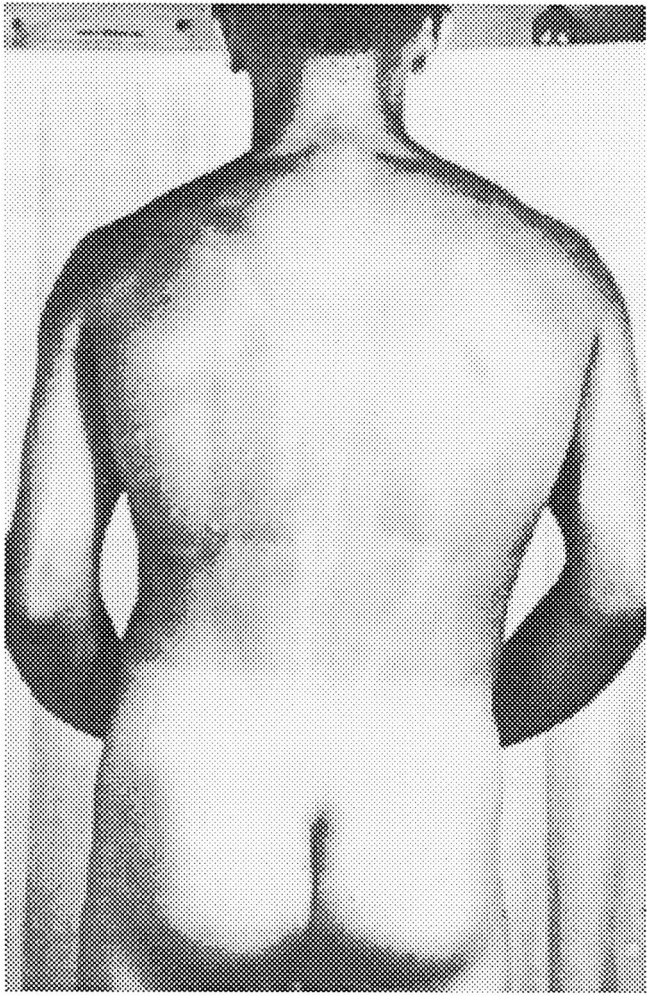


Figure 4-6. Casualty with generalized erythema and systemic intoxication.

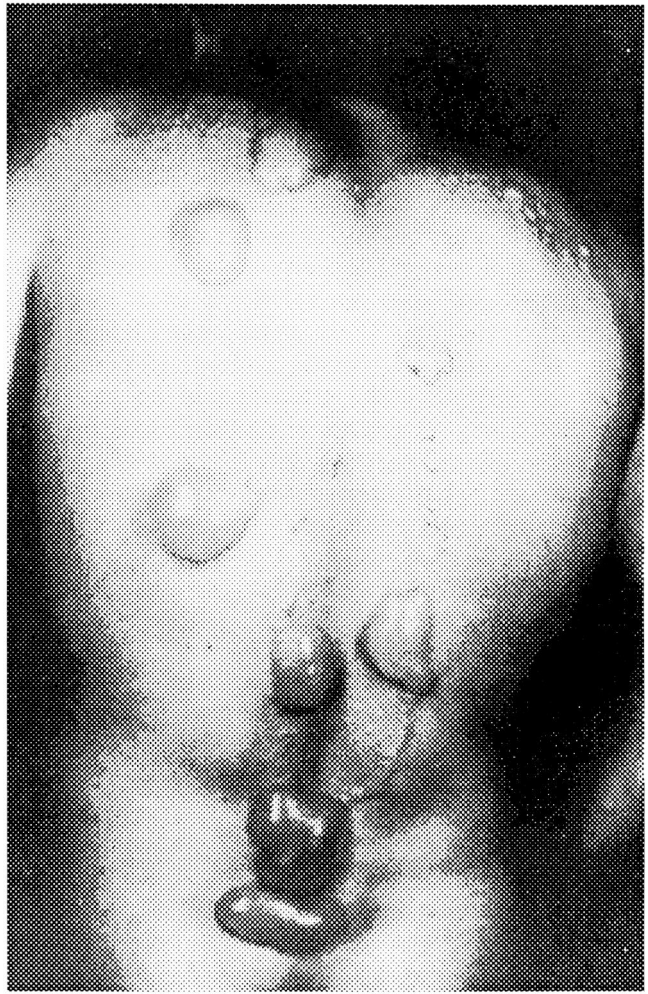


Figure 4-7. Casualty with severe vesication.

Click here for Figure 4-6.

Click here for Figure 4-7.

(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. Healing times for mustard blisters vary widely with both severity and anatomical location. In general, blisters of the face heal in 1 to 2 weeks. Blisters located in other areas may take slightly longer to heal; but if protected from infection, they will heal in 2 to 4 weeks. If cutaneous injury results in full-thickness coagulation necrosis, skin grafting may ultimately be necessary. However, a mustard burn of the skin is usually limited to the epidermis and does not require grafting (fig 4-13).

(3) Moderate contamination of mustard skin lesions with saprophytic bacteria, which causes 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 (fig 4-14).

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 caused by arsenical vesicants. Differentiation of mustard lesions from those produced by arsenical is based upon—

(1) History of exposure to mustard.

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

(3) A zone of erythema surrounding blisters (not predominant with arsenical). 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. However, the skin lesions of plant contact are on exposed skin and linear in configuration. The earliest affected areas of skin from mustard are the skin folds, groin, and inner aspects of the extremities.

d. Decontamination of Casualties. Casualties who have experienced liquid mustard contamination of the

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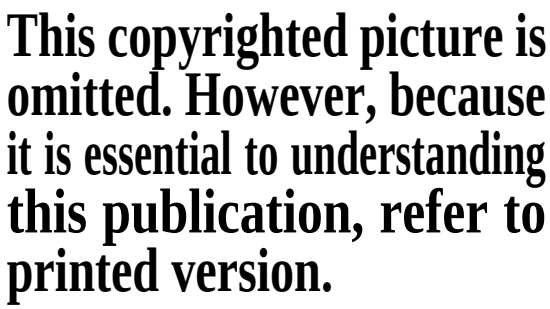
skin or clothing will seldom be received by the medical service in time to prevent subsequent blistering. Nevertheless, if erythema has not appeared, known or likely contaminated skin areas should be decontaminated as described in appendix D. Cut away and discard hair contaminated with liquid mustard. Decontaminate the exposed scalp with the M291 Skin Decontaminating Kit. If short of these substances, use 0.5 percent aqueous chlorine solution for decontamination of skin and hair. Wash off the decontaminating solutions promptly (within 3 or 4 minutes) to prevent additional skin injury, taking care that none of the solutions wash 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.

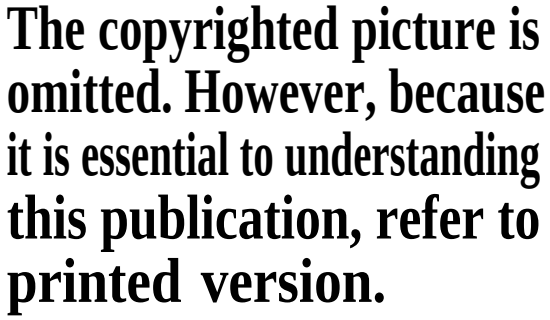
e. Treatment of Mustard Erythema. Mustard erythema in mild cases requires no treatment. If an annoying itch is present, considerable relief may be obtained with topical steroid creams or sprays. Severe erythema around the genitalia may become quite painful and associated weeping and maceration may ensue. 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 Blisters.

(1) Once blisters have broken, it is best to remove its ragged roof to decrease the possibility of secondary infection. Cleanse the area with tap water or saline, then apply sterile petrolatum gauze when the areas are small. Dressings should be changed and the wound inspected every 3 to 4 days. Small blisters on the face are opened and best left uncovered. Large blisters may best be treated by open methods. Apply about one-eighth of an inch thick layer of 10 percent mafenide acetate or silver sulfadiazine burn cream to the blisters as a topical antibiotic agent. Figure 4-15 shows a casualty with widespread vesication caused by mustard burns.

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(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. Figure 4-16 shows burns produced by the reaction of mustard vapor with sweat.

(2) Wounds which become infected must be treated with appropriate antibiotics after adequate cultures have been obtained. The medical officer 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 antibacterial drugs may be given either locally or systemically, as indicated. The early use of an appropriate topical antibacterial agent (such as mafenide acetate or silver sulfadiazine cream) may prevent a bacterial infection.

4-9. Effects of HD 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 epitheliums. 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, and focal atelectasis. Altogether, these changes may be insufficient to cause hypoxia and 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 early mortality from mustard among American troops in World War I (slightly more than 2 percent) was due almost entirely to such pulmonary complications following inhalation of vapor.

Click here for Figure 4-12.

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 loss of voice. A cough (worse at night) appears early and later becomes productive. Fever, dyspnea, rhonchi, and moist rales may develop. The incidence of bronchopneumonia is high. Convalescence is slow; the cough may persist a month or longer. Milder symptoms, like hoarseness, last only 1 or 2 weeks.

c. Treatment of Respirator 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-containing cough syrups. Laryngitis and tracheitis may be treated symptomatically with steam or sterile cool mist inhalations. If more severe respiratory tract injury is suspected, hospitalization may be advisable. If a bacterial pneumonia occurs, isolation of the specific organisms with their antibiotic sensitivities should be performed, then antibiotic therapy can be limited to the specific agents.

4-10. Systemic and Gastrointestinal Effects of HD

a. Pathology.

(1) Ingestion of mustard produces vacuoles 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 damage to the blood-forming organs mentioned in (2) below.

(2) With lesser skin or respiratory exposures to mustard, no apparent systemic lesions develop. However, 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 leukopenia, thrombocytopenia, and anemia. 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 hypovolemic shock may occur. It must be emphasized that severe systemic effects only occur when sufficient agent has been absorbed systemically. Lesser mustard exposures do not cause severe systemic effects.

c. Prognosis.

(1) With mild to moderate field exposures to mustard vapor, it is not anticipated that deaths will occur from the systemic effects of the absorbed mustard. However, death may occur from prolonged exposures to high concentrations 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 leukopenia in these cases may be regarded as grave prognostic signs. Bone marrow failure is the most frequent cause of late deaths.

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

d. 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 or aluminum-laminated pouches (meal, ready to eat (MRE) pouches), until examined by U.S. Army veterinary personnel and certified as safe to eat. Refer to FM 3-5, FM 8-10-7, and TB MED 577 for additional information.

e. Treatment of Systemic Mustard Poisoning.

(1) In the treatment of systemic symptoms, atropine subcutaneously (0. 4 to 0.8 mg; NOT the 2 mg 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, leukopenia, hemoconcentration, and shock, every effort should be made to maintain an adequate nutritional status and to replace the loss of fluid and electrolytes. There may be a need to monitor the white blood count, hemoglobin, and platelets in severe systemic poisoning. If the white blood count decreases significantly, isolation and appropriate antibiotics may be necessary. It has been suggested by some authorities that sodium thiosulfate will prevent or reduce damage from mustard, provided that it can be given IV within 20 minutes of exposure. Its efficacy is very doubtful if given later.

(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.

4-11. Nitrogen Mustards

The HNs are oily, colorless, pale yellow liquids, sparingly soluble in water but freely soluble in organic solvents. Some have a faint fishy odor, while others are odorless. Their volatility varies with the particular compound. All are persistent but not equally so. The most likely to be seen are HN1 and HN3. Nitrogen mustard (HN1) is more volatile and less persistent than HD but only one-fifth as vesicant to the skin as mustard. Nitrogen mustard (HN3) is less volatile and more persistent and about equal to HD in its vesicant effects. Nitrogen mustards are less readily hydrolyzed than HD. All their hydrolytic products, except the final ones, are toxic.

4-12. Effects of HN on the Eyes

a. Pathology and Symptoms. In single exposures, HN irritates the eye in doses which do not significantly damage the skin or respiratory tract. This irritation appears sooner than that from HD. Mild or moderate exposure causes light smarting and lacrimation within 20 minutes. Thereafter, symptoms may wax and wane until they become persistent about 2 1/2 hours later and reach the maximum in 8 to 10 hours. After more severe exposure, symptoms begin immediately and progress for 24 hours or longer. Mild exposure produces erythema and edema of the palpebral and bulbar conjunctival and superficial, steamy haziness of the cornea. Irritation, lacrimation, deep eye pain, miosis, and photophobia are usually present. After more severe exposure, these symptoms are followed by spotty hemorrhagic discolorations of the iris. The corneal epitheliums shows a roughened, lusterless surface, with areas of punctate staining demonstrable by instilling fluorescein. Severe exposure may cause the corned epitheliums to exfoliate. Slit lamp examinations will reveal clouding and edema of the corneal substance extending deep below the Bowman's membrane. Local necrosis of the cornea may rupture the globe.

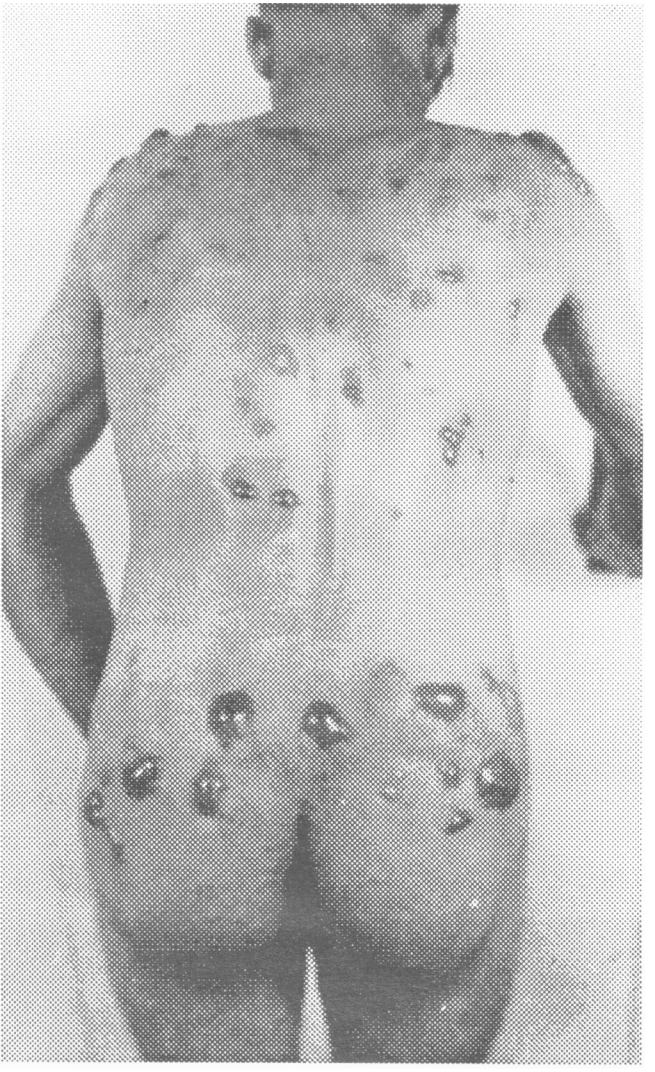


Figure 4-15. Casualty with widespread vesication.

The copyrighted picture is omitted. However, because it is essential to understanding this publicaiton, refer to printed version.

Click here for Figure 4-16.

b. Prognosis. The prognosis in contamination of the eye with any liquid HN is serious unless the agent is removed by immediate decontamination. Mild injury progresses to complete recovery in about 2 weeks; severe injury requires 9 to 12 weeks or longer. The cornea heals by vascularization, and scarring may be expected in severe cases. The iris is frequently left discolored and atrophied.

c. Treatment. The treatment is the same as for HD conjunctivitis (para 4-7 *b* (2)). In general, the lesions and symptoms are more severe, requiring intensive and early treatment. Spasms of the ciliary and orbicular muscles may require frequent instillation of atropine for relief of pain.

4-13. Effects of HN on the Skin

a. Pathology and Symptoms. In mild vapor exposures, there may be no skin lesions. After severe vapor exposure or after exposure to liquid HN, erythema may appear earlier than in HD contamination. There may be irritation and itching as with HD. Later, blisters may appear in the erythematous areas. The skin lesions are similar to those caused by HD.

b. Prognosis. Prognosis is similar to that of HD burns (para 4-8 *b).*

c. Treatment. If early decontamination has not been done, late decontamination should be performed even if erythema is already present and no liquid HN is visible on the skin. The rate of absorption of liquid HN through the skin is slower than that of HD. Therefore, to prevent systemic toxicity, decontamination should be done as early as possible (within 2 or 3 hours alter exposure even if it increases the severity of the local reaction).

4-14. Effects of HN on the Respiratory Tract

a. Pathology. The lesions caused by HN are similar to those caused by HD. The lesions decrease in severity down the respiratory tract from the point of entry. In the nose, larynx, and trachea, there may be swelling, erythema, and necrosis of the mucosa, followed by sloughing and fibrinous exudation. Small multiple ulcerations are commonly seen in the pharynx and tonsillar areas. Laryngeal edema and necrosis may lead to respiratory obstruction. In severe cases, the damage may extend to the bronchiole and alveoli.

Although pulmonary edema usually is not massive, secondary pulmonary infection is common.

b. Symptoms. The symptoms are the same as those due to HD; namely, delay in appearance, irritation of the nose and throat, hoarseness progressing to loss of voice, and a persistent cough. Fever, dyspnea, rhonchi, and moist rales may develop. Chemical pneumonitis may appear after the first 24 hours.

c. Prognosis. Mild inflammation of the trachea is likely to result in a persistent cough. Low-grade fever may persist a week or longer. The prognosis is grave if there is severe respiratory tract involvement. Late deaths due to pneumonia may occur.

d. Treatment. The treatment of respiratory tract involvement is the same as for HD (para 4-9 *c).*

4-15. Effects of HN on the Gastrointestinal Tract

Following oral administration or systemic absorption, HN injures the intestinal tract. The ingestion of 2 to 6 mg of HN causes nausea and vomiting.

4-16. Systemic Effects of HN

a. Pathology. The most specific effects of HN are on the hematopoietic and lymphoid tissues. These effects follow absorption from the intact skin, respiratory or gastrointestinal tract. In bone marrow, the degenerative changes can be detected within 12 hours and may progress to severe aplasia. The thymus, spleen, and lymph nodes involute rapidly, with necrosis and phagocytosis of their lymphocytes. This injury is demonstrable in the blood as a transient leukocytosis of a few hours duration, followed by severe lymphopenia, granulocytopenia, thrombocytopenia, and a moderate anemia. The blood picture may show little change other than lymphopenia for 5 to 10 days after exposure, when the white count may fall to 500 cells per cubic millimeter (mm^3) or lower. The various HNs differ in their ability to produce these changes.

b. Diagnosis. Diagnosis is based upon a history of exposure, a faint fishy odor on the skin and clothing, and the signs and symptoms described in paragraph 4-15 and *a* above.

c. Prognosis. Severe leukopenia, thrombocytopenia, and a hemorrhagic diathesis are grave manifestations.

d. Treatment. Frequent white blood cell and hematocrit determinations and examination of peripheral blood smears are necessary to institute proper treatment if anemia and thrombocytopenia occur. Severe vomiting and diarrhea may necessitate IV supplementation with balanced salt solutions or volume expanders. Sedatives, opiates, and atropine are to be used judiciously. The probability of infection with severe leukopenia may be significant and isolation of the patient to protect against infection is appropriate. If infection does occur, it should be vigorously treated with antibiotics.

Section III. ARSENICAL VESICANTS

4-17. Properties

a. These agents are organic dichloroarsines. The main ones are phenyldichloroarsine (PD) and chlorovinyldichloroarsine (L). Ethyldichloroarsine and methyldichloroarsine have also 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 arsenoxides, 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 permanent 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 cause sneezing and may produce irritation of the upper respiratory tract. More significant respiratory injury is unlikely from ordinary field concentrations of vapor.

4-18. Effects of Arsenical Vesicants on the Eyes

a. Pathology, Symptoms, and Prognosis. Arsenical vesicants cause severe damage to the eye. On contact, pain and blepharospasm occur instantly. Edema of the conjunctival and lids follows rapidly and closes 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. 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

conjunctival may follow very heavy exposure. All injured eyes are susceptible to secondary infection. Mild conjunctivitis duetoarsenical 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 for control of pain. 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 to combat infection after the first 24 hours. Sterile petrolatum applied to the lid margins will help prevent their sticking together. Irrigations of the eye should be sparing, employing only isotonic or slightly hypertonic solutions (example, 1 percent sodium chloride). Occlusive dressings or pressure on the globe must be avoided.

4-19. Effects of Arsenical Vesicants on the Skin

a. Pathology. Liquid arsenical vesicants produce more severe lesions of the skin than liquid mustard. Contamination of the skin is followed shortly by erythema, then by vesication which tends to cover the entire area of erythema. The surrounding halo of erythema is less noticeable than with mustard blisters, although the two are often indistinguishable. Microscopically, the blister roof is slightly thicker than the mustard blister roof, consisting of almost the complete thickness of the epidermis and showing more complete coagulation necrosis and less disintegrative necrosis than that of the mustard blister. The yellowish blister fluid is slightly more opaque than that of the mustard blister and, microscopically, contains more inflammatory cells. It contains a trace of arsenic but is nontoxic and nonvesicant. Within the corium and subcutaneous tissue, there is deeper injury to the connective tissue and muscle, greater vascular damage, and more severe inflammatory reaction than is exhibited in mustard burns. In large, deep, arsenical vesicant burns, there may be considerable necrosis of tissue, gangrene, and slough.

b. Symptoms. Stinging pain is felt usually in 10 to 20 seconds after contact with liquid arsenical vesicants. The pain increases in severity with penetration and in a few minutes becomes a deep, aching pain. Pain on contact with liquid arsenical vesicants usually gives sufficient warning so that decontamination may be begun promptly and deep burns avoided in conscious victims. After about 5 minutes of contact, there appears a gray area of dead epitheliums resembling that seen in corrosive burns. Erythema is like that caused by mustard but is accompanied by more pain. Itching and irritation persist for only about 24 hours whether or not a blister develops. Blisters are often well developed in 12 hours and are painful at first, in contrast to the relatively painless mustard blister. After 48 to 72 hours, the pain lessens.

c. Prognosis. The erythema of arsenical vesicants usually recedes more rapidly than the erythema of mustard and with less pigmentation. Small blisters heal in about the same time as those due to mustard. Large lesions may involve deep injuries which heal slowly and require skin grafts. After repeated burns, sensitization to arsenical vesicants occurs, as with mustard.

d. Treatment.

(1) Dimercaprol (British anti-lewisite (BAL)) ointment should be tried on contaminations of the skin which are seen before actual vesication has begun. Any protective ointment already on the skin must be removed before application of the BAL ointment because it destroys the latter. British anti-lewisite ointment is spread on the skin in a thin film, rubbed in with the fingers, allowed to remain at least 5 minutes, and later washed off with water. Occasionally, BAL ointment causes stinging, itching, or urticarial wheals. This condition lasts only an hour or so and should not cause alarm. Mild dermatitis may occur if BAL ointment is frequently applied on the same area of skin. Because of its dermatitis properties, BAL should not be used as a protective (barrier) ointment on unaffected skin.

(2) Some blistering is inevitable in most arsenical vesicant cases which come to the Medical Services. The treatment of the erythema, blisters, and denuded areas is identical with that for similar mustard lesions. A severe third degree burn involving a large surface area is similar to a thermal injury and must be managed by IV resuscitation to correct potential hypovolemic shock. Morphine and splinting of the affected parts may be necessary to relieve pain. Hospitalization is indicated when the involved body surface area is greater than 20 percent. Hospitalization may be indicated when the involved area is less than 20 percent but the depth of the skin involvement appears to be significant. The wound is debrided and treated with 10 percent mafenide acetate burn cream, or silver sulfadiazine topical burn cream.

4-20. Effects of Arsenical Vesicants on the Respiratory Tract

a. Symptoms. The vapors of arsenical vesicants are so irritating to the respiratory tract that conscious casualties will immediately put on a mask to avoid the vapor. No severe respiratory injuries are likely to occur except among the wounded who cannot put on masks and the careless who are caught without masks. The respiratory lesions are similar to those produced

by mustard except that, in the most severe cases, pulmonary edema may be accompanied by pleural effksion.

b. Prognosis. The prognosis is unknown because there have been no known human cases of poisoning by vapors of arsenical vesicants. Extrapolating from animal experiments, the prognosis probably is similar to that for respiratory injury by mustard.

c. Treatment. The treatment is a combination of that for mustard respiratory injury (para 4-9 *c)* and that for the systemic effects of arsenical vesicants (para 4-21 *c).*

4-21. Systemic Effects of Arsenical Vesicants

a. Pathology and Symptoms. Liquid arsenical vesicants on the skin, as well as inhaled vapor, are absorbed and may cause systemic poisoning. A manifestation of this is a change in capillary permeability, which permits loss of sufficient fluid from the bloodstream to cause hemoconcentration, shock, and death. In nonfatal cases, hemolysis of erythrocytes has occurred with a resultant hemolytic anemia. The excretion of oxidized products into the bile by the liver produces focal necrosis of that organ, necrosis of the mucosa of the biliary passages with peribiliary hemorrhages, and some injury of the intestinal mucosa. (Acute systemic poisoning from large skin burns causes pulmonary edema, diarrhea, restlessness, weakness, subnormal temperature, and low blood pressure in animals (hypovolemic shock)).

b. Prognosis. Burns severe enough to cause shock and systemic poisoning are life-threatening. Even if the patient survives the acute effects, the prognosis must be guarded for several weeks.

c. Treatment.

(1) *Indications for treatment.* The indications for systemic treatment, following exposure to arsenical vesicants by any route, are—

(a) A cough with dyspnea and frothy sputum, which may be blood tinged, and other signs of pulmonary edema.

(b) A skin burn the size of the palm of the hand, or larger, caused by a liquid arsenical vesicant which was not decontaminated within the first 15 minutes.

(c) Skin contamination by an arsenical vesicant covering 5 percent (about 1 square foot) or more of the body surface, in which there is evidence of immediate skin damage (gray or dead-white blanching of the skin), or in which erythema develops over the area within 30 minutes.

(2) *Types of treatment.* The following two types of treatment may be used:

(a) Local neutralization on and within the skin by a liberal application of BAL ointment. The affected skin is to be left covered with a layer of ointment. Remove any other protective ointment before treatment with BAL ointment.

(b) Intramuscular injection of BAL in oil (10 percent).

(3) *Dose of BAL in oil.*

(a) An immediate IM injection of BAL in oil (10 percent) is given deep into the muscles of the buttocks. Take every precaution against injecting into a blood vessel. Dosage must be adjusted to the estimated weight of the patient (0.5 ml per 25 pounds, up to a maximum of 4.0 ml) as follows:

125 pounds	2.5 ml
150 pounds	3.0 ml
175 pounds	3.5 ml
200 pounds and over	4.0 ml

(b) Intramuscular injection of BAL in oil should be repeated at different sites in the buttocks at 4, 8, and 12 hours after the initial injection, for a total of four equal doses.

(c) If pulmonary symptoms or other evidence of severe arsenical poisoning are present, the interval between the first and the second dose may be shortened to 2 hours. In severe cases, subsequent half doses should be given at the rate of one injection per day for 3 to 4 days.

(d) In toxic patients, liberal fluids by mouth (or IV if necessary) and high-vitamin, high-protein, high-carbohydrate diets are indicated. If shock is present, the usual supportive measures (such as IV administration of electrolyte solutions, blood transfusions, or other vascular volume expanders) are indicated.

(4) *Symptoms caused by BAL in oil.* Symptoms caused by BAL in oil appear 15 to 30 minutes after injection and last about 30 minutes. Unless unduly severe or prolonged, they do not contraindicate the full course of treatment. The symptoms may include—

- A feeling of constriction in the throat.
- A sense of oppression in the chest.
- Burning sensation of the lips.
- Mild lacrimation.
- Slight reddening of the eyes.
- Dryness of the mouth and throat.
- Generalized muscular aching.
- Abdominal pain.
- Mild to moderate tenderness and increased muscle tonus at the injection site.
- Mild restlessness and nervousness with sweating of the hands.
- Apprehension.
- Mild nausea and vomiting on eating.
- A transient rise in blood pressure.

4-22. Mixtures of Blister Agents

Arsenical vesicants such as L or PD are often mixed with mustard. These mixtures do not produce more severe lesions than either agent alone, but tend to confuse and make diagnosis difficult.

Section IV. PHOSGENE OXIME

4-23. Properties

a. Phosgene oxime (CX) (chemical name dichloroformoxime) is an example of the class of chemical agents called urticants (or nettle gases). These agents are primarily irritants to skin and mucous membranes, but they differ from mustard by producing an immediate sensation of pain. This pain may vary from a mild prickling to a feeling resembling that caused by a severe bee sting.

b. Phosgene oxime has a disagreeable, penetrating odor. It may appear as a liquid or as a colorless, low-melting point (crystalline) solid, readily soluble in water. Phosgene oxime has an appreciable vapor pressure. A powerful irritant, it is especially effective as a liquid.

4-24. Symptoms and Course of Lesions of Phosgene Oxime

Phosgene oxime is violently irritating to the mucous membranes of the eyes and nose. Even very low concentrations of it can cause lacrimation. Any exposure to liquid or vapor which produces pain will also produce skin necrosis at the site of contact. Within 30 seconds, the area of contact becomes blanched and is surrounded by an erythematous ring. This is followed by the appearance of a wheal within the next half hour. At about 24 hours, the original blanched area acquires a brown pigmentation. At 1 week, an eschar forms in the pigmented area; and at about 3 weeks, the eschar generally sloughs. Itching may be present throughout the course of healing. Some 20 percent of those exposed to CX may be expected to show healing delayed beyond 2 months.

4-25. Protection from Phosgene Oxime

A properly-fitting protective mask protects the respiratory system. The field protective mask, hood, and chemical protective overgarment protect the body.

4-26. Self-Aid

Because of the rapid reaction of CX with tissue, decontamination will not be entirely effective after pain has been produced. Use the M291 Skin Decontaminating Kit for skin decontamination. If the M291 kit is not available, flush the contaminated area as rapidly as possible with copious amounts of water to remove any CX which has not yet reacted with tissue.

4-27. Treatment for Phosgene Oxime Injury

Treat as any other ulcerated necrotic skin lesion with due consideration of other supportive measures, as indicated.

Section V. DISPOSITION OF PERSONNEL WITH BLISTER AGENT BURNS

4-28. General

a. Applicability. This information should be used as guidance by medical personnel confronted with casualties produced by blister agents. It assists medical personnel in the forward area in the disposition, rather than the treatment, of casualties. Therapy for blister agent burns is given in Sections II through IV.

b. Locations of Burns in World War I Allied Troops. During 1917-1918, many Allied troops with mustard burns were evacuated needlessly from the front lines. The analysis below shows the locations of mustard burns among 6,980 cases from World War I. (The incidence of permanent blindness among the eye cases was low.)

Location of burns	*Percent*
Eyes	86
Respiratory	75
Scrotum	42
Face	27
Anus	24
Legs	11
Buttocks	10
Hands	4
Feet	1.5

c. 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.

d. Types of Blister Agent Burns.

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

(a) The first 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 as soon as possible with or without treatment.

(b) The second type consists of the totally disabled who are incapable of either offensive

or defensive activity regardless of the urgency of the military situations. These individuals will be classed as casualties, promptly treated, and evacuated. Examples are injuries causing total disability and blindness, vesication of extensive areas of the trunk, or vesication of an entire limb.

(2) 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 section is confined to typical injuries within this group. In disposing of these cases, the medical officer will be influenced not only by the severity of the lesions, but also by the military situation, plus the general physical and mental condition of the individual and his military occupational skill.

e. Differentiation Among Injuries According to Agent. For simplicity, no effort is made here to differentiate between 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 Sections II through IV.

4-29. Eye Injuries

a. 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 (which varies with the degree of exposure) precedes the onset of symptoms. This period ranges from 4 to 12 hours after mild exposure and 1 to 3 hours after severe exposure.

b. Classification of Lesions of the Eyes. Eye lesions produced by mustard are divided into the following types.

(1) *Mild.* Of all the cases in World War I, 75 percent had mild burns of the eyes. The mild symptoms include itching, lacrimation, and a sensation of grit in the eye, followed by burning and sometimes by photophobia. There is a hyperemia of both the palpebral and bulbar conjunctival. The reaction in the latter usually begins as a band-shaped area running transversely across the eye with normal white bulbar conjunctival above and below it. Edema of the lids may also be present. Hospitalization is seldom required and recovery occurs in 1 to 2 weeks. Such cases are not classified as casualties.

(2) *Moderate.*

(a) In this group there is complete closure of the eyes from a combination of spasm and swelling of the lids about 3 to 6 hours after exposure. Blepharospasm and blurring of vision develop. There is marked hyperemia and edema of the conjunctival with a prominent interpalpebral band, edema of the lids, mild iritis, and edema of the epitheliums of the cornea producing a roughened appearance like that of an orange peel. The blepharospasm and edema of the lids may be too severe to enable the patients to open their eyes and, so, they may believe they are blind. Miosis occurs early.

(b) A mucoserous discharge is usually present and, although sterile in the early stage, it may cause the lids to stick together, resulting in accumulation of secretions in the conjunctival sac and predisposition to infection. Personnel in this condition are temporarily blind and will be evacuated as casualties. Early and prolonged hospitalization is required, with transfer to the care of an ophthalmologist when possible. Recovery occurs in 1 to 6 weeks, usually without loss of vision. Return to duty will depend upon the extent of residual corneal injury, photophobia, and blepharospasm.

(3) *Severe.*

(a) In this group the latent period is short, lasting 1 to 3 hours. There is deep ocular pain and headache, both of which may be severe, in addition to severe blepharospasm and blurred or dimmed vision. There is marked hyperemia and edema of the conjunctival with a blanched area of ischemic necrosis in the interpalpebral portion, chemosis, and edema of the lids, which the patient cannot open. The epitheliums and stroma of the cornea are damaged. Surface epitheliums is hazy in the early stage and will stain extensively or, in a punctate manner, with fluorescein within 24 hours. After 24 to 48 hours there is also edema of the stroma of the cornea and a deeper haze becomes apparent. Iritis and mucoserous discharge are also present. If the damage is progressive, there may be dense corneal opacification with deep ulceration and vascularization from the limbus. The cases with corneal ulcer heal slowly and may have relapses, some may present perforations into the anterior chamber. These casualties require hospital care and should be evaluated at the earliest possible moment.

(b) Droplets of a liquid blister agent contaminating the eye may produce similar effects. One eye alone may be involved or may be affected more severely than the other. In contrast to droplets of mustard alone, droplets of L or mixtures of L and mustard cause immediate, painful spasm of the lids.

(c) In disposing of eye casualties, medical officers must assure themselves that mild symptoms will not develop into severe inflammation

and temporary blindness within a few hours. Reference to time of exposure and rate of development of symptoms will guide them. If the effects are increasing rapidly, it is advisable to evacuate the case in anticipation of development of disability within the next few hours. Symptoms usually reach a maximum within 6 to 12 hours following exposure.

c. 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. Several hours following exposure of the eyes to mustard, it may be possible to determine whether personnel can remain on duty or will require evacuation.

4-30. Effects on the Respiratory System

a. Circumstances of Exposure. Most respiratory lesions are the result of prolonged exposure to relatively low concentrations of vapor. Severe casualties may result from unrecognized exposure to strong concentrations of mustard vapor. A fatiguing effect on the sense of smell may follow exposure to low concentrations of mustard, thus minimizing detection. Severe lesions occur in individuals who cannot mask, such as unconscious casualties and those with severe injuries to the face or upper extremities.

b. Latent Period. The effects of exposure upon the respiratory tract are characterized by a long latent period before the onset of symptoms, 18 to 36 hours intervening. Because the eyes are more sensitive to the agent than the nasal mucosa but are exposed simultaneously, respiratory effects should be expected to follow vapor burns of the eyes (and face) in unmasked personnel.

c. Mucous Membranes. The local action of mustard vapor on skin and eyes is matched by a similar effect on the mucous membranes of the respiratory tract. Most of the inhaled vapor is absorbed in the larger respiratory passages, including the bronchi, and very little remains to injure the pulmonary parenchyma.

d. Nose. In the nose the earliest visible effect is hyperemia and edema of the mucosa. This is associated with a profuse, thin, mucopurulent discharge. Degenerative changes in the epitheliums, varying in degree according to the extent of exposure, range from small discrete ulcerations to extensive sloughing. Nosebleeding is rare. Nasal injury seldom occurs alone and, as such, is usually a cause for hospitalization.

e. Pharynx. Acute inflammation of the pharynx usually appears 1 to 3 days after exposure to mustard vapor, although there may be a delay of as much as a week. There is mild dryness and soreness of the throat, aggravated by swallowing but rarely accompanied by regional lymphatic enlargement. Pharyngeal and laryngeal lesions may develop without significant nasal involvement, especially in mouth breathers. Upon inspection, the palate, uvula, tonsils, and pharynx are swollen. Multiple whitish ulcerations may appear, varying in size according to the severity of exposure. Pharyngeal injury is unlikely to occur alone. Secondary infection generally results in regional adenitis.

f. Larynx. Laryngeal involvement commonly results from inhaling mustard vapor, the lesions resembling those of the pharynx. Hoarseness, sometimes progressing to loss of voice, may last 3 to 6 weeks, and in rare instances, longer. This type of lesion alone may not require hospitalization, but it is almost invariably associated with more extensive injury to the respiratory tract.

g. Trachea and Bronchi. In the trachea and bronchi, a similar necrotizing and inflammatory process follows contact with mustard vapor. The exudative process results in the formation of a fairly thick, tenacious pseudodiphtheritic membrane in the larynx, trachea, and large bronchi. It may form a cast of the involved parts sufficient to prove fatal. This condition requires early hospitalization. Milder cases, however, have small ulcerations with hyperemia and edema of the mucosa and hypersecretion of mucous. Respiratory embarrassment, cough, tachypnea, and cyanosis are signs warranting prompt hospitalization.

h. Pulmonary Parenchyma. The action of mustard on the lung may cause chemical pneumonitis. Secondary infection may lead to lobular or lobar consolidation, the course being dominated by the characteristics of the type of pneumonia. Injury due to mustard in no way affects the treatment of the secondary infection. Antibiotics and supportive measures should be used as appropriate.

4-31. Cutaneous Burns

a. Evaluation of Cutaneous Burns. The following observations resulted from evaluation of lesions that have most generally led to disability of personnel exposed to blister agents during field trials and then participated in simulated combat exercises, obstacle course tests, and marches.

(1) Widespread vesication of the trunk produced casualties.

(2) Vesication localized in particular areas of the body produced casualties.

(3) Burns produced by high doses of the blister agent vapor to masked personnel (especially in tropical climates) cause severe casualties. These casualties are produced partly by edema and vesication of the skin and partly by constitutional reactions such as nausea, vomiting, and prostration.

(4) Burns produced by doses of vapor low enough to cause only such skin reactions as mild erythema, edema, burning, and itching usually do not produce casualties.

(5) The stage of development of the lesion must be considered when classifying an individual as a casualty or noncasualty.

b. Trunk and Neck.

(1) *Extensive vesication of the trunk.* All the cases considered under this heading should be evacuated promptly.

(a) Extensive vesication may occur over a large part of the trunk. Intervening areas of skin may be erythematous with pinpoint vesication. These burns are more likely to occur on the back than anteriorly. Some protection is afforded anteriorly by equipment such as webbing and ammunition pouches. The front of the uniform also gives some anterior protection because it does not cling to the body.

(b) Extensive vesication may be followed by fever, nausea, and vomiting. These conditions tend to occur more readily in tropical climates.

(c) Secondary bacterial infection may complicate the clinical course. The medical officer in a forward position is not likely to see infection of vesicated areas because such cases will have been evacuated before secondary infection develops.

(2) *Localized vesication of the trunk.*

(a) Vesication occurring within the natal cleft (between the buttocks) usually requires evacuation of the casualty. Walking becomes difficult, defecation is painful, and dressings require frequent changing. The lesion is usually most intense at the upper end of the cleft. Vesication of the buttocks usually results from sitting on contaminated ground or wearing contaminated trousers for prolonged periods. The vesicated area may extend forward across the perineum to involve the scrotum and the penis.

(b) Trivial burns, such as mild erythema affecting the natal cleft, are not of casualty severity. However, these burns require careful attention because walking or running aggravates the lesions and may break down injured skin.

(c) Single discrete blisters on the buttocks away from the natal cleft do not produce casualties.

(d) Blisters on the trunk generally require protective dressings to avoid friction of clothing. Medical officers must decide whether or not dressings should remain in position during regular duty.

c. Burns Caused by High Doses of Vapor. After exposure to a high dose of mustard vapor, especially under tropical conditions, nausea, vomiting, and symptoms of collapse are usually evident before erythema completely develops. It is important to note that this occurs also among personnel who are masked during exposure. Constitutional symptoms may persist several days, during which burns will increase in severity. Cases of this type should be classed as casualties. Severe vapor burns of the trunk give a generalized erythema but include pale gray areas that eventually vesicate or become necrotic. It is common to see patches of unaffected skin as a result of protection by overlying equipment.

d. Burns Caused by Low Doses of Vapor. Mild vapor burns cause erythema, itching, and irritation but do not produce casualties. The medical officer should always consider the interval after exposure in relation to the severity of the burn. Mild lesions may represent early phases of severe exposure to vapor. When the period of lapse since exposure is uncertain, rapid development and presence of constitutional symptoms may help to determine the severity.

e. Sensitization Due to Multiple Exposures to Mustard.

(1) Watch for the characteristic appearance of "reexposure" burns. This manifestation may occur in individuals as a result of exposure to mustard 1 to 3 weeks (or more) previously. A small percentage of these casualties will become sensitized to the agent and will react differently, both qualitatively and quantitatively, upon reexposure.

(2) Sensitization will be followed by a more rapid onset of symptoms upon reexposure. Erythema, with or without edema, and pronounced itching and burning usually appear within 1 hour. Lower concentrations of mustard are required to produce effects in a sensitized individual than in a nonsensitized. When erythema and edema result from exposure to a low dose, they generally develop rapidly and subside within 2 to 3 days. Also, vesication heals more rapidly in the sensitized individual.

(3) One of the most frequent manifestations of reexposure in sensitized personnel is the development of a morbilliform rash. Another characteristic reaction is the appearance of eczematoid dermatitis surrounding old lesions, whether or not they are healed. This may last for several days and resembles dermatitis venenate (from poison ivy). Similar phenomena due to sensitization have been known to occur with L and with the nitrogen mustards.

f. Arms.

(1) After treatment, most service members with blister agent injuries of the arms are permitted to continue with their duties. Vesication, when localized, produces little or no disability.

(2) Extensive vesication involving the axillae and the elbows, volar or dorsal aspects, partially impairs the movement of the limbs at those joints. Edema of the surrounding tissue tends to further immobilize the extremities. The dorsal aspects of the elbow and forearm are common sites of severe burns because these parts touch contaminated ground when service members are firing in the prone position. Cases of this type should be evacuated.

(3) Widespread vesication of the arms results in partial disability. Cases of this type should be evacuated.

g. Hands.

(1) Blister agent burns of the hands are often

encountered. These burns tend to cause a degree of disability out of proportion to the size of the lesions. Considerable care and judgment are required in correct disposition.

(2) The palms are more resistant to vesication but not entirely. Blisters affecting the palms are characteristically painful and slow to heal.

(3) A solitary lesion of limited extent may result in little or no disability if treated properly.

(4) Burns from liquid vesicant on the dorsum of the hand result in severe local reactions characterized by intense edema of the backs of the hands and fingers. Pain is characteristic and is intensified by movement of the fingers or wrist. These cases should be regarded as casualties. An individual exposed within the previous 24 hours and reporting for treatment, with apparently tribal blisters, may be totally incapacitated the following day. Sharp erythema of the dorsum of the hand, with beginning vesication 12 to 24 hours after exposure, indicates a lesion that will progress to extensive vesication and edema. Under such circumstances, the individual should be evacuated when first seen.

(5) More commonly, the lesions consist of scattered small vesicles and limited areas of erythema. These lesions can be protected satisfactorily and the individuals returned to duty.

(6) Exposure to vesicant vapor produces diffuse erythema of the dorsum of the hand and wrist. Higher doses cause edema and vesication as well; cases of this type require evacuation.

h. The Lower Extremities.

(1) When the lower extremities are involved, the knees are the most common sites of burns from liquid vesicant. These lesions and those of the ankles often result in incapacitation by interfering with locomotion. Movement of the joints tends to aggravate existing lesions by increasing edema. A further disabling factor is introduced by the wearing of firm dressings applied to mobile joints.

(2) Vesication often spreads over the kneecaps, upward onto the thighs, and down toward the feet. These burns tend to be extensive and are associated with edema often extending halfway up on the thigh and down the leg. Medical officers should evacuate casualties presenting such lesions.

(3) In general, burns of the leg are more incapacitating than burns of the thigh.

(4) It has been shown that the presence of many superficial blisters on the legs and thighs alone is not enough to make a service member incapable of carrying out routine military duties. Individuals with such lesions, having protective dressings, were able to take part in daily marches and routine gun drills. In disposing of these cases, the medical officer will consider the mental and physical status of these individuals, their motivation, their military occupational specialty, and the tactical situation at the time. Such cases are in the category of partial disability. After suitable dressings have been applied, service members with high morale and robust physiques may be returned to duty.

(5) A relatively small blister or group of blisters situated in the popliteal area may reduce the efficiency of service members so much that they may require evacuation. This is due to aggravation of the lesions by movement of the limbs and interference with ambulation. However, blisters affecting this area are not necessarily casualty producing. (Inflammation, edema, infection, and lesions on other parts of the body should be considered when deciding upon the disposition of an individual.) Available evidence indicates that the mustard blister, size for size, is potentially more incapacitating than a blister from L. This results from the tendency of mustard blisters to be associated with erythema and edema, while the L blisters usually cause less local reaction.

(6) Vesicant lesions develop also near the ankles at the tops of the shoes. Blistered areas occurring at such unprotected points are associated with severe pain due to circulatory impairment and tense edema of the leg. These cases should be evacuated.

(7) Vapor burns of the legs tend to be most aggravated in the popliteal spaces. Pinpoint vesication is often found here. Higher doses cause intense erythema with scattered areas of vesication over the entire surface of the leg. Such lesions are invariably casualty producing and are generally accompanied by severe burns elsewhere, frequently with severe systemic effects.

(8) Mild vapor burns of the legs produce irritation and itching common to all widespread vapor burns. While these effects are troublesome, they are not casualty producing. Service members with mild vapor burns should be returned to duty.

(9) Extensive vesication of the feet is uncommon. The soles are protected by shoes and are more resistant to vesication. Burns on the dorsal aspect of the foot are often associated with local reactions like those seen on the backs of the hands. Individuals with these burns, especially if widespread over the foot, find it difficult or impossible to wear shoes and will require evacuation. Small discrete blisters may be of noncasually significance. These blisters may be effectively protected so as to allow wearing of shoes and walking with little discomfort.

i. The Genitalia.

(1) The genital region, in addition to the eyes and the respiratory tract, is highly sensitive to blister agent burns. In World War I such burns produced many casualties. The majority of these burns were caused by vapor. Despite present methods of protection against blister agents, medical officers (especially in tropical areas) may be confronted with many such burns.

(2) Vapor is a more common cause of burns affecting the male genitalia than a liquid agent. Erythema may not be conspicuous. The most prominent feature of the burn is the edema involving the penis and scrotum. Fluid accumulates most readily in the prepuce, distending its entire circumference and forming a characteristic semitranslucent ring around the corona. In more severe cases, the entire body of the penis becomes edematous. Female genitalia are affected in a similar manner, the most prominent feature being edema of the labia. In severe burns, fluid may also accumulate in the labia.

(3) These lesions cause apprehension as well as physical discomfort. Occasionally, vesication is superimposed on the edema. Spotty ulceration is not infrequent at the tip of the prepuce where it may become secondarily infected. In severe cases associated with marked edema, retention of urine may result from both mechanical and reflex effects.

(4) In mild cases, objective changes of the scrotum often tend to pass undetected due to the normal pigmentation, elasticity, and looseness of the skin. Even considerable edema may not be enough to reveal its presence. In severe cases the scrotum may become grossly enlarged. The rugae may be partly or completely obliterated. Pinpoint vesication may occur, usually after a lapse of a few days. The scrotum tends to break down resulting in small, painful ulcers and fissures.

(5) Burning is the most prominent subjective symptom in involvement of the genitalia. Apprehension and anxiety are distressing during the presence of the objective changes described in (3) and (4) above. As edema decreases, itching starts and may persist long after the acute effects have subsided. Sometimes this itching is intolerable. The scrotum may continue to crack and ulcerate for a considerable period, causing pain and irritation.

(6) Mild exposure of the genital region typically is followed by a delay in the development of symptoms, often for as long as 4 to 10 days.

(7) Patients with mild burns without edema or vesication, but who complain of irritation and burning, may be safely returned to duty following treatment. In disposing of mild burns of the genitalia, medical officers must assure themselves that the symptoms are not too early to be judged with finality. Severe cases should be evacuated on the basis of the apprehension that may be suffered as well as the physical discomfort involved.

j. Systemic Effects of Cutaneous Burns.

(1) Severe systemic effects due to blister agents probably will be encountered only with disabling skin lesions. The medical officer should be familiar with the signs and symptoms. These include anorexia, nausea, vomiting, depression, and fever, and are far more prone to occur in hot than in temperate climates. Malaise and nausea generally are the first reactions and may progress either to mild, transient vomiting or to severe, persistent vomiting and retching. Anorexia may be the only complaint in mild reactions. The actual time of onset of symptoms is 4 to 12 hours after exposure, and symptoms often occur before skin injury is manifest. No rule can be given for the duration of systemic symptoms, although casualties usually have recovered from severe vomiting within 24 to 36 hours. Anorexia and nausea may persist for a longer time.

(2) The temperature may remain elevated for several days. Mental depression may follow mustard burns and persist for several days.

(3) Service members with systemic reactions will generally be casualties, particularly in view of the probability of associated extensive skin burns. Such cases should be evacuated quickly.

k. Secondary Bacterial Infection in Blister Agent Burns. This paragraph considers the problem of secondary bacterial infection after blister agent injuries only as it influences the disposition of affected personnel in forward positions. For management and treatment of such cases, see sections II through IV.

(1) Secondary bacterial infection may result if adequate wound care is not given. Compared to the incidence of infection in thermal and traumatic wounds, the incidence of sepsis in mustard lesions is remarkably low, according to observations made at experimental installations.

(2) Secondary infection becomes manifest several days after injury. Medical officers are not likely to see secondary infection with extensive blister agent burns in the front lines because severe cases will have been evacuated early.

(3) Infection of small lesions does not require evacuation. Infection of multiple lesions is likely to be an indication for evacuation, particularly if constitutional effects are associated. Infection is particularly disabling when it involves the feet, hands, genitals, or tissue overlying the joints of the limbs.

(4) Secondary infection is more likely to occur in severe, rather than mild, vapor injury to the respiratory tract. Severe respiratory symptoms will almost always be associated with severe eye effects. Respiratory lesions may not develop for several days, and by then the individual should have been evacuated as an eye casualty.

(5) Secondary infection is uncommon as a sequel to mild degrees of mustard conjunctivitis and ordinarily would not prevent an individual from continuing duty.

(6) Mild conjunctival burns may be associated with pharyngitis, laryngitis, and tracheitis, increasing in severity for several days. Occasionally, more extensive respiratory infection may ensue.

CHAPTER 5
LUNG-DAMAGING AGENTS (CHOKING AGENTS)

5-1. General

a. Chemical agents which attack lung tissue, primarily causing pulmonary edema, are classified as lung-damaging agents (choking agents). They include phosgene (CG), diphosgene (DP), chlorine, and chloropicrin (PS). Best known of these agents is CG. Agents in this class are called lung-damaging agents because irritation of the bronchi, trachea, larynx, pharynx, and nose may occur and, with pulmonary edema, contribute to the sensation of choking. Blister agents and certain systemic agents also may injure the respiratory tract. Since the action of CG is typical of the lung-damaging agents, it is used as the example in this chapter.

b. Persons exposed to CG need not be withdrawn during combat, unless signs of pulmonary distress appear. The physician at the supporting MTF should so advise the responsible commander.

5-2. Protection

The protective mask or a gas-particulate filter unit (collective protector) gives protection against lung-damaging agents.

5-3. Properties of CG

At ordinary temperatures and atmospheric pressure, CG is a colorless gas. The boiling point of CG is 47 °F (8.2 °C); it is extremely volatile making it a nonpersistent chemical agent. The vapor density of CG is 3.4 times that of air. Phosgene may remain for long periods of time in trenches and other low-lying areas. In low concentrations, CG has a smell resembling new mown hay. Phosgene is readily soluble in organic solvents and fatty oils. In water, CG is rapidly hydrolyzed with the formation of hydrochloric acid and carbon dioxide.

5-4. Pathology

Aside from mild conjunctival irritation, the direct effects of exposure to CG are confined to the lungs. Changes in other organs are secondary to the pulmonary alterations. The outstanding feature of CG poisoning is massive pulmonary edema. This is preceded by damage to the bronchiolar epitheliums, development of patchy areas of emphysema, partial atelectasis, and edema of the perivascular connective tissue. The trachea and large bronchi are usually normal in appearance. This contrasts with the findings in chlorine and PS poisoning in which both structures may show serious damage to the epithelial lining with desquamation. The lungs are large, edematous, and darkly congested. Edema fluid (usually frothy) pours from the bronchi and may be seen escaping from the mouth and nostrils. With exposure to very high concentrations, death may occur within several hours. In most fatal cases, pulmonary edema reaches a maximum in 12 hours, followed by death in 24 to 48 hours. If the casualty survives, resolution commences within 48 hours, and in the absence of complicating infection, there may be little or no residual damage.

5-5. Symptoms

During and immediately after exposure, there is likely to be coughing, choking, a feeling of tightness in the chest, nausea, and occasionally vomiting, headache, and lacrimation. The presence or absence of these symptoms is of little value in immediate prognosis. Some patients with severe coughs fail to develop serious lung injury, while others with little sign of early respiratory tract irritation develop fatal pulmonary edema. There may be an initial slowing of the pulse, followed by an increase in rate. A period follows during which abnormal chest signs are absent and the patient may be symptom-free. This interval commonly lasts 2 to 24 hours but may be shorter. It is terminated by the signs and symptoms of pulmonary edema. These begin with cough (occasionally substernally painful), dyspnea, rapid shallow breathing, and cyanosis. Nausea and vomiting may appear. As the edema progresses, discomfort, apprehension, and dyspnea increase and frothy sputum develops. Rales and rhonchi are audible over the chest, and breath sounds are diminished. The patient may develop shock-like symptoms, with pale, clammy skin, low blood pressure, and a feeble, rapid heartbeat.

5-6. Diagnosis

Irritation of the nose and throat by CG may be mistaken for upper respiratory tract infection. Difficulty in breathing and complaint of tightness of the chest may suggest nerve agent poisoning or an acute asthmatic attack. Noncardiac pulmonary edema is like that produced by other agents and may be confused with the edema associated with heart failure. Diagnosis can only be established with certainty from a definite history of exposure to CG.

5-7. Prognosis

Prognosis during the acute phase should be guarded because of the insidious nature of the poisoning. Most deaths occur within the first 48 hours. The few which occur later are due largely to bronchopneumonia. Casualties from CG who survive more than 48 hours usually recover without sequelae. Exposure to CG rarely results in development of chronic bronchitis and bronchiectasis. Long-term pulmonary effects are generally the result of intercurrent infection or other exposures. The majority of U.S. Army deaths from CW agents used in World War I was caused by CG.

5-8. Self-Aid

a. The protective mask should be put on immediately when any of the conditions described in paragraph 1-7 *a* exist. Other indications of a CG attack are—

(1) Odor like newly mown hay. (Do not rely upon odor as an indication of a chemical attack.)

(2) Irritation of the eyes.

b. If some CG has been inhaled, normal combat duties should be continued unless there is difficulty in breathing, nausea and vomiting, or more than the usual shortness of breath during exertion.

c. If any of the above symptoms occur, there should be quiet rest until medical evacuation is accomplished.

5-9. Treatment

a. Rest and Warmth. A casualty exposed to a lung-damaging agent should be kept at rest until the danger of pulmonary edema is past, but the operational situation may prevent this. Tightness of the chest and coughing should be treated with immediate rest and comfortable warmth. The casualty should be evacuated in a semiseated position if dyspnea or orthopnea make a supine posture impractical. Mandatory evacuation by litter in cases of significant respiratory involvement has been advocated.

b. Sedation. Sedation should be used sparingly. Codeine in doses of 30 to 60 mg may be effective for cough. Restlessness may be a manifestation of hypoxia; therefore, only cautious use of sedatives is advised. Use of sedatives should be withheld until adequate oxygenation is assured and facilities for possible respiratory assistance are available. Barbiturates, atropine, analeptics, and antihistamines are all contraindicated.

c. Oxygen. Hypoxemia may be controlled by oxygen supplementation. Early administration of positive airway pressure (intermittent positive pressure breathing (IPPB), positive end-expiratory pressure (PEEP) mask ("PEEP mask"), or, if necessary, incubation with or without a ventilator) may delay and/or minimize the pulmonary edema and reduce the degree of hypoxemia.

d. Antibiotics. Antimicrobial therapy should be reserved for acquired bacterial bronchitis/pneumonitis. Prophylactic therapy is not indicated.

e. Steroids. After exposure to a sufficiently high dose of CG or similar agent, pulmonary edema will follow. Administration of corticosteroids has been recommended, but proof of their beneficial effects is lacking. It has been suggested that, when steroid treatment is initiated within a very short time of the exposure, this therapy may lessen the severity of the edema. Rest, warmth, sedation, and oxygen are also of great importance, as indicated above. Doses of steroids used are much greater than those used in asthma. Treatment for exposure to a lung-damaging agent or similar compound (except for zinc chloride smoke, for which an extended regimen is essential) should be judged on the basis of precautionary treatment for what seems a mild but possibly dangerous exposure, and definitive treatment for an exposure which is definitely expected to endanger life. Two regimes are in use: one using dexamethasone-sodium phosphate and the other using beclomethasone dipropionate or betamethasone valerate. In either case, treatment should be started as soon as possible, ideally within 15 minutes of exposure.

(1) Using dexamethasone-sodium phosphate, the procedure is as follows:

(a) Treatment should start at the earliest possible moment with the inhalation of the steroid from an inhaler. Four puffs (or strokes) should be inhaled at once. This should be followed by 1 puff every 3 minutes until any sense of irritation is overcome. After this, 5 puffs should be inhaled every 15 minutes until a total of 150 puffs have been inhaled (that is, the entire contents of one standard inhaler are finished). When this stage has been reached, 1 puff should be inhaled hourly during the day, and, as a preparation for sleep, 5 puffs should be inhaled every 15 minutes until a total of 30 puffs have been reached. This regimen should be repeated each day for at least 5 days or longer if there are any abnormalities, including indications of pulmonary edema or infiltrates on the chest x-ray, after which treatment may be withdrawn. If recovery is slow, the dosage may be reduced to 6 puffs a day and continued until recovery is complete.

(b) Definitive treatment for exposures which definitely endanger life should include the above regime of inhaled steroid treatment, supplemented by systemic steroids as follows:

Day 1	1,000 mg prednisolone, IV
Day 2 and 3	800 mg prednisolone, IV
Day 4 and 5	700 mg prednisolone, IV

Beginning with day 6, the dose of systemic steroids should be reduced as soon as possible, provided that the chest x-ray remains clear. If further early systematic treatment is necessary, adrenaline may be

given in the acute stage of bronchial spasm and oxygen may be necessary. Treatment of severe cases is very difficult because of tissue damage. Absolute rest and administration of oxygen are fundamental. Expectorants may also be used. Bronchopneumonia is treated by antibiotics.

(2) Using beclomethasone dipropionate or betamethasone dipropionate, the procedure is as follows: (The differences occur due to the various absorption characteristics of these steroids. Limited systemic therapy is necessary, even for precautionary treatment.)

(a) Treatment should commence as soon as possible with the inhalation of 10 puffs of the steroid from an inhaler. Five puffs should be given each hour for the next 10 hours. Then 1 puff should be given hourly for the next 24 hours for as long as inhalational therapy is considered necessary (at least 5 days). Systemic therapy is needed even for precautionary treatment during the first 24 hours and should commence as soon as possible with the IV injection of 20 mg of betamethasone or the equivalent dose of another systemic steroid. This dose should be repeated IV or IM every 6 hours for at least the first 24 hours. During the next 5 days, inhalation therapy should be continued but systemic therapy may be reduced based on clinical response and chest x-ray improvement.

(b) Definitive treatment may call for longer periods of systemic therapy.

(c) Prednisolone, betamethasone, or methylprednisolone are preferred to other steroids for systemic use, as there is evidence that these steroids do not interfere with collagen metabolism. Antibiotic coverage should be considered with these high doses of steroids in patients predisposed to pulmonary infection complications. Side effects of high steroid dosages should be accepted provided they do not themselves endanger life. Any indication of pulmonary fibrosis will necessitate antifibrotic treatment.

5-10. Convalescent Care

Absolute rest must be continued until the acute symptoms have disappeared. Individuals must be closely monitored for signs of recovering from the acute effects of the CG poisoning. When the acute symptoms disappear, individuals should be—

a. Gotten out of bed.

b. Hospitalized as short a time as possible.

c. Encouraged and trained to resume physical exertion to minimize neurasthenic symptoms which may be a major disabling feature in these patients.

CHAPTER 6
BLOOD AGENTS (CYANOGENS)

6-1. General
Blood agents produce their effects by interfering with oxygen utilization at the cellular level. Inhalation is the usual route of entry. Hydrogen cyanide (AC) and cyanogen chloride (CK) are the important agents in this group. Cyanogen chloride also has a choking effect (para 7-1). These agents can be dispersed by artillery shell, mortar shell, rocket, aircraft spray, and bomb. All blood agents are nonpersistent.

6-2. Protection
The protective mask with a fresh filter gives protection against field concentrations of blood agent vapor. For protection, MOPP 4 is needed when exposed to or handling liquid AC.

6-3. Properties
a. Hydrogen Cyanide. Hydrogen cyanide is a colorless, highly volatile liquid with a density 30 percent less than water. It boils at 70°F (26.5°C) and freezes at 7°F (-13.3°C). It is highly soluble and stable in water. It has a faint odor, somewhat like peach kernels or bitter almonds, and sometimes cannot be detected even in lethal concentrations. Because AC is highly volatile, in the gaseous state it dissipates quickly in the air.

b. Cyanogen Chloride. This is a colorless, highly volatile liquid with a density 18 percent greater than water. Cyanogen chloride boils at 59°F (12.5°C) and freezes at 20°F (-6.9°C). Although only slightly soluble in water, CK dissolves readily in organic solvents. The vapor of CK, heavier than air, is very irritating to the eyes and mucous membrane surfaces. The pungent, biting odor of CK is masked by its irritating and lacrimatory properties. Although non-persistent, CK vapor may remain in the jungle and forest for some time under suitable weather conditions.

6-4. Pathology
a. Hydrogen cyanide is thought to act by combining with cytochrome oxidase (an enzyme essential for oxidative processes of the tissues) and blocking the electron transport system. This results in impairment of cellular oxygen use. The CNS (particularly the respiratory center) is especially susceptible to this effect, and respiratory failure is the usual cause of death. In high concentrations, the amount of AC inhaled in a few breaths may be enough to cause immediate death without anatomical changes. After exposure to lower concentrations, there may be small areas of hemorrhage and softening in the brain which are more pronounced in delayed deaths. Death from AC leaves the blood well-oxygenated. Death from AC also causes the skin to have a pink color similar to that seen in carbon monoxide (CO) poisoning.

b. Cyanogen chloride acts in two ways. Its systemic effects are similar to those of AC, but it also has local irritant effects on the eyes, upper respiratory tract, and lungs. Cyanogen chloride damages the respiratory tract, resulting in severe inflammatory changes in the bronchioles and congestion and edema in the lungs. The fluid in the lungs may accumulate much faster than in CG poisoning. All concentrations of CK produce eye irritation and lacrimation.

6-5. Symptoms
a. The symptoms of AC depend upon the agent concentration and the duration of exposure. Typically, either death occurs rapidly or recovery takes place within a few minutes after removal from the toxic atmosphere. In high concentrations, there is increased depth of respiration within a few seconds. This stimulation may be so powerful that casualties cannot voluntarily hold their breath. Violent convulsions occur after 20 to 30 seconds with cessation of respiration within one minute. Cardiac failure follows within a few minutes. Following moderate exposure, weakness of the legs, vertigo, nausea, and headache appear very early. These may be followed by convulsions and coma which may last for hours or days, depending on the duration of exposure to the agent. If coma is prolonged, recovery may disclose residual damage to the CNS. The CNS damage may be manifested by irrationality, altered reflexes, and unsteady gait which may last for several weeks or longer; temporary or permanent nerve deafness has also been described. In mild cases, there may be headache, vertigo, and nausea for several hours before complete recovery.

b. The signs and symptoms of CK are a combination of those produced by AC and a lung irritant. Initially, CK stimulates the respiratory center and then rapidly paralyzes it. In high concentrations, however, its local irritant action may be so great that dyspnea is produced. Exposure is followed by

immediate intense irritation of the nose, throat, and eyes, with coughing, tightness in the chest, and lacrimation. Afterwards, the exposed person may become dizzy and increasingly dyspneic. Unconsciousness is followed by failing respiration and death within a few minutes. Convulsions, retching, and involuntary urination and defecation may occur. If these effects are not fatal, the signs and symptoms of pulmonary edema may develop. There may be persistent cough with much frothy sputum, rales in the chest, severe dyspnea, and marked cyanosis.

6-6. Diagnosis

a. The diagnosis of AC poisoning is suggested by the history, the odor (if detected), the rapid onset of symptoms, and the pink color of the casualties' skin.

b. In casualties exposed to CK, the diagnosis is suggested by the intense irritation and the rapid onset of symptoms.

6-7. Prognosis

a. Hydrogen Cyanide. Usually death occurs rapidly or there is prompt recovery. Occasionally, when there is prolonged tissue anoxia, residual injury of the CNS may persist for weeks; some of the damage may be permanent.

b. Cyanogen Chloride. Recovery from the systemic effects is usually as prompt as in AC poisoning. However, a higher incidence of residual damage to the CNS is to be expected. Depending on the concentration of CK to which the casualty has been exposed, the pulmonary effects may develop immediately or may be delayed until the systemic effects have subsided. Thus, early prognosis must be guarded.

6-8. Self-Aid

a. Hydrogen Cyanide. If you get a sudden stimulation of breath or detect an odor like bitter almonds during a chemical attack, *put on your musk immediately*. Speed is absolutely essential, the effects of this agent are so rapid that within a few seconds you will not be able to put on your mask. Hold your breath until the mask is on, if at all possible. This may be very difficult because of the agent's strong respiratory stimulation.

b. Cyanogen Chloride. Put on your mask immediately if you experience any irritation of the eyes, nose, or throat.

6-9. Buddy Aid

Service members not masked must put on their mask immediately if any vapors of AC or CK are present. Service members who are unable to mask should be masked by the nearest available person (buddy).

6-10. Treatment

a. In AC or CK poisoning, if the patient's respirations are feeble or have ceased, immediately administer assisted ventilation with oxygen and sodium nitrite and sodium thiosulfate *(b* below). Before the treatment is rendered, remove the patient from the contaminated environment. Continue assisted ventilation until spontaneous breathing returns or until 10 minutes after the last sign of heart activity has occurred.

b. Sodium nitrite and sodium thiosulfate must ONLY be administered by IV. Intravenously inject 10 ml of a 3 percent solution (300 mg) of sodium nitrite over a period of 3 minutes. Intravenously inject 50 ml of a 25 percent solution (12.5 gm) of sodium thiosulfate over a 10-minute period. The sodium nitrite is given to produce methemoglobin, thus sequestering the cyanide on the methemoglobin. The sodium thiosulfate combines with the sequestered cyanide to form thiocyanate which is excreted from the body.

c. The decrease in blood pressure following sodium nitrite injections is negligible unless the patient is allowed to get into an upright position. The development of a slight degree of cyanosis is evidence of a desirable degree of methemoglobin formation (methemoglobinemia). It is not anticipated that at the above dosages an extreme or injurious degree of methemoglobinemia will develop. If it does, however, it should be treated by 100 percent oxygen inhalation.

d. The lung irritant effects of CK are treated as in CG poisoning (para 5-9).

PART TWO
CONVENTIONAL MILITARY CHEMICAL INJURIES
CHAPTER 7
RIOT CONTROL AGENTS (IRRITANT AGENTS AND VOMITING AGENTS)

Section I. IRRITANT AGENTS

7-1. General

Irritant agents (lacrimators) are local irritants which in very low concentrations act primarily on the eyes, causing intense pain and lacrimation. Higher concentrations irritate the upper respiratory tract and the skin and sometimes cause nausea and vomiting. These agents may be dispersed as fine particulate smoke (aerosols) or in solutions as droplet aerosols. Examples of irritant agents are O-chlorobenzylidene malononitrile (CS), chloroacetophenone (CN), chloroacetophenone in chloroform (CNC), bromobenzylcyanide (CA), and dibenz-(b,f)-1,4-oxazepine (CR). They are used primarily in training and in riot control. Under certain conditions and with Presidential approval, they may also be used in combat. Some pulmonary irritants, such as CK and PS, are also lacrimators.

7-2. Protection

a. Protection against field concentrations of irritant agents is provided by the protective mask and ordinary field clothing secured at the neck, wrists, and ankles. The protective hood may also be worn with the mask. Individuals who handle CS should wear rubber gloves, protective mask with hood, rubber boots, and rubber apron. The BDU should be secured at the neck, wrists, and ankles.

b. Following exposure, clothing and individual equipment should be inspected for agent residue. If a residue is found, individuals should change or decontaminate the clothing to protect themselves and other unmasked persons. Decontaminate CS-contaminated clothing by airing for a few minutes.

7-3. Properties

a. Agent CS. Agent CS is a white crystalline solid which melts at 194 °F (90 °C) and is stable under ordinary storage conditions. It has a pungent, pepper-like odor. A CS cloud is white at the point of release and for several seconds after release. Agent CS is disseminated by burning, exploding, and forming an aerosol. It may also be used in liquid form in an appropriate solvent. This agent is faster-acting, about 10 times more potent, and less toxic than CN.

b. Agent CR. Agent CR is a pale yellow crystalline solid which melts at 163 °F (73 °C) and is stable in organic solutions. It has limited volubility in water and is not hydrolyzed in aqueous solutions. It has a pepper-like odor. The agent is currently used only in solution for dissemination in liquid dispensers. The solution in the dispensers contains 0.1 percent CR in 80 parts propylene glycol and 20 parts water. In organic solutions, CR is an eye irritant at concentrations of 0.0025 percent or even lower. Agent CR differs from CS in being less toxic when inhaled but skin effects are more pronounced and longer lasting. It is more persistent in the environment and on clothing.

c. Agents CN and CA. Agent CN is a white crystalline solid, boiling at 478 °F (248 °C) and freezing at 129°F (54°C). Agent CA is usually a liquid, boiling at 468°F (242°C) and freezing at 77°F (25°C). Agent CN may also be used in liquid form in appropriate solvents. The odor of CN is like that of apple blossoms. The odor of CA is like that of sour fruit. These agents may appear as a bluish-white cloud at the point of release. A solid agent is dispersed as fine particulate smoke and as vapor from burning munitions, such as lacrimator candles and grenades. Liquid agents may be dispersed from airplane spray or bursting munitions.

7-4. Effects

a. Agent CS.

(1) *Eyes and respiratory tract.* When an unmasked person enters a cloud of CS, the effects are felt almost immediately. Incapacitation begins in 20 to 60 seconds, depending upon the degree of agent concentration. The effects last for 5 to 10 minutes after removal to fresh air. There is marked burning pain in the eyes with copious lacrimation and blepharospasm, thin mucous nasal discharge, coughing, and dyspnea. Following heavy exposures, there may be nausea and vomiting. Exposure to

extremely high concentrations in an enclosed space may cause tracheitis and bronchitis. Even if that happens, permanent damage is very unlikely.

(2) *Skin.*

(a) Warm, moist skin (especially on the face, neck, ears, and body folds) is susceptible to irritation by CS. A stinging sensation may occur promptly, even at moderately low concentrations. Higher concentrations may cause an irritant dermatitis with erythema and, rarely, blisters on the same body regions. Stinging subsides after 5 to 10 minutes, even with continued exposure. An increase in the stinging is noted upon the individual's removal to fresh air. Repeated exposures may cause delayed hypersensitivity with allergic contact dermatitis. Individuals engaged in bulk handling and exposed to large quantities of CS report stinging sensations in warm, moist skin areas. Inflammation and blistering similar to sunburn may occur after a heavy or prolonged exposure, especially if the individual's skin is fair. Figures 7-1 and 7-2 show burns caused by exposure to CS.

(b) The following solution can be applied as a wash or spray to cleanse unbroken skin of CS handlers. (If this is painful in the eyes or in wounds, normal saline should be used instead.) To make the solution, add 100 gm of sodium bicarbonate, 50 gm of sodium carbonate, and 15 ml of 10 percent benzalkonium chloride solution to 1,500 ml of distilled water. The solution is stable and should be prepared in advance of need. The solution may be issued to CS handlers, as required, without a prescription.

b. Agent CR. Agent CR is similar in effect to CS, but the minimal effective concentration is lower and the lethal concentration (LCt) is higher. Symptoms and treatment are similar to those of CS.

c. Agents CN and CA.

(1) *Eyes and respiratory tract.* The vapors or smokes of these agents cause basically the same reactions as does CS. However, their toxicities are generally higher and their effectiveness as lacrimators are generally lower than CS; that is, higher concentrations of CN or CA are required to produce an irritant effect equivalent to that of CS. Recovery is quick if exposure is brief, but prolonged exposure may cause conjunctivitis and photophobia. Extremely high concentrations of these agents in enclosed spaces may cause tracheitis, bronchitis, pulmonary edema, or cerebral edema. Exposures of this magnitude are rare.

Click here for Figure 7-1.

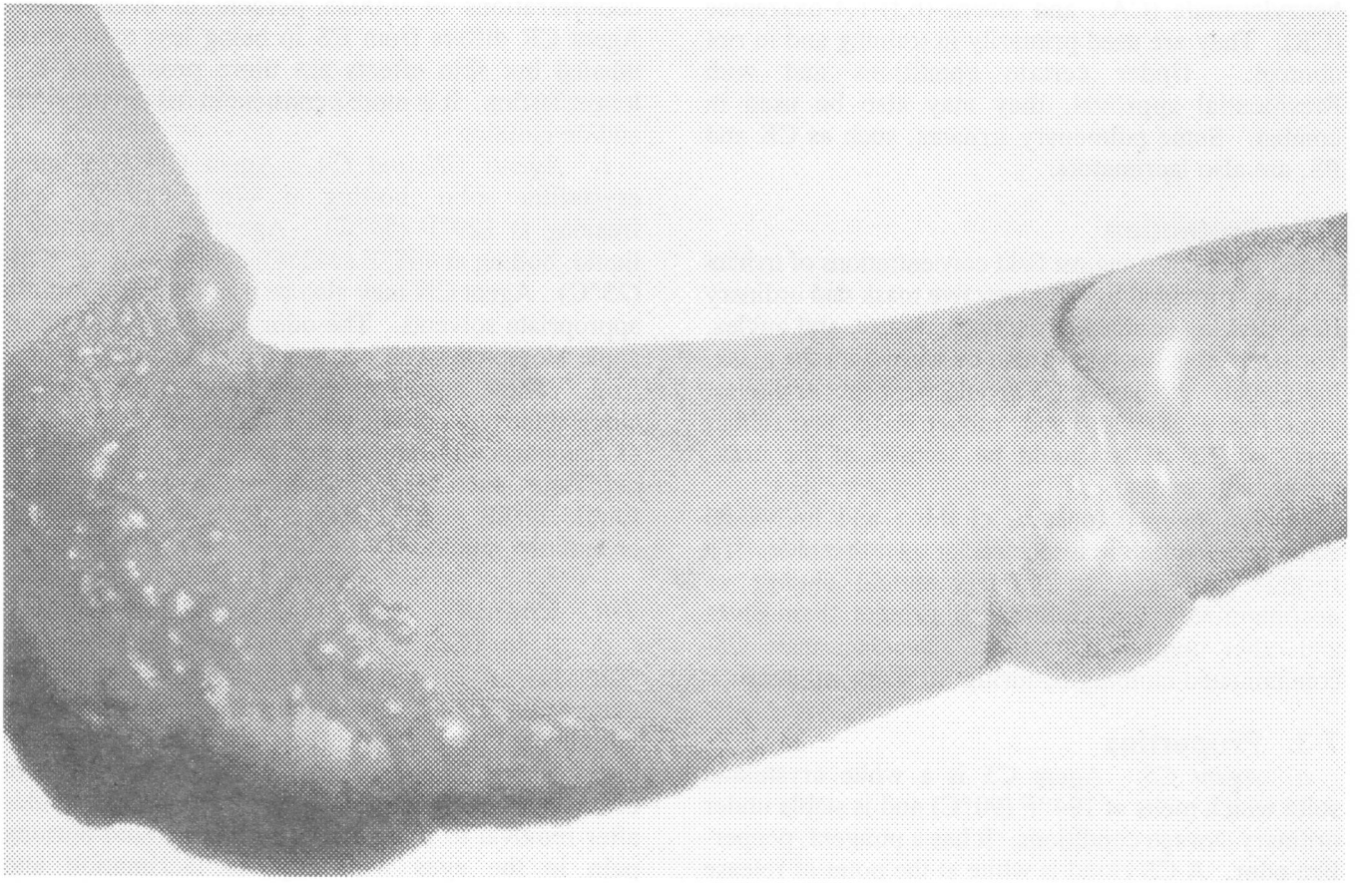


Figure 7-1. Skin burn caused by exposure to CS.

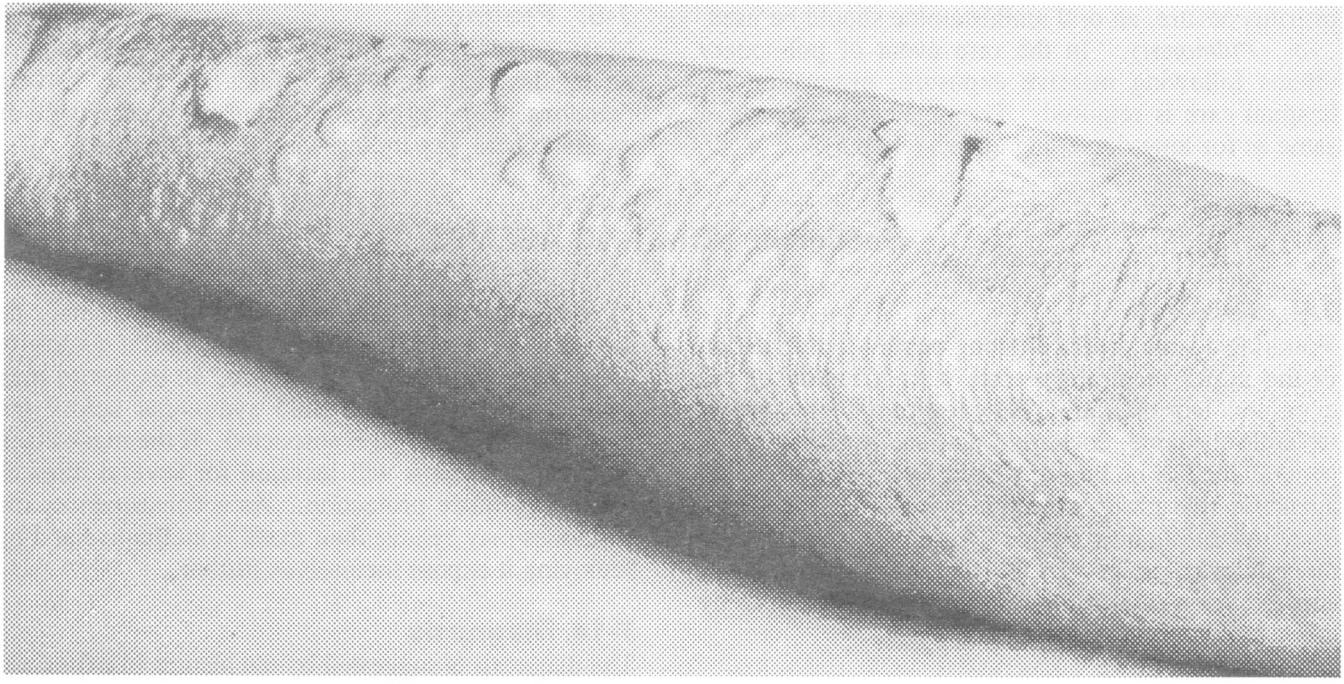


Figure 7-2. Burn caused by CS, showing desquamation of skin.

Click here for Figure 7-2.

(2) *Skin.* Stinging of the skin and, with higher concentrations, irritant dermatitis may occur in warm, humid weather. These agents are potential skin sensitizers although apparently less so than CS. Droplets of liquid or particles of solid lacrimators in the eyes may be corrosive and produce burns like those of a strong acid.

7-5. Diagnosis

a. Agent CS. Diagnosis of exposure to CS is made from the pepper-like odor, the presence of intense eye effects, dyspnea, coughing, and mucous rhinorrhea.

b. Agent CR. Diagnosis of exposure to CR is similar to the diagnosis of CS. CR produces a burning sensation in the nose and sinuses.

c. Agents CN and CA. Diagnosis of exposure to these agents is made from their odors and from the marked coughing and dyspnea, in addition to the eye effects in paragraph 7-4 *a* (1) above. Also, headache and mental depression may appear as late effects of CN exposure.

7-6. Self-Aid

Put on the protective mask, clear it, and keep your eyes open as much as possible. Move out of the contaminated environment, if possible. When your vision clears, go on with your duties. When it is safe to do so, remove the mask and blot away the tears. **DO NOT** rub the eyes. If drops or particles have entered the eye, try to forcibly open it and flush it with copious amounts of water. Chest discomfort usually can be relieved merely by talking. If exposure has been heavy, significant cutaneous reaction may develop. The cutaneous reaction can be prevented by immediately flushing the skin with copious amounts of water.

7-7. Treatment

a. Eyes. Ordinarily, the effects on the eyes are self-limiting and do not require treatment. If large particles or droplets of the agent are in the eye, treatment as for corrosive materials may be required. This is much less likely in CS exposure than in CA or CN exposure. Prompt irrigation of the eye with copious amounts of water is essential. Impacted particles of the agent may be removed mechanically. After complete decontamination, an ophthalmic corticosteroid ointment maybe used. Patients heavily exposed to CN or CA must be observed closely for development of corneal opacity and iritis. If either condition develops, promptly evacuate the patient for definitive ophthalmologic treatment.

b. Skin. Ordinarily, early (up to 1 hour) erythema and stinging sensation are transient and do not require treatment. Delayed erythema (irritant dermatitis) may be treated with a bland shake lotion (such as calamine lotion) or a topical corticosteroid (0.10 percent triamcinolone acetonide, 0.025 percent fluocinolone acetonide, 0.05 percent flurandrenolone), depending upon severity. Cases with blisters should be managed as a second degree burn. Treat oozing acute contact dermatitis with a wet dressing of 1 to 40 Burow's solution or colloidal oatmeal for 30 minutes, three times daily. The topical steroid should follow the wet dressing immediately. Secondary infections are treated with appropriate antibiotics. If significant

pruritus occurs, an oral antihistamine should be used.

c. Pulmonary. In the rare event of pulmonary effects following massive exposure, evacuation for hospital care is required. Treatment is basically the same as for lung-damaging agents (chap 5).

7-8. Prognosis

Most persons affected by irritant agents require no medical attention. Casualties are rare. Severe reactions of the eyes or the skin may take days or weeks to heal, depending upon their severity.

Section II. VOMITING AGENTS

7-9. General

Vomiting agents produce strong pepper-like irritation in the upper respiratory tract with irritation of the eyes and lacrimation. They cause violent uncontrollable sneezing, coughing, nausea, vomiting, and a general feeling of bodily discomfort. The principal agents in this group are diphenylchloroarsine (DA), diphenylaminochloroarsine (Adamsite (DM)), and diphenylcyanoarsine (DC). They are dispersed as aerosols and produce their effects by inhalation or by direct action on the eyes.

7-10. Protection

The protective mask gives adequate protection against field concentrations of vomiting agents. No protective clothing is required.

7-11. Properties

All three agents (DA, DC, and DM) are crystalline solids which are usually dispersed by heat as fine particulate smokes. When concentrated, DM smoke is canary yellow; DA and DC smokes are white. All are colorless when diluted with air. Low concentrations of these agents are effective and may not be detectable at the time of exposure.

7-12. Pathology

Vomiting agents produce local inflammation of the upper respiratory tract, the nasal accessory sinuses, and the eyes.

7-13. Symptoms

Vomiting agents produce a feeling of pain and a sense of fullness in the nose and sinuses, accompanied by a severe headache, intense burning in the throat, and tightness and pain in the chest. Irritation of the eyes and lacrimation are produced. Coughing is uncontrollable and sneezing is violent and persistent. Nasal secretion is greatly increased and quantities of ropy saliva flow from the mouth. Nausea and vomiting are prominent. Mental depression may occur during the progression of symptoms. Mild symptoms, caused by exposure to very low concentrations, resemble those of a severe cold. The onset of symptoms may be delayed for several minutes after initial exposure (especially with DM). Therefore, an exposure may occur that can produce mild symptoms before the presence of the smoke is suspected. If the mask is put on then, symptoms will increase for several minutes despite adequate protection. As a consequence, the casualties may believe their mask is ineffective and by removing it expose themselves further.

7-14. Diagnosis

The diagnosis is suggested by the history of exposure, the concurrence of respiratory and eye irritation with nausea, and the relatively rapid spontaneous improvement which occurs despite the original miserable appearance and condition of the patient.

7-15. Self-Aid

Put on the protective mask and wear it in spite of coughing, sneezing, salivation, and nausea. If necessary, lift the mask from the face briefly to permit vomiting or to drain saliva from the facepiece. Replace, clear, and recheck your mask. Carry on with your duties as vigorously as possible—this will help lessen and shorten the symptoms. Combat duties usually can be performed despite the effects of vomiting agents.

7-16. Treatment

Few cases should reach the MTF because recovery is usually prompt. Symptomatic relief may be obtained by using a current antiemetic such as trimethobenzamide hydrochloride (Tigan™) which can be given IM, IV, orally, or rectally. Aspirin may be given to relieve headaches and general discomfort.

7-17. Prognosis

Symptoms of exposure to field concentrations of vomiting agents usually disappear in 20 minutes to 2 hours, leaving no residual injury. However, a few instances of severe pulmonary injury and death have occurred due to accidental exposures to high concentrations in confined spaces.

CHAPTER 8

SMOKES

8-1. General

a. Smokes obscure vision and are used to hide troops, equipment, and areas from detection. Chemicals used to produce smokes include hexachloroethane, grained aluminum, and zinc oxide (HC) mixture, special petroleum oils (fog oil (SGF2)), diesel fuel, red phosphorus (RP) in a butyl rubber matrix, and white phosphorus (WP) plasticized or impregnated in wool felt wedges. Sulfur-trioxide chlorosulfonic acid solution (FS) and titanium tetrachloride (FM) are seldom used in current operations. The chemical composition of the petroleum-based and colored smokes are similar to the bulk materials from which they are generated. The ignition of the HC mixture produces primarily zinc chloride and only traces of CG and CO. Burning phosphorus mixtures produce smokes composed of highly concentrated (60-80 percent) polyphosphorus acids.

b. Most smokes are not hazardous in concentrations which are useful for obscuring purposes. However, any smoke can be hazardous to health if the concentration is sufficient or if the exposure is long enough. Medical personnel should be prepared to treat potential reactions to military smokes once such smokes have been introduced to the battlefield. Exposure to heavy smoke concentrations for extended periods (particularly if near the source of emission) may cause illness or even death. Except with oil smoke, high concentrations of smoke generated in closed spaces are extremely dangerous. High concentrations of HC smoke generated under these conditions have caused fatalities. **NEVER** use HC munitions indoors or in closed compartments.

8-2. Protection Against Smokes

The protective mask gives the respiratory tract and the eyes adequate protection against all smokes. The protective mask should always be worn when smoke screens are in use. Both FS and FM are highly corrosive acids in liquid form; always wear protective clothing when handling them. Solid WP is an incendiary and should not be handled. Skin irritation can occur upon exposure to the phosphorus smokes because of their high acid content. Zinc chloride has produced skin lesions and burns, generally at the site of a recent injury such as an abrasion, burn, or chapping. If diesel fuel is left on the skin too long, it can produce dermatitis. Personnel can reduce exposure to smokes by rolling down their sleeves. Showering and laundering clothing following exposure to smokes will also reduce the risk of skin irritation and sores.

8-3. Petroleum Oil Smokes

a. Physical Properties. These smokes are produced by vaporizing fuel oils in smoke generators or engine exhausts. The generator or engine exhaust vaporizes either SGF2 or diesel fuel and forces it into the air where it condenses into a dense white smoke.

b. Physiological Properties. Petroleum oil smokes are the least toxic smokes. They seldom produce ill effects. Even prolonged exposure to these smokes has not been known to cause lipoid pneumonia.

8-4. Zinc Oxide Mixtures

a. Properties. Zinc oxide mixture is a combination of hexachloroethane, aluminum powder, and zinc oxide. On burning, the mixture produces zinc chloride which rapidly absorbs moisture from the air to form a grayish white smoke. The more humid the air, the more dense the HC smoke. This smoke can be dispersed by grenades, candles, pots, artillery shells, and special air bombs. HC smoke has a sweetish, acrid odor, even at moderate concentrations. HC smoke can elicit nose, throat, and chest irritation, cough, and slight nausea in some individuals. At high concentrations, severe respiratory distress is present which may be fatal.

b. Pathology. Primary damage is largely confined to the upper respiratory tract and is due to the irritant and corrosive action of zinc chloride. Hyperemia of the larynx, trachea, and large bronchi occur, along with functional narrowing of the smaller air passages. In severe exposures, chemical pneumonia with some pulmonary edema appears. Growth of cuboidal epitheliums from the bronchioles into the alveoli (sometimes completely lining or filling the alveoli) is often seen in fatal cases, with widespread evidences of anoxia (bronchiolitis fibrosa obliterans).

c. Symptoms. Case reports of accidental exposure to moderate and high concentrations of HC smoke have shown a syndrome which includes delayed onset of more severe symptoms and slow resolution. The immediate signs or symptoms include tightness in the chest, sore throat or hoarseness, and cough. At higher exposure levels there is paroxysmal coughing, nausea,

and retching. With supportive therapy, these symptoms disappear rapidly and the patient appears normal within 6 hours. The onset of fever, rapid pulse rate, malaise, shortness of breath, retrosternal pain, abdominal cramps, and cyanosis can occur up to 48 hours after exposure. Chest x-rays associated with severe exposures have demonstrated a dense, diffuse, infiltrative process present in one or both lung field(s). Repeat chest x-ray films will show progression of the infiltrate even though the physical examination of the chest is normal. Final resolution of the infiltrate may be delayed for a month or longer, even though the patient is asymptomatic during this period. In fatal cases, shock and respiratory insufficiency, as well as infection, are the causes of death.

d. Self-Protection. Put on the mask at once in all concentrations of HC smoke. If nausea, vomiting, or difficulty in breathing develops, report for medical treatment as soon as the combat situation permits. Meanwhile, carry on with the combat duties as fully as possible.

e. Treatment. The early symptoms due to bronchial constriction may be relieved by the subcutaneous injection of 0.5 mg (0.5 ml of a 1:1000 solution) of epinephrine hydrochloride, repeated in 20 to 30 minutes if necessary. Aspirin will help relieve general discomfort. If exposure has been heavy, administer BAL in oil IM. The dose and procedure are the same as for arsenical vesicants (sec III, chap 4), except that injections are continued for 48 hours at 4-hour intervals. Therapeutic efficacy of BAL in oil has been challenged. Steroid therapy has been considered efficacious and oxygen therapy required (see para 5-3, chap 5).

f. Prognosis. The prognosis is related entirely to the extent of the pulmonary damage. All exposed individuals should be kept under observation for at least 48 hours. Most individuals recover in a few days. At moderate exposures, some symptoms may persist for 1 to 2 weeks. In severe exposures, survivors may have reduced pulmonary function for some months after exposure. The severely exposed patient may progressively develop marked dyspnea, cyanosis, and die.

8-5. Sulfur Trioxide-Chlorosulfonic Acid

a. Properties. Sulfur trioxide-chlorosulfonic acid is a standard smoke mixture for aircraft spray tanks. It is a heavy, strongly acid liquid which, when dispersed in the air, absorbs moisture to form a dense white fog consisting of small droplets of hydrochloric and sulfuric acids. In moderate concentrations, it is highly irritating to the eyes, nose, and skin.

b. Pathology. Local inflammation of the eyes, respiratory tract, and skin may be seen after severe exposures to the smoke. Contact with liquid FS produces acid burns.

c. Symptoms. The symptoms are usually limited to a prickling sensation of the skin. However, exposure to heavy concentrations or long exposures to ordinary field concentrations may result in severe eye, skin, and respiratory tract irritation. Conjunctival irritation and edema, lacrimation, and mild photophobia may occur. Mild cough and soreness in the chest and moderate chemical dermatitis of the exposed skin are occasionally seen. Splashes of liquid in the eye are extremely painful and cause mineral acid burns with corneal erosions. Liquid FS on the skin may cause painful acid burns.

d. Self-Aid. Wear the mask in all concentrations of FS smoke which cause coughing, irritation to the eyes, or a prickling sensation of the skin. If the skin is splashed with liquid FS, wash it off at once with water. If liquid FS gets into the eye, forcibly hold the eye open and flush it with water, then report for medical treatment as soon as the combat situation permits.

e. Treatment.

(1) *Eye.* Irrigate the contaminated eye with water or saline solution as soon as possible. Examine the cornea for erosion by staining it with fluorescein. If corneal erosion is severe, transfer the patient to the care of an ophthalmologist. If this is not practicable, mydriasis should be accomplished with the use of atropine sulfate.

(2) *Skin.* Wash irritated skin or skin burns with water and then with a sodium bicarbonate . solution. After washing, treat the burns as thermal burns of like severity.

f. Prognosis. The skin burns, conjunctival lesions, and respiratory irritation heal readily. Corneal erosions are more serious and may lead to residual scarring.

8-6. Titanium Tetrachloride

a. Properties. Liquid FM is a corrosive which decomposes on contact with moist air, yielding a dense white smoke composed of titanium dioxide, titanium oxychloride, and hydrochloric acid. It may be dispersed as an aircraft spray or by explosive munitions.

b. Pathology. Liquid FM produces acid burns of the skin or eyes.

c. Symptoms. Exposure of the eyes to the spray will cause conjunctivitis with Iacrimation and photophobia, but this seldom causes significant corneal injury. Liquid splashes cause acid burns of the skin and severe eye injury including some corneal erosion.

d. Self-Aid. Wear the mask in all concentrations of FM smoke which irritate the nose or the throat. Wash any liquid splash off the skin with water. If spray or liquid splash has entered the eye, forcibly open the eye and flush it with water, then report for medical attention as soon as the combat situation permits.

e. Treatment. The treatment is similar to that for FS (para 8-5 *e).*

f. Prognosis. The prognosis is good except in rare instances where corneal erosions may lead to some permanent scarring.

8-7. White Phosphorus Smoke

a. Properties. White phosphorus is a pale yellow waxy solid that ignites spontaneously on contact with air. The flame produces a hot, dense white smoke composed of particles of phosphorus pentoxide, which are converted by moist air into phosphoric acid (para 9-4). It is usually dispersed by explosive munitions. The WP smoke irritates the eyes and nose in moderate concentrations. In an artillery projectile, WP is contained in wedges which ignite immediately upon exposure to air and fall to the ground. Up to 15 percent of the WP remains within the charred wedge and can reignite if the felt is crushed and the unburned WP is exposed to the atmosphere.

b. Pathology. For burns due to particles of burning WP, see paragraph 9-4.

c. Symptoms. Field concentrations of the smoke may irritate the eyes, nose, and throat. Casualties from WP smoke have not occurred in combat operations.

d. Self-Aid. Wear the protective mask in all concentrations of WP smoke that cause any cough or irritation. Since the WP remaining in felt wedges can cause thermal injury, do not handle the charred wedges on the ground without protective covering. For self-aid against particles of burning WP, see paragraph 9-4 *a.*

e. Treatment. Generally, treatment of WP smoke irritation is unnecessary. Spontaneous recovery is rapid. For treatment of thermal injury due to large particles of burning WP, see paragraph 9-4 *b.*

f. Prognosis. No permanent injury results from exposure to WP smoke.

8-8. Red Phosphorus Smoke

This smoke is similar to WP smoke, see paragraph 8-7 for information.

8-9. Colored Smokes

a. Properties. These smokes are produced by explosive dissemination of dyes.

b. Physiological Properties. There are no reports of ill effects produced by exposure to these smokes.

CHAPTER 9

INCENDIARY AGENTS

9-1. General

Incendiary agents are used to burn supplies, equipment, and structures. The main agents in this group are thermite (TH), magnesium (MG), WP, and combustible hydrocarbons (including oils and thickened gasoline). Chemical fire extinguishers containing carbon dioxide should not be used in confined spaces to extinguish thermite or magnesium incendiaries. When carbon tetrachloride is in contact with flame or hot metal, it produces a mixture of CG, chlorine, CO, and hydrochloric acid. The field protective mask does not protect against some agents such as CO.

9-2. Thermite

Thermite incendiaries are a mixture of powdered iron oxide, powdered aluminum, and other materials. Thermite incendiaries are used for attacks on armored fighting vehicles. Thermite incendiaries burn at about 3600°F (2000°C) and scatter molten iron. Explosive charges are frequently added, which make control hazardous. Particles of iron that lodge in the skin produce multiple small deep burns. The particles should be cooled immediately with water and removed. Afterwards, the treatment is that used for other thermal burns.

9-3. Magnesium and Its Alloys

Magnesium burns at about 3600°F (2000°C) with a scattering effect similar to that of TH. Its particles produce deep burns. Healing is slow unless these particles are removed quickly. Removal is usually possible under local anesthesia. When explosive charges have been added to a magnesium bomb, the fragments may be embedded deep in the tissues, causing the localized formation of hydrogen gas and tissue necrosis.

9-4. White Phosphorus

Incandescent particles of WP may produce extensive burns. The burns usually are multiple, deep, and variable in size. The particles continue to burn unless deprived of atmospheric oxygen. The smoke irritates the eyes and the nose in moderate concentrations. Figure 9-1 shows a casualty with WP burns of the face.

a. Self-Aid.

(1) If burning particles of WP strike and stick to the clothing, take off the contaminated clothing quickly before the WP burns through to the skin.

(2) If burning WP strikes the skin, smother the flame with water, a wet cloth, or mud. Keep the WP covered with the wet material to exclude air until the particles can be removed.

(3) Try to remove the WP particles with a knife, bayonet, stick, or other available object. It may be possible to remove some particles by rubbing with a wet cloth.

(4) Report for treatment as soon as the combat situation permits.

b. Treatment.

(1) Since WP will ignite spontaneously and continue to burn when exposed to air, oxygen must be excluded until the agent is removed from the burn or the wound.

(2) At the earliest opportunity, all WP particles must be removed from the skin.

(a) Initially, the affected area is bathed in a bicarbonate solution to neutralize phosphoric acid, which will then allow removal of visible WP. Particles often can be located by their emission of smoke when air strikes them, or by their phosphorescence in the dark. In dark surroundings, fragments are seen as luminescent spots.

(b) Promptly debride the burn if the patient's condition will permit removal of bits of WP which might be absorbed later and possibly produce systemic poisoning. DO NOT apply oily-based ointments until it is certain that all WP has been removed. Following complete removal of the particles, treat the lesions as thermal burns.

(3) Once the particles have been removed, they must be placed in a container to prevent injury to others in the surrounding area.

(4) If the eyes are affected, treatment must be initiated immediately. The most effective treatment is to neutralize any phosphoric acid present by irrigating with 5 percent bicarbonate solution (5/6 cup (7 ounces) of bicarbonate dissolved in a gallon of water). Continue irrigation for 10 to 15 minutes using copious amounts of normal saline or room temperature water. Upon completion of irrigation, a wet dressing/cloth or mud should be applied to stop the WP burning by depriving it of oxygen. All WP particles that are readily accessible must be removed promptly. Since WP is readily soluble in oil and certain other solutions,

oily dressings or eye ointments must not be used. White phosphorus fumes are also irritating to the eyes and the respiratory tract. The lids must be separated and a local anesthetic instilled to aid in the removal of all embedded particles. Once all particles have been removed from eyes, atropine ophthalmic ointment should be instilled. Transfer the patient to the care of an ophthalmologist as soon as possible.

NOTE

Cupric (copper) sulfate has been used by U.S. personnel in the past and is still being used by some nations. However, copper sulfate is toxic and its use will be discontinued. Copper sulfate may produce kidney and cerebral toxicity as well as intravascular hemolysis.

CLick here for Figure 9-1.

9-5. Combustible Hydrocarbon Incendiaries

a. General. Burns may be produced by flame weapons, oil incendiary bombs (which may also contain phosphorus and sodium), and firebombs containing thickened gasoline. Lung damage from heat and irritating gases may be a complication added to the injuries from incendiaries, especially in confined places. Morphine should be given cautiously to patients with pulmonary complications. The treatment of burns caused by those agents is similar to that for other thermal burns.

b. Flame Weapon Attack. As flame and burning fuel fills an enclosed area, the oxygen content of the air is reduced. A hot toxic atmosphere containing large amounts of CO, unburned hydrocarbons, and smoke is produced. The coolest and least contaminated air is found at floor level.

(1) *Casualties.* Deaths may occur during or shortly after a flame attack due to the heat, the toxic atmosphere, or suffocation caused by laryngeal or glottic edema. Survivors may have thermal burns of the skin and upper respiratory tract and pulmonary damage from the hot flames.

(2) *Protection.* The floor level is the safest area during a flame attack. Any kind of cover affords some protection from heat. A wool blanket is excellent. The protective mask may give partial protection against smoke, but none against CO.

(3) *Treatment.* Remove casualties to fresh air as soon as possible. Assisted ventilation (using oxygen, if available) should be administered if breathing has ceased. Treat skin burns as thermal burns. If there are burns about the face, laryngeal burning with subsequent edema-producing respiratory obstruction may occur. Incubation, tracheotomy, or cricothyroid cannulation may be required. The general treatment of the casualty produced by flame attack does not differ from the treatment of one with extensive thermal burns from other sources.

c. Firebomb Attack. A firebomb is a large tank containing 100 or more gallons of thickened gasoline that is air dropped. When it strikes the ground, the fuel is ignited by phosphorus igniters and a large fireball of intense heat is produced, lasting about 4 to 6 seconds. A wide area of ground covered with burning thickened gasoline may continue to burn for 10 to 12 minutes.

(1) *Casualties.* Deaths may be caused by the intense heat or by suffocation from edema of the larynx or glottis. Thermal burns of the skin and upper respiratory tract may occur in the survivors. Danger from a toxic atmosphere is small in firebomb attacks in an open or in a well-ventilated enclosure.

(2) *Treatment.* Rapidly remove burning clothing and brush off burning fuel with a gloved hand. In general, treatment is similar to that used after flame weapon attacks.

d. Oral Replacement of Body Fluids in a Contaminated Atmosphere. In severe burns, lost body fluid must be replaced quickly to prevent shock. In a contaminated atmosphere, fluids that are being replaced orally must be administered to the patients without disrupting their MOPP. Oral fluid replacement may be accomplished by using the protective mask drinking tube and observing the following

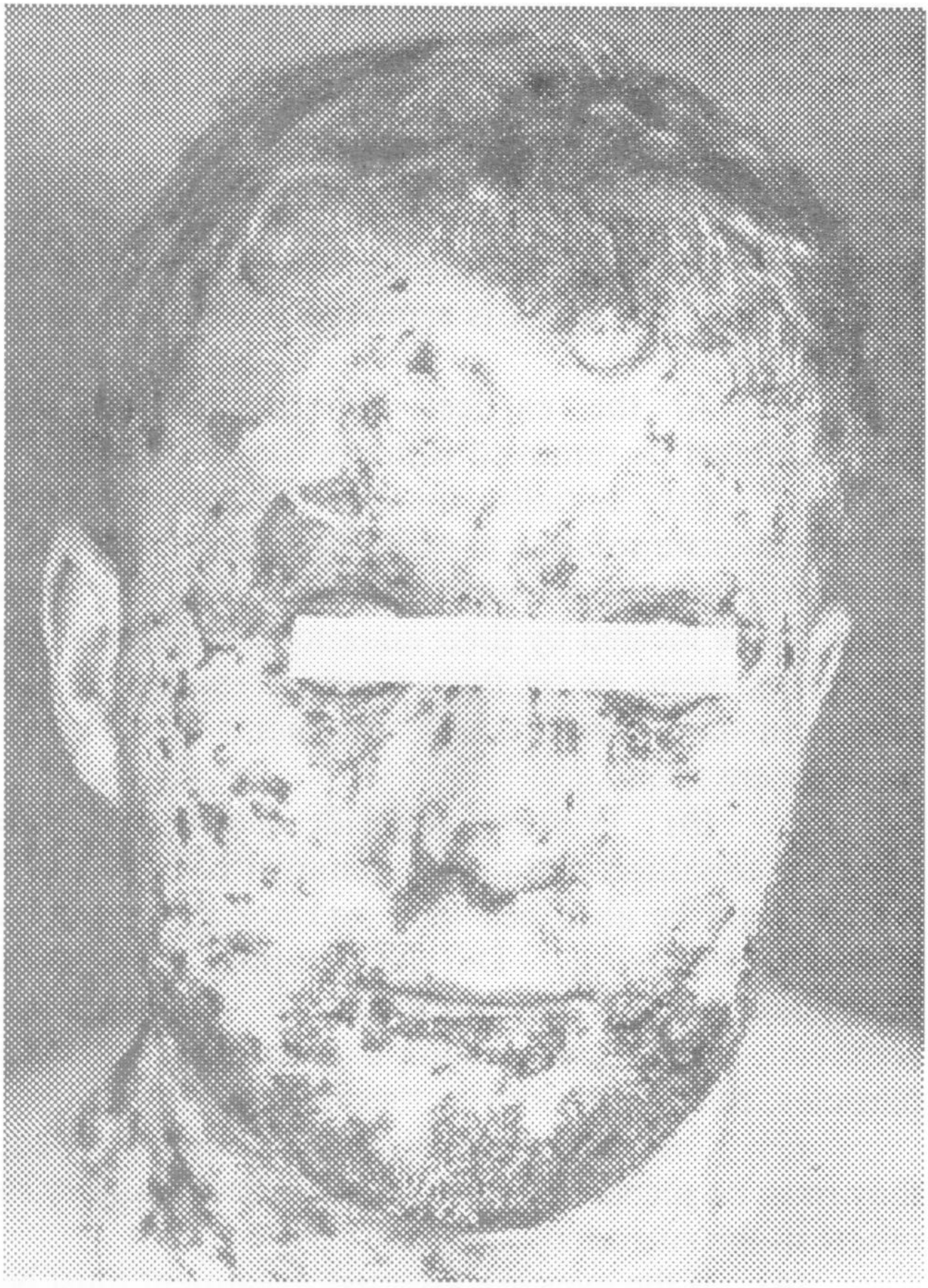


Figure 9-1. Casualty with white phosphorus burns.

procedures:

(1) Do not remove the patient's protective clothing or mask.

(2) If the patient's protective clothing has burned away, replace it with a dry uncontaminated dressing or an improvised dressing, a sheet, a blanket, a mattress cover, or similar article.

(3) Remove the patient's canteen from its carrier. Check the canteen for contamination. If contaminated, decontaminate it before using.

(4) If the patient is conscious, is not vomiting, and does not have a stomach wound, open the valve on the mask, to position the drinking tube.

(5) Insert the protruding end of the drinking tube into the protective canteen cap. Be sure the seal is tight.

(6) Gradually give the water to the patient, a few sips every few minutes. If the patient does not become nauseated, gradually increase the fluid intake. At the first sign of nausea, stop giving the water until the nausea subsides. (Administration of IV fluids, however, is the preferred method of replacing body fluids and should be accomplished when possible.) To start an IV, cut the sleeve of the protective jacket to expose the forearm. Start the IV as usual, pull the protective jacket over the IV needle and tube assembly and tape the sleeve to return the protective posture to the arm.

(7) Arrange evacuation of the casualty to an uncontaminated area as rapidly as possible.

CHAPTER 10

NOXIOUS CHEMICALS

10-1. General

a. Toxic vapors from chemicals (other than those used in military operations) may be encountered, particularly in confined or poorly ventilated spaces. Most of these vapors are thermal decomposition products of chemical elements present in a wide variety of materials.

b. The most widely encountered noxious chemicals are CO, chlorine vapor, oxides of nitrogen, hydrogen sulfide, and ammonia.

c. The field protective mask and collective protectors are of no value against CO or ammonia, and are of only limited value against oxides of nitrogen and hydrogen sulfide. A special type of canister is required for protection against CO and hydrogen sulfide (TB MED 502). Protection against the above vapors may be obtained with a self-contained breathing apparatus.

10-2. Carbon Monoxide

a. Physical Properties. Pure CO is a colorless, tasteless, odorless gas. It is lighter than air into which it diffuses rapidly.

b. Occurrence in Military Operations. Carbon monoxide is formed by gun blasts, bursting shells, internal combustion engines, fires in confined spaces, and the incomplete combustion of fuels.

c. Pathology. Asphyxiation is caused by the inactivation of blood hemoglobin through a combination with CO. The resultant anoxia may produce nervous system changes. Postmortem examinations reveal little beyond the characteristic cherry red color of the blood and hemorrhages in the brain.

d. Symptoms. Carbon monoxide is very insidious in its action and poisoning may occur without appreciable initial signs. The symptoms progress from throbbing headaches, vertigo, yawning, and poor visual acuity, to the development of cherry red mucous membranes, weakness and coma, subnormal temperature, feeble pulse, and death.

e. Diagnosis. The diagnosis is made from the circumstances of exposure and the appearance of cherry red skin and mucous membranes color.

f. Protection. In general, exposure to CO should be avoided whenever possible. Adequate ventilation should be provided for all enclosed spaces where CO may be produced. The safety of air in the space for people to breathe may be tested by standard CO indicator or detector devices. Individuals required to enter closed areas where high concentrations of CO are (known or suspected to be) present must be provided with respiratory protective devices. For the approved devices, refer to TB MED 502.

g. Treatment. Remove the victim to pure air. If respirations are weak or absent, begin assisted ventilation at once. Oxygen, if available, should be given by a face mask, preferably under pressure (up to 3 atmospheres). The patient should be kept warm and at rest (sedated, if necessary). After resuscitation, initial supportive measures (such as the need for parenteral fluids and pressor drugs) can best be decided by the medical officer. Ordinarily, methylene blue solution, morphine, and atropine should NOT be used (TB MED 269).

h. Prognosis. The longer the period of the coma, the less the chance for recovery. Most mildly exposed individuals recover with early treatment. Tachycardia and dyspnea may continue for months. There may be CNS disturbances ranging from simple neuritis to mental deterioration.

10-3. Oxides of Nitrogen

a. Physical Properties. The term "oxides of nitrogen" applies to a mixture consisting of nitric oxide, nitrogen dioxide, and nitrogen tetroxide. Nitric oxide is colorless. The other oxides are red-brown gases.

b. Occurrence in Military Operations.

(1) The danger of nitrous fume poisoning is great if high explosives (such as smokeless powder or cordite) are burned or detonated in poorly ventilation areas. This may occur in gun pits, armored vehicles, ship magazines, and turrets. This may also occur in mining and tunneling operations.

(2) In addition, nitrous fumes are emitted from fuming nitric acids (white and red) and are generated by the combustion of certain plastics.

c. Pathology. Inhalation of nitric oxide causes the formation of methemoglobin and does not appear to lead to any tissue lesions. Inhalation of nitrogen dioxide results in the formation of nitrite that leads to a fall in blood pressure and to the production of methemoglobin. Inhalation of high concentrations of nitrogen dioxide (above 0.5 mg per liter) causes rapid death without the formation of pulmonary edema. Somewhat lower concentrations result in death with

the production of yellow, frothy fluid in the nasal passages, mouth, and trachea and marked pulmonary edema. The findings in other tissues are negligible.

d. Symptoms. The symptoms following inhalation of nitrous fumes are due chiefly to nitrogen dioxide. The symptoms presented depend upon the concentration of the gas. Exposures to higher concentrations cause severe local irritation with choking and burning in the chest, violent coughing, yellow staining of the mucous membranes, expectoration of yellow-colored sputum, headache, and vomiting. Often, these early symptoms may be mild or entirely absent. After 2 to 24 hours, symptoms start with coughing, nausea, vomiting, frothy sputum, dyspnea, cyanosis, convulsions, and signs of lung edema. This train of symptoms may result in death. At exposures to very high concentrations for short periods of time, the onset of symptoms is very sudden and marked. Convulsions, unconsciousness, and respiratory arrest occur within a short time and death may follow rapidly.

e. Diagnosis. The diagnosis is made from the history, symptoms described, and sometimes the pungent odor of the gas or the yellow discoloration of the exposed mucous membranes.

f. Treatment. Treatment of casualties with symptoms of pulmonary irritation is the same as for CG poisoning (chap 5).

g. Prognosis. The few cases with symptoms referable to the CNS either die quickly or, on removal to fresh air, recover spontaneously. Fatal cases usually die within 48 hours. Bronchopneumonia and varying degrees of pulmonary fibrosis and emphysema often follow recovery from the acute stage.

10-4. Hydrogen Sulfide

a. Physical Properties. This colorless gas in low concentrations has the odor of rotten eggs. In high concentrations it may dull the sense of smell and be difficult to recognize.

b. Occurrence in Military Operations. This gas is produced during the decomposition of sulfur-containing compounds in sewers, waste, coal bins or stacks, holds of ships, and waterfront excavations.

c. Pathology. In low concentrations (less than 0.15 mg per liter), hydrogen sulfide may produce inflammation of the eyes, nose, and throat if breathed for periods of 1/2 to 1 hour. Higher concentrations (0.75 mg per liter or greater) are rapidly fatal, presumably by combination of the hydrogen sulfide with the respiratory tissue pigments and the subsequent paralysis of the respiratory center.

d. Symptoms. The symptoms depend upon the concentration of the gas. At the lowest concentrations, the effects are chiefly on the eyes; that is, conjunctivitis, swollen eyelids, itchiness, smarting, pain, photophobia, and blurring of vision. At higher concentrations, respiratory tract symptoms are more pronounced. Rhinitis, pharyngitis, laryngitis, and bronchitis may occur. Pulmonary edema may result. At very high concentrations, unconsciousness, convulsions, and cessation of respiration rapidly develop.

e. Treatment. Immediately remove the casualty from the contaminated atmosphere and administer assisted ventilation with oxygen, if possible. Treatment of pulmonary edema is the same as for that caused by CG (chap 5).

10-5. Ammonia

a. Physical Properties. Ammonia is a colorless gas, soluble in water, with a pungent odor. Liquid ammonia is a vesicant.

b. Occurrence in Military Operations. This gas has not been used in warfare but may be encountered in industrial accidents, bombings involving refrigeration plants, and holds of ships as a product of decomposing material.

c. Pathology. Exposure to high concentrations of ammonia produces prompt and violent irritation of the eyes and respiratory tract. There may be spasm and edema of the glottis or necrosis of the laryngeal mucous membranes. Pulmonary edema may develop and may be complicated by bronchopneumonia.

d. Symptoms. High concentrations produce violent, burning pain in the eyes and nose, lacrimation, sneezing, pain in the chest, cough, spasm of the glottis, and pulmonary edema. Often there is a temporary reflex cessation of respiration with spasm of the glottis. Edema of the glottis at a later period may seriously interfere with breathing. Concentrations of 0.1 percent are intolerable to humans.

e. Treatment. Treatment consists of prompt removal to pure air and administration of assisted ventilation. Later measures are directed toward the treatment of pulmonary edema, bronchitis, and pneumonia.

f. Prognosis. The mortality is high following severe exposure. With low concentrations, recovery is usually rapid although bronchitis may persist.

10-6. Hazards Caused by Fire

a. Fire produced by explosions, incendiaries, or other causes creates extreme problems. In fires, death may be caused not only by blast and direct flame but by anoxia, CO, heat, nitrous fumes, toxic fumes from burning plastics, and smoke. An oxygen rescue breathing apparatus must be used for protection in these circumstances.

b. The filter element of the field protective mask (which is designed to protect the wearer's respiratory system against the effects of chemical agents) provides only limited protection against smoke. Duration of the protection depends upon the type of smoke and its concentration. The filter element (or canister) does not generate oxygen but filters smoke and many agents

out of the air as they pass through it. Therefore, the field protective mask should not be used in air containing less than 16 percent oxygen or more than 3 percent CO, or in air having heavy concentrations of smoke from oil fires. In each case that smoke penetrates the protective mask, a new filter element (or canister) should be provided before continuing further use of the mask. Some chemicals such as CO and ammonia are not filtered by the field protective mask.

APPENDIX A

RECOGNITION OF A CHEMICAL CASUALTY

A-1. General

a. Medical personnel must be familiar with the signs and symptoms of chemical agent poisoning to avoid repetition of the experience of World War I. Medical officers frankly admitted that they were severely handicapped by their lack of experience in treating gas poisoning cases. They were often in doubt as to whether they were dealing with men suffering from gas poisoning or not.

b. Medical and tactical intelligence channels should communicate with each other as early as possible. Threat information on potential use of CW weapons / agents by enemy forces is important for planning and executing health service support operations. Once CW weapons have been used, identification of agents will be important to medical intelligence channels for operational purposes.

c. Medical units should rely on information not only from detectors and intelligence sources, but also from the casualties themselves. This applies particularly to agents for which at present there is no satisfactory detector (such as incapacitating agents). Some of the problems in the recognition and diagnosis of casualties suffering from the effects of CW operations are discussed here. Medical personnel must remember that with nerve agents, signs and symptoms may range from mild (such as miosis, headache and tightness of the chest) to signs and symptoms associated with severe poisoning (such as convulsions and respiratory failure). The nature and timing of symptoms will vary with the route of exposure. Although choking agents are less likely to be employed, the possibility of their use must not be forgotten. The danger is that the quiescent period which follows the initial poisoning might be mistaken for recovery with service members being sent back to duty even after a lethal dose. Battle casualties must be carefully examined to exclude the possibility of a psychometric agent having been used; especially those whose behavioral changes are not compatible with the physical signs of disability. When chemical agents have been used by the enemy, it is important that the fullest and earliest information be given to medical units. The information is used to facilitate the diagnosis of individual cases and to permit the arrangement for the reception of casualties.

A-2. Types of Casualties

a. On the chemical battlefield, the following types of casualties may be seen:

(1) *Conventional casualties.*

(a) Conventional casualties with no chemical injury and with no contamination of their clothing and equipment.

(b) Conventional casualties with no chemical injury but with contamination of their clothing and equipment.

(2) *Direct chemical casualties.*

(a) Chemical casualties with no other injury.

(b) Mixed casualties with conventional and chemical injuries. Since chemical munitions often include burst charges, such injuries may occur as part of a chemical agent attack. They may also be present when the chemical injury and conventional injury occur at different times. Other types of mixed casualties may be from nuclear or biological weapons used as well as the chemical weapons. Also, mixed casualties may result when chemical injuries are combined with natural illnesses (infectious disease still accounts for the majority of casualties in conventional warfare).

(3) *Indirect chemical casualties.*

(a) Casualties suffering CSR occur often in warfare, but may be more frequent where the CW threat exists. The service member will have the additional stress of isolation from wearing the chemical protective ensemble; additional fatigue when wearing the garments; and fear of chemical agents. As in World War I, the differential diagnosis between the CSR casualties and chemical casualties may sometimes be difficult.

(b) Some chemical agent antidotes can have undesirable side effects when taken inappropriately, or in large enough quantities. Atropine, for instance, causes decreased heat tolerance at a dose of 1 mg. Higher doses can cause tachycardia, dryness of the mouth, and decreased sweating. Medical personnel must be aware of the side effects of available antidotes and be alert for their appearance.

(c) Wearing the protective ensemble makes dissipation of excess body heat more difficult. Wearing the mask also makes water intake very

difficult. Both will increase the probability of heat injury (heat exhaustion or heat stroke).

A-3. Recognize Chemical Casualties

a. Any individual who suddenly becomes a casualty without being wounded or who is suffering a greater degree of incapacitation than is compatible with his wound should be considered a possible chemical casualty. The differential diagnosis will include the possibility of psychiatric casualties. It is unlikely that chemical agents will produce single casualties under field conditions. Also, a chemical attack should be suspected with any sudden increase in numbers of unexplained casualties. If chemical operations are unlikely, and if only a few service members are affected, another toxic hazard may be more probable (for example, CO).

b. Under operational conditions, the medical situation may be complicated by the psychological effects. To determine if the casualty has been caused by a chemical agent, the medical officer should ask questions along the following lines:

- Was the casualty wearing full MOPP at the time of the attack?
- Were there any aircraft or artillery bombardment in the area at the time of the attack?
- Was there any evidence of spray, liquid droplets, or smoke?
- Was anyone else affected and if so, what affects?

Did the casualty notice any unusual smell? (This is not a very reliable indicator under battle conditions, but it should be considered.)

c. To recognize a chemical casualty, the identity of the agent must be determined.

(1) The medical officer should look for the following signs and symptoms:

- An unexplained sudden runny nose.
- A feeling of choking or tightness in the chest or throat.
- Blurring of vision and difficulty in focusing the eyes on close objects.
- Irritation of the eyes.
- Unexplained difficulty in breathing or increased rate of breathing.
- Sudden feeling of depression.
- Anxiety or restlessness.
- Dizziness or light-headedness.
- Slurred speech.
- Nausea.
- Muscular weakness.

(2) Also question the patient about the delay or rapidity of the onset of symptoms. Was there any delay between exposure or contamination and the onset of effects? If so, how long was the delay?

- Did the effects persist after adjustment of the protective mask?
- Has the casualty used any self-injection device or did anyone else use any injection devices on the casualty? If so, did the symptoms improve or deteriorate?
- Is the casualty's behavior normal?

d. To assess the dose of agent received by the patient, determine the following:

- Was the casualty exercising or at rest?
- Was the casualty in the open or under cover?
- For how long was the agent inhaled?
- How long was the interval between suspected contamination and decontamination?

APPENDIX B

CARE OF CONTAMINATED CLOTHING AND EQUIPMENT AT MEDICAL TREATMENT FACILITIES

B-1. General

a. Care must be taken to prevent the spread of chemical agents in the MTFs. The spread of chemical agents can injure patients and medical personnel. Chemically contaminated clothing, blankets, and other equipment must be kept out of the MTF. Contaminated items must be decontaminated or disposed of to prevent spread of contamination.

b. Contaminated clothing and equipment are removed from the casualty as soon as possible. However, clothing removal must not compromise the individual's medical condition.

B-2. Disposition of Contaminated Clothing and Blankets

a. An area downwind of the MTF or in a leeward exposed topside position afloat should be designated as a clothing dump. Contaminated blankets and clothing, except impermeable chemical protective overgarments and rubber gloves, are transferred to this dump as conditions permit. If possible, the contaminated material is placed in plastic bags, stored in closed airtight containers, or covered with earth to prevent the escape of toxic vapors. On land, this dump is at least 75 yards (meters) downwind from the MTF and living quarters. The dump should be clearly marked with standard chemical contamination markers (FM 8-10-7 and FM 3-3/FMFM 11-17).

b. Casualties are not admitted to or removed from an MTF or other enclosed spaces in clothing or blankets known to be contaminated. To do so may result in serious injury to other patients and medical personnel from contact with the liquid agent or from the vapor which accumulates in confined spaces.

c. The medical officer should notify the proper authority of the—

- Existence of the dump for contaminated clothing and blankets.
- Exact location and size of the dump.
- Type of chemical contamination.

B-3. Replacement of Contaminated Blankets

a. To prevent the supply of blankets from becoming exhausted, those lost by contamination must be replaced.

b. An informal check on the number of contaminated blankets sent to the clothing dump is kept so that the number of replacements required are known.

c. If conditions permit, replacements are requisitioned through the normal medical supply channels. If time or the tactical situation does not permit replacement through normal medical supply channels, replacements may be requested from the nearest source of supply with which the unit has contact.

d. Succeeding echelons in the medical supply chain should request replacement blankets promptly as their supplies are displaced forward.

B-4. The Chemical Protective Ensemble

a. All personnel handling or treating chemically contaminated casualties must be at MOPP 4 (para 1-7 *a*).

b. The chemical protective overgarment is not removed until the danger of contamination has been eliminated. Contaminated chemical protective overgarments may be worn safely for 24 hours or as prescribed in FM 3-4. Personnel must also be at MOPP 4 while decontaminating litters, ambulances, and other equipment. Field Manual 3-4 gives further guidance on individual protection using the complete ensemble and FM 3-5 contains the procedure to be followed in the MOPP gear exchange. It should be noted that wearing the chemical protective ensemble places limitations on the capability of medical personnel to treat a casualty.

B-5. Disposition of Contaminated Gloves and Chemical Protective Overgarments

a. Air, Land, and Naval Operations.

(1) Contaminated gloves and overgarments are placed in a closed plastic bag and segregated for further disposal.

(2) Ordinarily, medical units cannot decontaminate impermeable protective equipment. Such contaminated equipment is placed in chemical agent-tight containers to await later decontamination. If this is not possible, the items are discarded.

b. Shipboard Operations. For shipboard

operations at sea, contaminated clothing and materials are dumped over the side. In port, store items in metal cans with tight-fitting lids or airtight plastic bags for later disposal.

B-6. Decontamination

Contaminated blankets and clothing are removed from the clothing dump by direction of the responsible officer. For specific information on decontamination, see FM 3-5.

a. Impermeable Protective Clothing, Aprons, Gloves, and Boots. Liquid contaminants on impermeable protective clothing should be neutralized or removed as quickly as possible. The quickest decontamination is that performed while clothing is being worn ((3) below). If slurry is not available, blot liquid off with available material (for example, rags). This should be done immediately if clothing is contaminated by splashes or large drops of chemical agent. Complete decontamination may be done by one of the following methods:

(1) *Aeration.* If the contamination is light or is caused by vapor, the articles can be decontaminated by airing outdoors in the wind and sunlight for several days.

(2) *Water.* Immerse heavily contaminated articles in hot soapy water at a temperature just below boiling for 1 hour. Do not stir or agitate. After 1 hour, remove the articles, rinse in clear water, and drain. While items are still hot and wet, pull apart any surfaces that are stuck together. Hang them up to dry. Repeat the process if necessary.

(3) *Slurry.* Decontaminate impregnated items (worn by depot personnel) by spraying or applying slurry immediately after contamination. After a few minutes, wash off the slurry with water. This can be done while the clothing is being worn.

b. Protective Masks, Web, Canvas, and Leather Equipment.

(1) *Protective masks.* Masks that have been exposed to droplets or vapor may be decontaminated as indicated below.

(a) If the mask is decontaminated immediately after contamination (thus avoiding absorption of the agent into the rubber), the following methods may be used:

1. Wash external parts of the mask with hot soapy water and rinse with clear water. Do not allow water to get into the filter elements. This method is practical for G-agents if the contamination is external and relatively light. Contaminated carriers may be scrubbed with hot soapy water, rinsed, drained, and air dried.

2. Decontaminate the mask by using the M291 Skin Decontaminating Kit or the M258A1 Skin Decontamination Kit (para 1-10). When using the M258A1 kit, use the DECON-2 WIPE first, then DECON-1 WIPE. The procedure for the M258A1 is a reverse of the skin decontamination procedure. This is required so that you do not leave a residue on the lens from the DECON-2 WIPE.

(b) Mask and carriers lightly contaminated by vapor only may be decontaminated by airing in sunlight and wind.

(2) *Web and canvas equipment.* First-aid pouches and other web and canvas equipment may be decontaminated by boiling 1 hour in water. The addition of soap speeds this process against all agents, particularly the G-agents. After removal from the boiling water, rinse, air dry, and return the items to service. This kind of equipment can also be decontaminated by using bleach slurry and other methods (FM 3-5).

(3) *Leather equipment.* Leather quickly absorbs liquid chemical agents. Initial decontamination should be done as rapidly as possible by using the M295 DPIE. Perform thorough decontamination when the situation permits. For thorough decontamination, soak shoes, straps, and other leather equipment in water heated to 122°F to 131°F (50°C to 55°C) (about as hot as the hand can stand it) for 4 to 6 hours, then air dry without excess heat. See FM 3-5 for additional information on decontamination of leather equipment.

B-7. Care of Litters

a. Protection. Provide emergency protection of canvas litters by covering them with materials such as ponchos, plastic sheeting, or shelter halves.

b. Decontamination.

(1) Canvas litter. If possible, take litters apart and decontaminate components as follows:

(a) Canvas. Decontaminate litter canvas by immersion in boiling water for 1 hour. If available, add 4 pounds of sodium carbonate (washing soda) to each 10 gallons of water. After boiling with washing soda, rinse with clear water.

(b) Wood. Apply a 30 percent aqueous slurry of bleach and let it react for 12 to 24 hours. Repeat applications if necessary. Then swab the wood dry and let it aerate at elevated temperatures, if possible.

(c) Metal (unpainted). Decontaminating solution number 2 is the most rapid and effective decontaminant for metals. It is effective for all chemical agents. Apply this solution to all contaminated surfaces by spray, broom, or swab; after 30 minutes, flush with water. After decontamination, aerate the metal outdoors for several hours.

(d) If the litter cannot be taken apart, decontaminate it with bleach slurry or by flushing it with hot soapy water. Then aerate the litter outdoors.

(2) *Decontaminable litter.* Apply a 5 percent chlorine solution to the entire surface of the litter and

handles / poles. If the 5 percent chlorine solution is not available, remove gross contamination by scraping with a stick or other object, then use the M295 DPIE. DO NOT use DS2 on the decontaminable litter; it may dissolve the litter fabric.

B-8. Verify Completeness of Decontamination

a. Residual Hazards. Despite the best efforts to completely decontaminate equipment, there is still a chance that a residual hazard may exist. This hazard may be due to deeply absorbed chemical agents in porous materials. These absorbed agents can emerge as chemical vapors, posing a risk to both patients and medical personnel.

b. Monitor Decontaminated Equipment. Use the Chemical Agent Monitor (CAM) to check each item prior to its being placed into the general supply area. If time allows, complete the following:

(1) Place individual items of equipment in separate clean plastic bags and seal them. Place the bags in the sun or in a heated unoccupied structure. Allow the bags to warm for 30 minutes. At the end of the 30 minutes, slightly unseal the bag, immediately place the nozzle of the CAM into the opening and observe for any indication of residual vapor hazard.

(2) If residual contamination is found, bury the item unless it is an essential item of equipment. If it is an essential item of equipment, repeat the decontamination process, then recheck as in (1) above.

APPENDIX C

MEDICAL MANAGEMENT AND TREATMENT IN CHEMICAL OPERATIONS

C-1. General

All MTFs must be prepared to receive mass casualties caused by exposure to chemical agents. A mass casualty situation exists when the number and type of casualties exceed the local medical support capabilities for their care. If the unit follows conventional operational SOPs, an overwhelming backlog of work will rapidly accumulate. Such backlogs can result in avoidable loss of life and limb with suffering. Therefore, plans for mass casualty situations must be prepared and units must be trained in applying these plans. The unit must be ready to operate with minimal confusion. Medical units must provide medical treatment to these casualties and supervise their decontamination. Normally, individual service members are responsible for their own decontamination. For casualties who are injured and unable to decontaminate themselves, this process has to be performed by buddy aid or at an MTF by nonmedical personnel from the supported unit.

a. At Echelons I and II (unit and division) including nondivisional units, the supported unit commander must provide 8 nonmedical personnel to perform patient decontamination. At Echelons III and IV (corps and COMMZ) hospitals, a 20-man patient decontamination augmentation team or 20 nonmedical personnel must be provided to perform patient decontamination. The base cluster commander or units within the geographical area of the hospital must provide the 20 nonmedical personnel.

b. Medical personnel must supervise patient decontamination personnel. The final determination on the completeness of patient decontamination rests with medical personnel.

c. If the supported units do not have the necessary resources to provide nonmedical personnel, the units (not the medical services) must address this issue with higher headquarters.

C-2. Objectives of Health Service Support in Chemical Operations

The objectives of health service support in chemical operations are to—

a. Return to duty the maximum number of personnel as soon as possible.

b. Manage casualties so that chemical agent injuries are minimized and any other injuries or illnesses are not aggravated.

c. Protect persons handling contaminated casualties or working in contaminated areas.

d. Avoid spreading contamination in ambulances, other evacuation vehicles, MTFs, and adjoining areas.

e. Continue the MTFs operations so that normal services unrelated to the medical treatment of chemical agent injuries are maintained.

C-3. Planning for the Management and Treatment of Chemically Contaminated Casualties

The initial management and treatment of casualties contaminated with a chemical agent will vary with the tactical situation and the nature of the contaminant. Therefore, each MTF must have a plan and put it into effect immediately, then modify it to meet each specific situation. Patient decontamination sites are collocated with an MTF. This ensures medical supervision of patient decontamination is available. Specifics on management of chemically contaminated patients at the MTF are found in FM 8-10-7. Each MTF has identical medical equipment sets (MES) for chemical agent patient decontamination and treatment. The numbers of each type of MES vary, depending on the echelon of care. Example: The battalion aid station (BAS) has one chemical agent patient decontamination MES and two chemical agent patient treatment MES. Each MTF must be prepared to treat—

- Chemical agent casualties generated in the geographical area of the MTF.
- Patients received from a forward and, in some cases, a lateral MTF.

C-4. Emergency Medical Treatment of Chemically Contaminated Casualties

a. Chemical agent casualties received at an MTF may also have traumatic wounds or illnesses due to other causes. Management of these patients must minimize the chemical agent injuries without aggravating their traumatic wounds or illnesses.

b. Triage of the arriving casualties is extremely important. A decision is made whether EMT or

decontamination of the casualty requires priority. Airway management and/or control of hemorrhage may be equal to or more urgent than treatment for chemical agent poisoning. Therefore, EMT measures may have to be performed in rapid sequence with decontamination or by simultaneous team actions.

c. For vesicant-contaminated casualties who have traumatic injuries or other illnesses, decontamination should be accomplished as soon as the situation permits. However, the general principle "better blistered and living than decontaminated and dead" must be followed. Lifesaving measures for a traumatic injury or some illnesses must be given priority over immediate decontamination, although the delay may increase the chemical agent injury.

d. When a contaminated casualty has another injury or illness resulting in respiratory difficulty, hemorrhage, or shock, the order of priority for emergency action is as follows:

(1) Administer chemical agent antidote, if available.

(2) Control respiratory failure (provide assisted ventilation) and/or massive hemorrhage.

(3) Decontaminate the casualty.

(4) Administer additional EMT for shock, wounds, and illnesses which are so severe that delay may be life or limb threatening.

(5) Evacuate the casualty as soon as possible, if necessary.

C-5. Patient Decontamination Methods

a. Patient decontamination serves two purposes: It prevents the patient's system from absorbing additional contaminants. It also protects medical personnel treating the patient and other patients from contamination. Accumulated contamination in the MTF is a serious threat to medical personnel and patients. Accumulated contamination may also impose a serious medical logistical burden on the unit. The effectiveness of decontamination is strongly influenced by the time lapse between initial contamination and decontamination. In many cases, the patient may have absorbed dangerous quantities of a contaminant before arriving at the MTF.

b. Each service member is trained in self-aid and buddy aid decontamination and is equipped to do so. However, any patient arriving at an MTF from a chemically contaminated area is considered contaminated, unless there is positive proof to the contrary.

c. A decontamination area is established on the downwind side of the MTF. It is provided with overhead protection such as plastic sheeting, trailer covers, ponchos, or tarpaulins. Only those patients requiring treatment at a forward MTF will have their protective overgarments and other clothing removed. Needless removal of protective clothing only increases the patient's vulnerability to liquid agent exposure with resultant increased injury. Also, forward MTFs do not have replacement protective overgarments. Any ambulatory patient decontaminated by clothing removal becomes a litter patient; he must be placed in a PPW for protection from chemical agents during evacuation. Patients not requiring treatment at a forward MTF, but requiring evacuation to the next echelon MTF must have their MOPP gear and equipment spot decontaminated. Spot decontamination will remove gross contamination, reducing the hazard to the casualty and evacuation personnel.

d. Every person entering the decontamination area (including patients) must be masked or have other respiratory tract protection in place. Most contaminants are removed by carefully removing all clothing. The patient's protective mask is not removed. Remove the mask hood, overgarments, booties and boots, the BDU, and undergarments. For step-by-step procedures in performing patient decontamination, see FM 8-10-7.

e. After patients have been decontaminated, exercise rigid control to prevent exposing their unprotected skin to a liquid chemical agent. Skin exposure to a chemical agent vapor must be minimized even though the exposure required for significant effect is much greater. After treatment in the clean treatment area or CPS, the patient is placed in a PPW and taken to the evacuation point to await evacuation. Medical personnel must monitor patients at the evacuation point to ensure that their condition remains stable; if their condition changes, additional treatment may have to be provided before evacuation.

f. Ambulatory patients may be able to decontaminate themselves and assist with the decontamination of other ambulatory patients. Their overgarments are not removed unless they must enter the clean treatment area or CPS for treatment. For patients not entering the clean treatment area or CPS, spot decontaminate the overgarment to remove gross contamination. When possible, have them proceed in groups of two or three to facilitate control. Ambulatory patients require constant observation and periodic assistance during the decontamination process. The aidman at the decontamination point removes all bandages from patients that will be treated at the MTF. Bandages are not replaced unless needed to control bleeding. After decontamination, each patient goes through the shuffle pit to the clean treatment area where wounds are treated and if possible, protective covering is restored. Restore protective covering by taping holes or tears in the protective overgarment. Patients are then returned to duty or go to the evacuation point, as their conditions dictate. Ambulatory patients with injuries that do not require immediate attention but require treatment at a higher echelon MTF are evacuated in their MOPP ensemble. EXAMPLE: A patient with a broken arm has a

stabilizing splint on. This individual does not require treatment at the BAS; however, his MOPP gear must be spot decontaminated to remove gross contamination before evacuation to the Echelon II MTF.

C-6. Logistics

a. Provisions must be made to ensure that medical personnel are supplied and equipped to manage and treat contaminated casualties. Also, supplies and equipment must be provided for protection of personnel manning the contaminated areas. Medical supplies are stored or stocked in a manner that reduces potential loss from chemical contamination.

b. Patient protective wraps must be available for casualties whose injuries require decontamination (clothing removal) for treatment in the clean treatment area. After treatment, decontaminated patients must be placed in PPWs before they are moved to the evacuation point (para *C-5e* above).

C-7. Training

Commanders must ensure that medical personnel and decontamination team members (provided by the supported unit) are trained to manage, decontaminate, and treat chemical agent contaminated casualties. Personnel must be trained to protect themselves from chemical agent injuries. In addition, provisions must be made for practice exercises to enable them to accomplish their responsibilities with speed and accuracy. For example: Decontaminating a casualty with speed is achieved through practice. Training emphasis should be placed on the following subjects:

- Employing individual protection.
- Practicing personal decontamination.
- Using chemical agent detection paper and the CAM to monitor for and detect chemical agents.
- Providing EMT.
- Performing casualty decontamination.
- Evacuating decontaminated casualties.
- Evacuating contaminated casualties.
- Sorting and receiving contaminated casualties into a system designed for the treatment of both contaminated and noncontaminated casualties.
- Patient lifting and transfer techniques.

C-8. Casualty Evacuation

a. Contaminated casualties should be decontaminated as close to the areas where they were contaminated as possible. Their MOPP gear and clothing should not be removed until they arrive at an MTF. Evacuation by ground ambulance must not be delayed for completion of decontamination. Upon arrival at the MTF where treatment will be provided, all contaminated clothing and equipment (except the protective mask) are removed and the skin and protective mask are decontaminated; spot decontaminate the skin. Decontaminated patients will not be a hazard to persons handling, treating, or transporting them. After decontamination at the field MTF, the patient is placed in the clean holding area to await admission into the CPS or clean treatment area. They must be protected from recontamination. Patients will keep their protective mask on until they are in the clean treatment area (away from the hotline) or are in the air lock of the CPS (see FM 8-10-7).

(1) Once treated, the patient is placed in a PPW before movement to the evacuation pickup point. The PPW protects the individual from further contamination. Individuals inside the PPW no longer have to wear the protective mask and are evacuated as clean. A plastic window in the PPW permits patient observation. A patient in a PPW and left in a sunny area is subject to heat build up. The protective mask remains with the patient during evacuation even though it may not be worn.

(2) If a chemical attack occurs, medical units in the evacuation system can expect to receive contaminated casualties because of the need for hasty evacuation. Therefore, extreme care must be taken to avoid spreading the contamination.

b. Before contaminated casualties are evacuated by Army aircraft or landing craft, they should be decontaminated. Otherwise, the vapor from the chemical agent may endanger the crew and other personnel, as ventilation is poor in aircraft compartments and other enclosed spaces. If casualties cannot be decontaminated before evacuation, they should be evacuated by ground ambulance. These casualties should wear their protective masks. The hazards of the chemical agent to other persons can be further minimized by applying the following measures:

- Prepare each litter by placing an impermeable cover over it and an open blanket on top of the cover.
- Place the casualties on the prepared litters and fold the sides of the blankets over them. Although this measure helps protect other persons, it increases the casualties' exposure to the contaminant and increases the possibility for heat injuries.
- Provide as much ventilation during transport as the weather and other conditions permit.
- When the casualties are removed from the litters, the impermeable covers and blankets must remain with them. If the litters have not been protected with impermeable covers, they must be treated as contaminated.

c. Patients being evacuated by Air Force aeromedical evacuation aircraft, in essentially all cases, will have been decontaminated as a result of admission to a MTF.

APPENDIX D

INDIVIDUAL DECONTAMINATION PROCEDURES

D-1. Detailed Procedures for Decontaminating the Eyes

Any suspected chemical agent contamination of your eyes or face must be removed immediately. In most cases, you will not be able to identify the agent before decontamination. Quickly obtain overhead shelter to protect yourself while performing the following decontamination process:

a. Remove and open your canteen.

b. Take a deep breath and hold it.

c. Lift your mask away from your face. Do not take the mask off.

d. Flush (irrigate) your eye or eyes immediately with copious amounts of water. To irrigate the eyes with water (from a canteen or other container of uncontaminated water), tilt your head to one side, open the eyelids as wide as possible, and slowly pour water into the eye so that it will run off the side of your face to avoid spreading the contamination. **DO NOT** use your fingers or gloved hand to hold the eyelids apart. Instead, open your eyes as wide as possible and pour the water as indicated. You must irrigate your eyes despite the presence of toxic vapors in the atmosphere. Hold your breath and keep your mouth closed to prevent contamination and absorption through the mucous membranes. Neutralize chemical agent residue along the flush path on the face.

e. Reseal, clear, and check your mask. Then resume breathing.

f. If the skin is contaminated while flushing your eyes, then decontaminate the face. Follow the procedure outlined in paragraphs D-2 or D-3 below.

D-2. Detailed Procedures for Decontaminating the Skin (Hands, Face, Neck, Ears, and Other Exposed Areas) Using the M291 Kit

The M291 Skin Decontaminating Kit (fig D-1) is provided to service members for skin decontamination. This kit may also be used to decontaminate selected individual equipment, such as load-bearing equipment, protective gloves, mask, hood, and weapon.

WARNING

The M291 kit is for external use only. Keep decontaminating powder out of the eyes; it may be slightly irritating to the eyes. Use water to wash toxic agent out of eyes. You may also use a 0.5 percent chlorine solution to wash toxic agent out of cuts or wounds.



Figure D-1. M291 Skin Decontaminating Kit.

a. Put on your mask and hood. Do not zip the hood. Do not pull the draw strings. Do not fasten the shoulder straps.

b. Seek overhead cover or use a poncho for protection against further contamination.

c. Remove one skin decontaminating packet from the carrying pouch.

d. Tear open quickly at notch. Although any notch may be used to open the packet, opening at the TEAR LINE will place applicator pad in a position that is easier to use.

e. Remove applicator pad from packet and discard empty packet.

f. Unfold applicator pad and slip finger(s) into handle.

g. Thoroughly scrub exposed skin on one hand (back of hand, palm, and fingers) until completely covered with black powder from the applicator pad.

h. Switch applicator pad to other hand and repeat procedures in step *g* above. Do not discard the applicator pad at this time.

NOTES

1. If you were masked with the hood zipped and drawstring pulled tight when you were contaminated, stop. Discard the applicator pad, put on your protective gloves and go to steps below. If, however, you were masked, but the zipper and drawstring were not secured, go to step n below.

2. Procedure is the same regardless of protective mask type. Ignore mention of hood when using the Navy Chemical Protective Overgarment, the hood is attached to the jacket.

WARNING

Injury or death may result if you breathe toxic agent while doing step *i*. If you need to breathe before you finish, reseal your mask, clear and check it, get your breath, then resume the decontaminating procedure.

i. Thoroughly scrub exposed skin of face until completely covered with black powder from the applicator pad.

(1) Hold breath, close eyes, grasp mask beneath chin, and pull hood and mask away from chin enough to allow one hand between the mask and your face. Hold mask in this position during steps (2) through (6).

(2) Scrub up and down across face beginning at front of one ear to nose to other ear.

(a) Scrub across face to corner of nose.

(b) Scrub extra stroke at corner of nose.

(c) Scrub across nose and tip of nose to other corner of nose.

(d) Scrub extra stroke at corner of nose.

(e) Scrub across face to other ear.

(3) Scrub up and down across face beginning where step (2) ended, to mouth to other end of jawbone.

(a) Scrub across cheek to corner of mouth.

(b) Scrub extra stroke at corner of mouth.

(c) Scrub across closed mouth to center of upper lip.

(d) Scrub extra stroke above upper lip.

(e) Scrub across closed mouth to other corner of mouth.

(f) Scrub extra stroke at corner of mouth.

(g) Scrub across cheek to end of jawbone.

(4) Scrub up and down across face beginning where step (3) ended, to chin and to other end of jawbone.

(a) Scrub across the under jaw to chin, cupping chin.

(b) Scrub extra stroke at center of chin.

(c) Scrub across the under jaw to the end of the jawbone.

(5) Turn your hand out, and quickly wipe the inside of the mask that touches your face.

(6) Discard applicator pad.

(7) Immediately seal mask, clear, and check it.

j. Remove second skin decontaminating packet from carrying pouch.

k. Tear open quickly at notch.

l. Remove applicator pad from packet, and discard empty packet.

m. Unfold applicator pad and slip finger(s) into handle.

n. If you were already masked when you became contaminated and skipped steps *i* through *m*, continue using the same applicator pad. Without breaking the seal between the face and mask, thoroughly scrub skin of neck and ears until completely covered with black powder.

o. Redo hands until completely covered with black powder.

p. Discard applicator pad.

q. Put on your protective gloves.

r. Fasten hood.

s. Remove powder with soap and water when operational conditions permit. It does not matter how long the powder stays on your skin.

t. Bury the used pads and packets, if circumstances permit.

NOTE

The M291 Skin Decontaminating Kit is replacing the M258A1 Skin Decontamination Kit. For U.S. Army personnel, when replaced with the M291, the M258A1 decontamination kit will be used for decontamination of individual equipment only.

D-3. Detailed Procedures for Decontaminating the Skin (Hands, Face, Neck, Ears, and Other Exposed Areas) Using the M258A1 Kit

The M258A1 Skin Decontamination Kit (fig D-2) is provided service members for performing skin decontamination. This kit can also be used to decontaminate selected individual equipment, such as load-bearing equipment, protective gloves, mask, hood, and weapon.

WARNING

The ingredients of the DECON-1 and DECON-2 packets of the M258A1 kit are poisonous and caustic and can permanently damage the eyes. Keep the solutions out of the eyes, mouth, and open wounds. Use water to wash the toxic agent out of the eyes. Do not use water to wash mustard out of wounds or off the skin. Mustard may be removed by thorough immediate wiping.

WARNING

Complete decontamination (WIPES 1 and 2) of the face must be done as quickly as possible. The quicker the agent is removed, the less amount will be absorbed. One minute after exposure is preferable to 2 minutes, 2 minutes are preferable to 3 minutes. Do not attempt to decontaminate your face or neck before putting on a protective mask.

NOTES

1. Use the buddy system to decontaminate skin areas that you cannot reach.

2. Chemical agent blisters are actually burns and should be treated as such. Blisters which have ruptured are treated as open wounds. See FM 21-11 for burn first-aid procedure.

a. Put on the protective mask (if not already on). Do not zip the hood. Do not pull the drawstrings. Do not fasten the shoulder straps.

b. Seek overhead cover or use a poncho for protection against further contamination.

c. Remove the M258A1 kit from the mask carrier or web belt. Open the kit and remove one DECON-1 WIPE packet by its tab.

d. Fold the packet on the solid line marked BEND, then unfold it.

e. Tear open the packet at the notch, remove the wipe and fully unfold it.

f. Wipe your skin starting with your hands.

NOTES

1. If you have a chemical agent on your face, do steps *g* through *t*. If you do not have an agent on your face, do step *m*, continue to decontaminate contaminated skin areas, then go to step *n*.

2. You must hold your breath while doing steps *g* through *l*. If you need to breathe before you finish, reseal your mask, clear and check it, then continue.

g. Hold your breath, close your eyes, and lift the hood and mask from your chin.

h. Scrub up and down from ear to ear.

(1) Start at an ear.

(2) Scrub across the face to the corner of the nose.

(3) Scrub an extra stroke at the corner of the nose.

(4) Scrub across the nose and the tip of the nose to the corner of the nose.

(5) Scrub an extra stroke at this corner of the nose.

(6) Scrub across the face to the other ear.

i. Scrub up and down from the ear to the end of the jawbone.

(1) Begin where step *h* ended.

(2) Scrub across the cheek to the corner of the mouth.

(3) Scrub an extra stroke at the corner of the mouth.

(4) Scrub across the closed mouth to the center of the upper lip.

(5) Scrub an extra stroke above the upper lip.

(6) Scrub across the closed mouth to the other corner of the mouth.

(7) Scrub an extra stroke at this corner of the mouth.

(8) Scrub across the cheek to the end of the jawbone.

j. Scrub up and down from one end of the jawbone to the end of the other jawbone.

(1) Begin where step *i* ended.

(2) Scrub across and under the jaw to the chin, cupping the chin.

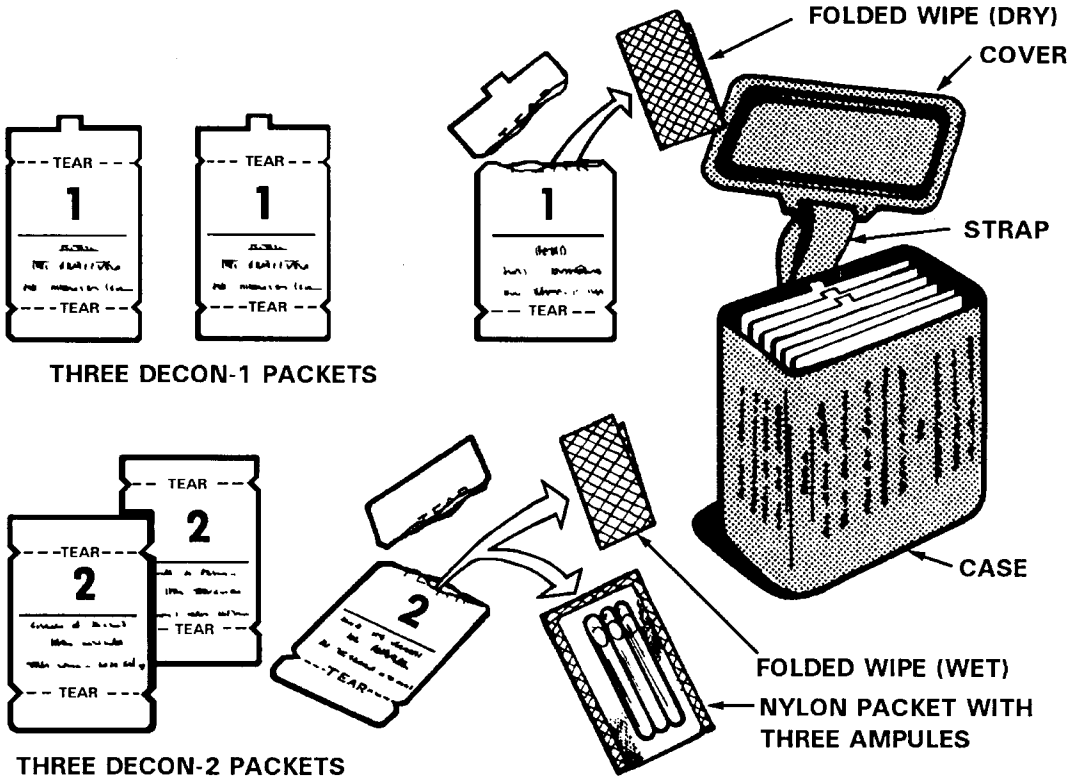


Figure D-2. M258A1 Skin Decontamination Kit.

(3) Scrub an extra stroke at the cleft of the chin.

(4) Scrub across and under the jaw to the other end of the jawbone.

k. Quickly wipe the inside of the mask which touches the face.

l. Reseal, clear, and check the mask. Resume breathing.

m. Using the same DECON-1 WIPE, scrub the neck and the ears.

n. Rewipe the hands.

o. Drop the wipe to the ground.

p. Remove one DECON-2 WIPE packet, and crush the encased glass ampules between the thumb and fingers. DO NOT KNEAD.

q. Fold the packet on the solid line marked CRUSH AND BEND, then unfold it.

r. Tear open the packet quickly at the notch and remove the wipe.

s. Fully open the wipe. Let the encased crushed glass ampules fall to the ground.

t. Wipe your hands.

NOTE

If you have an agent on your face, do steps *u* through *ee.* If you need to breathe before you finish, reseal your mask, clear and check it, then continue.

u. Hold your breath, close your eyes, and lift the hood and mask from your chin.

v. Scrub up and down from ear to ear.

(1) Start at an ear.

(2) Scrub across the face to the corner of the nose.

(3) Scrub an extra stroke at the corner of the nose.

(4) Scrub across the nose and tip of the nose to the other corner of the nose.

(5) Scrub an extra stroke at the corner of the nose.

(6) Scrub across the face to the other ear.

w. Scrub up and down from the ear to the end of the jawbone.

(1) Begin where step *v* ended.

(2) Scrub across the cheek to the corner of the mouth.

(3) Scrub an extra stroke at the corner of the mouth.

(4) Scrub across the closed mouth to the center of the upper lip.

(5) Scrub an extra stroke above the upper lip.

(6) Scrub across the closed mouth to the other corner of the mouth.

(7) Scrub an extra stroke at the corner of the mouth.

(8) Scrub across the cheek to the end of the jawbone.

x. Scrub up and down from one end of the jawbone to the end of the other jawbone.

(1) Begin where step *w* ended.

(2) Scrub across and under the jaw to the chin, cupping the chin.

(3) Scrub an extra stroke at the cleft of the chin.

(4) Scrub across and under the jaw to the end of the other jawbone.

y. Quickly wipe the inside of the mask which touches the face.

z. Reseal, clear, and check the mask. Resume breathing.

aa. Using the same DECON-2 WIPE, scrub the neck and ears.

bb. Rewipe the hands.

cc. Drop the wipe to the ground.

dd. Put on your protective gloves and any other protective clothing, as appropriate. Fasten your hood straps and neck cord.

ee. Bury the decontaminating packet and other items dropped on the ground, if possible.

D-4. Procedures for Decontaminating Individual Equipment Using the M295 Kit

a. The M295 Decontamination Packet, Individual Equipment (DPIE) is designed for use in decontamination of individual equipment. The contents of this kit are identical to those contained in the M291, except that the packets are much larger.

b. Use a stick or other object to remove any thickened spots of chemical agent from the equipment.

c. Remove one decontamination packet from the kit.

d. Open the packet, remove the pad, and place your fingers through the slot in the pad.

e. Rub all surface areas of the equipment with the pad.

WARNING

The M295 is not approved for use on the skin by the FDA. Only use the M295 kit on equipment. Keep the decontaminating powder out of the eyes; it may be slightly irritating to the eyes.

APPENDIX E
PROCEDURES FOR ADMINISTERING THE NERVE AGENT ANTIDOTES

E-1. Injection Site

The injection site for administering the MARK I and CANA (fig E-1) is normally in the outer thigh muscle. The thigh injection site is the area about a hand's width above the knee to a hand's width below the hip joint (fig E-2). It is important that the injections be given into a large muscle area. If the individual is thinly-built, then the injections should be administered into the upper outer quarter (quadrant) of the buttocks (fig E-3). Injecting in the buttocks of thinly-built individuals avoids injury to the thigh bone.

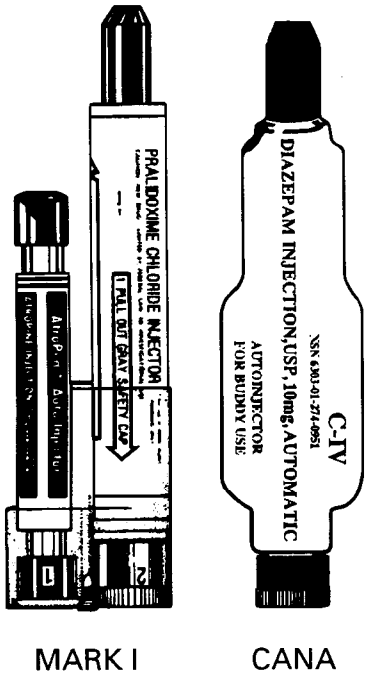


Figure E-1. Nerve agent antidotes, MARK I and CANA.

E-2. Self-Aid

If you experience any or all of the nerve agent poisoning **MILD** symptoms (para 2-5 *b* (l), you must **IMMEDIATELY** self-administer the MARK I (fig E-1). Follow the procedure given below.

a. Immediately put on your protective mask.

b. Remove one MARK I from your protective mask carrier, pocket of the MOPP suit, or other location as specified by your unit SOP. (In cold weather, the MARK I should be stored in an inside pocket of your clothing to protect the antidote from freezing. A frozen MARK I cannot be used to provide you with antidote, when needed. However, the MARK I can still be used after complete thawing.)

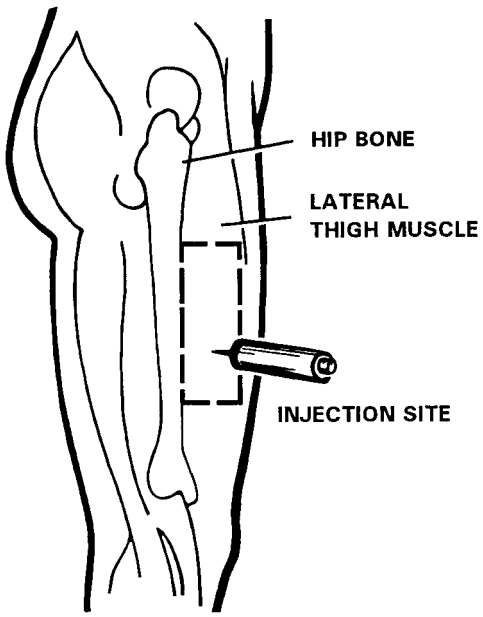


Figure E-2. Thigh injection site.

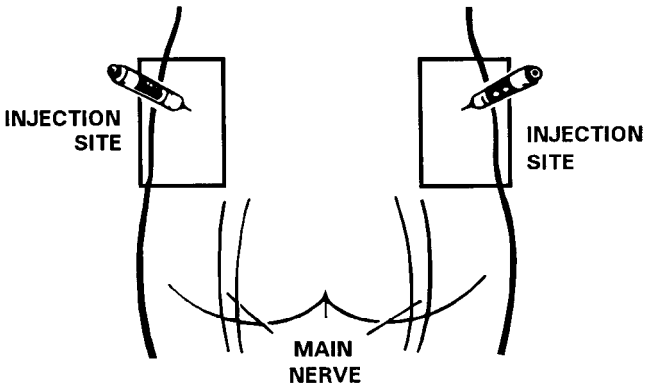


Figure E-3. Buttocks injection site.

c. With your nondominant hand, hold the autoinjectors by the plastic clip so that the larger autoinjector is on top (fig E-4A) and both are positioned in front of you at eye level.

d. With your dominant hand, check the injection site (thigh or buttocks) for buttons or objects in pockets which may interfere with the injections.

e. With this same hand, grasp the **atropine** autoinjector (the smaller of the two) with the thumb and first two fingers (fig E-4B). **DO NOT** cover or hold the needle end with your hand, thumb, or fingers—you might accidentally inject yourself. An accidental injection into the hand **WILL NOT** deliver an effective dose of the antidote, especially if the needle goes through the hand.

f. Pull the injector out of the clip with a smooth motion (fig E-4C). The autoinjector is now armed. **DO NOT** touch the needle end.

g. Hold the autoinjector with your thumb and two fingers (pencil writing position). Be careful not to inject yourself in the hand!

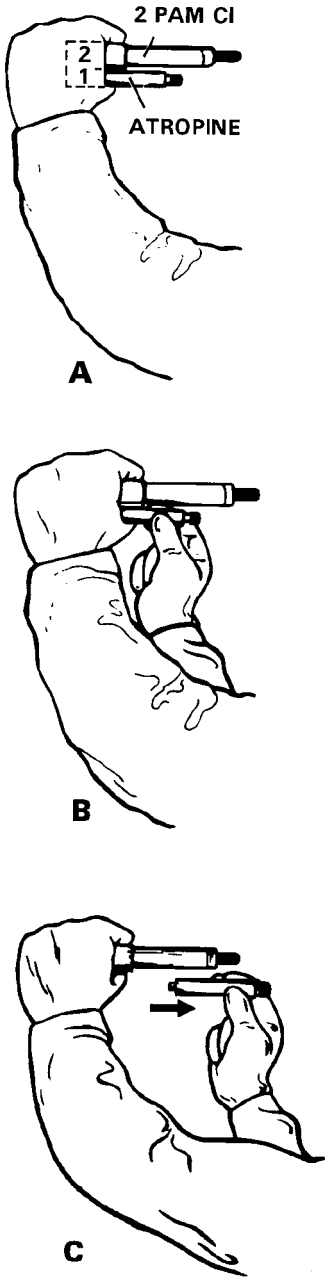


Figure E-4. Removing atropine autoinjector from clip.

h. Position the green (needle) end of the injector against the injection site (thigh or buttock). **DO NOT** inject into areas close to the hip, knee, or thigh bone.

(1) On the outer thigh muscle (fig E-5).

OR

(2) If you are thinly-built, inject yourself into the upper outer quarter (quadrant) of the buttock (fig E-6). There is a nerve that crosses the buttocks; hitting this nerve can cause paralysis. Therefore, you must only inject into the upper outer quarter (quadrant) of the buttocks.

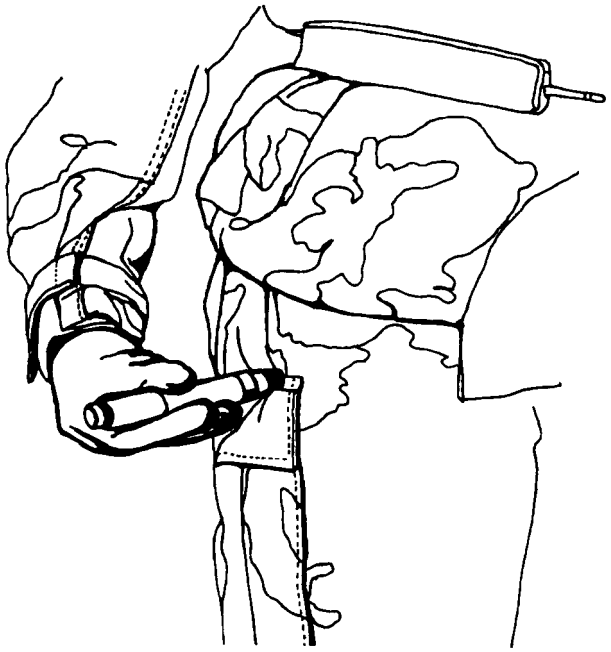


Figure E-5. Thigh injection site for self-aid.



Figure E-6. Buttocks injection site for self-aid.

i. Apply firm, even pressure (not jabbing motion) to the injector until it pushes the needle into your thigh (or buttocks). Make sure you **DO NOT** hit your mask carrier, any buttons, or objects in your pocket. Using a jabbing motion may result in an improper injection or injury to the thigh or buttocks.

j. Hold the injector firmly in place for at least 10 seconds. The seconds can be estimated by counting "one thousand one," "one thousand two," and so forth. Firm pressure automatically triggers the coiled spring mechanism. This plunges the needle through the clothing into the muscle and at the same time injects the antidote into the muscle tissue.

k. Carefully remove the autoinjector from your injection site.

l. Place the used atropine injector carefully between the little finger and the ring finger of the hand that is holding the remaining autoinjector and the clip (fig E-7A). Watch out for the needle!

m. Pull the **2 PAM Cl** injector (the larger of the two) out of the clip (fig E-7B and C) and inject yourself in the same manner as steps *f* through *k* above, holding the black (needle) end against your outer thigh (or buttocks).

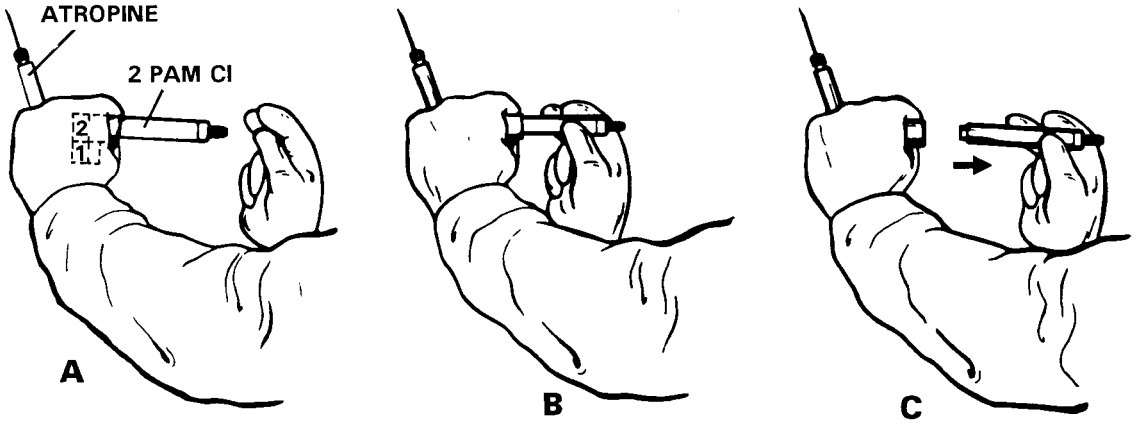


*Figure E-7. Removing **2 PAM Cl** autoinjector from clip.*

n. Drop the clip *without dropping the used injectors.*

o. Attach the used injectors to your clothing (fig E-8).

(1) Push the needles of the used injectors (one at a time) through a pocket flap of your protective overgarment jacket. Use the sleeve pocket flap on the new overgarment jacket.

(2) Bend each needle to form a hook. It is important to keep track of all used autoinjectors so that medical personnel can determine how much antidote has already been given. Knowing how much antidote has been given enables them to provide the proper follow-up treatment, if needed. Be careful not to tear your protective garment and gloves with the needles.

p. Massage the injection site, if time permits.

q. After administering the first set of injections, wait 5 to 10 minutes. After administering one set of injections, you should decontaminate your skin (app D), if necessary, and put on any remaining protective clothing.

(1) If your heart beats very rapidly and your mouth becomes very dry you have received enough antidote to overcome the dangerous effects of the nerve agent. **DO NOT** give yourself another set of injections. If you are able to walk without assistance (ambulate), know who you are, and where you are, you **WILL NOT** need the second set of injections. (If not needed, giving yourself a second set of MARK I injections may create a nerve agent antidote overdose, which could cause incapacitation.)

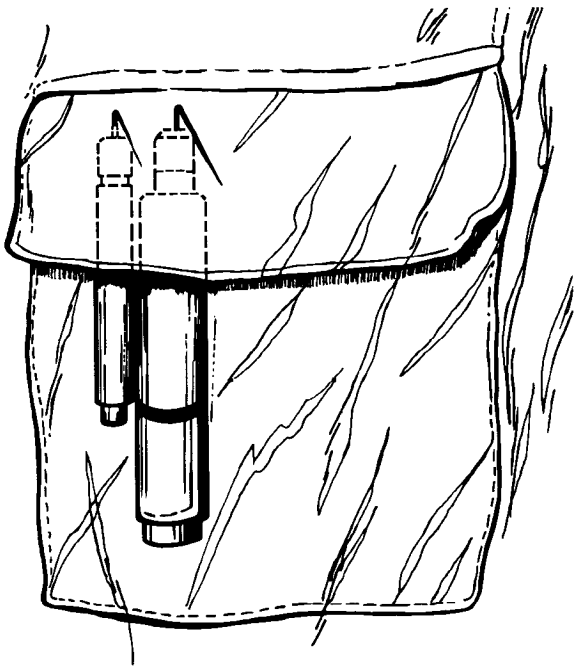


Figure E-8. One set of used autoinjectors attached to pocket flap.

(2) If you continue to have symptoms of nerve agent poisoning, seek someone else (a buddy) to check your symptoms and administer the additional sets of injections, if required.

E-3. Buddy Aid

Service members may seek assistance after self-aid (self-administering one MARK I) or may become incapacitated after self-aid. A buddy must evaluate the individual to determine if additional antidotes are required to counter the effects of the nerve agent. Also, service members may experience SEVERE symptoms of nerve agent poisoning (para 2-5 *b* (2)); they will not be able to treat themselves. In either case, other service members must perform buddy aid as quickly as possible. Before initiating buddy aid, determine if one set of MARK I injectors has already been used so that no more than three sets of the antidote are administered. Buddy aid also includes administering the CANA with the third MARK I to prevent convulsions. Follow the procedures indicated below.

WARNING

Squat, DO NOT kneel, when masking the casualty or administering the nerve agent antidotes to the casualty. Kneeling may force the chemical agent into or through your protective clothing.

a. Mask the casualty (follow the procedure outlined in FM 3-4).

b. Position the casualty on his or her side (swimmers position).

c. Position yourself near the casualty's thigh.

d. Remove all three MARK I sets (or the remaining sets) of the antidote autoinjectors and the CANA autoinjector from the inside pocket of the casualty's protective mask carrier, pocket of the **MOPP** suit, or other location as specified in your unit SOP. Do not use your own MARK I sets on a casualty. You may not have any antidote if needed for self-aid.

e. With your nondominant hand, hold the set of injectors by the plastic clip, so that the larger injector is on top (fig E-4A) and both are positioned in front of your body at eye level.

f. With your dominant hand, check the injection site (thigh or buttocks) for buttons or objects in pockets which may interfere with the injections.

g. With this same hand, grasp the atropine autoinjector (the smaller of the two) with the thumb and first two fingers (fig E-4B). **DO NOT** cover or hold the needle end with your hand or fingers—you might accidentally inject yourself.

h. Pull the injector out of the clip with a smooth motion (fig E-4C). Make sure your hand **DOES NOT** cover the needle end. Holding or carrying the needle (green) end of the autoinjector may result in accidentally injecting yourself.

i. Hold the autoinjector with your thumb and two fingers pencil writing position). Be careful not to inject yourself in the hand!

j. Position the green (needle) end of the injector against the casualty's injection site (thigh or buttocks).

(1) On the casualty's outer thigh muscle (fig E-9) .

WARNING

DO NOT inject into areas close to the hip, knee, or thigh bone.

OR

(2) If the casualty is thinly-built, inject the antidote into the buttocks. Only inject the antidote into the upper outer portion of the casualty's buttocks (fig E-10). This avoids hitting the nerve that crosses the buttocks (fig E-3). Hitting this nerve can cause paralysis.



Figure E-9. Injecting the casualty's thigh.



Figure E-10. Injecting the casualty's buttocks.

k. Apply firm, even pressure (not a jabbing motion) to the injector until it pushes the needle into the casualty's thigh (or buttocks). Make sure you do not hit the casualty's mask carrier or any objects in the individual's pockets. Using a jabbing motion may result in an improper injection or injury to the thigh or buttocks.

l. Hold the injector firmly in place for at least 10 seconds. The seconds can be estimated by counting "one thousand one," "one thousand two," and so forth.

m. Carefully remove the atropine autoinjector from the casualty's injection site.

n. Place the used injector carefully between the little finger and the ring finger of the hand that is holding the remaining autoinjector and the clip (fig E-7A). Watch out for the needle!

o. Pull the **2 PAM C1** injector (the larger of the two) out of the clip (fig E-7B and C) and inject the casualty in the manner described in steps *g* through *l* above, holding the black (needle) end against the casualty's outer thigh (or buttocks).

p. Drop the clip *without dropping the used injectors.*

q. Carefully lay the used injectors on the casualty's side.

r. Repeat the procedure immediately (steps *d* through *p* above), using the *second* and *third* sets of MARK I autoinjectors.

s. Grasp the CANA autoinjector with your dominant hand with the needle end extending beyond your thumb and two fingers. With your other hand, pull the safety cap off the autoinjector base. The injector is now armed. **DO NOT** touch the black (needle) end. To do so you may accidently inject yourself.

t. Position the black (needle) end of the auto-injector against the casualty's injection site (thigh or buttocks).

u. Apply firm, even pressure (not a jabbing motion) to the injector until it pushes the needle into the casualty's thigh (or buttocks). Make sure you do not hit the casualty's mask carrier or any objects in the individual's pockets.

v. Hold the injector firmly in place for at least 10 seconds. The seconds can be estimated by counting "one thousand one," "one thousand two," and so forth.

w. Carefully remove the CANA autoinjector from the casualty's injection site.

x. Attach the CANA and all three MARK I sets of used injectors to the casualty's clothing (fig E-11).

(1) Push the needles of the used injectors (one at a time) through one of the pocket flaps of the casualty's protective overgarment.

(2) Bend each needle to form a hook. Be careful NOT to tear the casualty's protective garments or your gloves with the needles.

y. Massage the injection site if time permits.

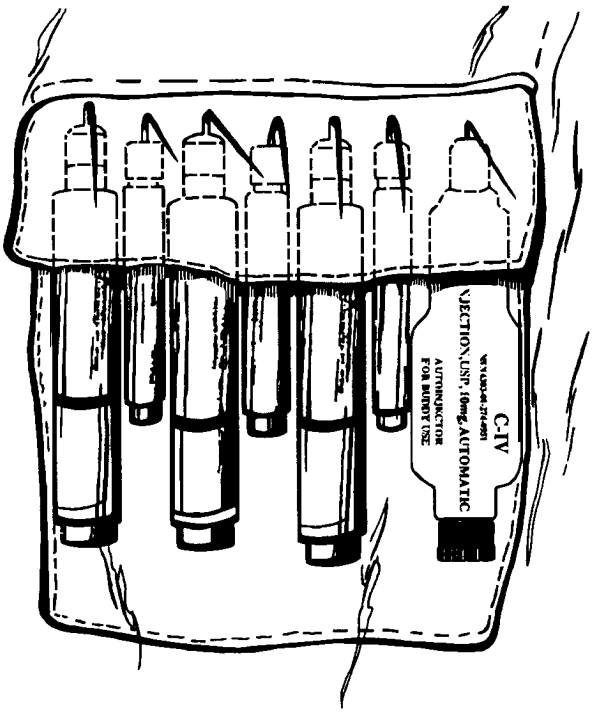


Figure E-11. Three sets of used MARK I autoinjectors and one CANA autoinjector attached to pocket flap.

GLOSSARY

Section I. ABBREVIATIONS AND BREVITY CODES

AC	hydrogen cyanide (also called hydrocyanic acid)
AFM	Air Force manual
AFP	Air Force pamphlet
AMEDD	Army Medical Department
AMEDDC&S	Army Medical Department Center and School
app	appendix
AR	Army regulation
BAL	British anti-lewisite (dimercaprol)
BAS	battalion aid station
BDU	battle dress uniform
BZ	incapacitating agent
C	Celsius/centigrade
CA	bromobenzylcyanide (riot control agent)
CAM	chemical agent monitor
CANA	convulsant antidote for nerve agent (diazepam)
CG	phosgene (lung-damaging/choking agent)
CK	cyanogen chloride
CLS	combat lifesaver
CN	chloroacetophenone (riot control agent)
CNC	chloroacetophenone in chloroform (riot control agent)
CNS	central nervous system
CO	carbon monoxide
CPS	collective protection shelter
CR	dibenz-(b,f)-l,4-oxazepine (riot control agent)
CS	O-chlorobenzylidene malononitrile (riot control agent)
CSR	combat stress reaction
CW	chemical warfare
CWC	Chemical Weapons Convention
CX	phosgene oxime (blister agent)
DA	diphenylchloroarsine (vomiting agent); Department of the Army
DC	diphenylcyanoarsine (vomiting agent)
DD/DOD	Department of Defense
DM	diphenylaminochloroarsine (Adamsite) (vomiting agent)
DNA	deoxyribonucleic acid
DP	diphosgene (choking agent)
DPIE	decontamination packet, individual equipment (M295)
DMAP	4-dimethylaminophenol-hydrochloride
DS2	decontaminating solution number 2 (a corrosive decontaminating solution)
EEG	electroencephalogram
EMT	emergency medical treatment
F	Fahrenheit
FDA	Food and Drug Administration
fig	figure
FM	titanium tetrachloride; field manual (when used with a number)
FMFM	Fleet Marine Force manual
FMTF	field medical treatment facility
FS	sulfur-trioxide chlorosulfonic acid solution (smoke mixture)

GA	Tabun (a G-agent)
G-agent	a nerve agent
GB	Sarin (a G-agent)
GD	Soman (a G-agent)
GF	a nerve agent
gm	gram(s)
H	European countries term for HD (sulfur mustard)
HC	a mixture of grained aluminum, zinc oxide, and hexachloroethane (a smoke producer)
HD	sulfur mustard (a blister agent)
HL	mustard/lewisite mixture
HN	nitrogen mustard (a blister agent)
HN1	type of HN (mustard)
HN3	type of HN (mustard)
IAW	in accordance with
IM	intramuscular
IPE	individual protective equipment
IPPB	intermittent positive pressure breathing
IV	intravenous
L	lewisite (a blister agent)
LCt	lethal concentration
LSD	d-lysergic acid diethylamide
MES	medical equipment sets
MG/mg	magnesium; milligram(s)
ml	milliliter(s)
mm_3	millimeter(s)
mm^3	millimeter(s) cubed
MOPP	mission-oriented protective posture
MRE	meal, ready to eat
MTF	medical treatment facility
NAAK	Nerve Agent Antidote Kit (MARK I) containing atropine and 2 PAM C1
NAPP	Nerve Agent Pyridostigmine Pretreatment Tablet Set
NATO	North Atlantic Treaty Organization
NAVFAC P	Naval Facilities Engineering Command Publications
NBC	nuclear, biological, and chemical
NSN	national stock number
Pam	pamphlet
para(s)	paragraph(s)
PD	phenyldichloroarsine (blister agent)
PEEP	positive end-expiratory pressure
PPW	patient protective wrap
PS	chloropicrin (irritant agent)
RP	red phosphorus
RTD	return to duty
SGF2	fog oil (smoke producing product)
SOP	standing operating procedure
STANAG	standardization agreement
TB MED	tecluical bulletin, medical
TH	thermite (incendiary)
™	trademark
TM	technical manual
2 PAM C1	pralidoxime chloride
U.S.	United States
V-agent	a nerve agent (In some countries V-agents are known as A-agents)
VX	O-ethyl methyl phosphonothiolate (a V-agent)
WBGT	wet bulb globe temperature
WP	white phosphorus

Section II. DEFINITIONS AND TERMS*

ABC-MS Chemical Agent Detector Paper	A chemical agent detector paper used to detect and identify liquid V- and G-type nerve agents and H-type blister agents. It does not detect chemical agent vapors.
acetylcholine	A chemical compound formed from an acid and an alcohol which causes muscles to contract (neurotransmitter). It is found in various organs and tissues of the body. It is rapidly broken down by an enzyme, cholinesterase. Excessive production of acetylcholine at the motor end-plates (such as found in nerve agent poisoning) may result in neuromuscular block.
acetylcholinesterase	An enzyme (a protein produced in the cells) which stops (inactivates) the action of acetylcholine by separating the acetylcholine into its components of acetic and choline. This occurs as soon as acetylcholine has produced a muscle contraction. Nerve agents combine with acetylcholinesterase to prevent it from performing its inactivation of acetylcholine.
aerosols	A suspension or dispersion of small particles (solids or liquids) in a gaseous medium.
alveoli	Microscopic air sac in the lungs where oxygen and carbon dioxide diffusion takes place through the alveolar walls.
amphetamine	A central nervous system stimulant. May be used as an incapacitating agent. Most common form is a tablet.
analeptic	A drug which stimulates the central nervous system. It is primarily used in the treatment of poisoning by drugs which depress the central nervous system. Examples are amphetamine and caffeine.
anorexia	Loss of appetite.
anoxemia	Inadequate oxygenation of the blood.
anoxia	Lack of oxygen.
antibiotic	A naturai or synthetic substance that inhibits the growth of or destroys microorganisms. Used extensively in the treatment of infectious diseases.
anticholinergic (also cholinolytic)	An agent or chemical that blocks or impedes the action of acetylcholine, such as the antidote atropine.
anticholinesterase	A substance which blocks the action of cholinesterase (acetylcholinesterase) such as nerve agents.
anticonvulsant	Class of medications that prevent or relieve convulsions. Example: diazepam.
antidote	A substance which neutralizes toxic agents or their effects (for example, atropine, 2 PAM Cl).
antihistamine	A drug that counteracts the action of histamine. It is often used in the treatment of allergies.
aphonia	Inability to phonate or produce speech sounds.

*See also JCS Pub 1-02 for definitions.

aplasia	Failure to produce cellular products from an organ or tissue, such as blood cells from the bone marrow, after a toxic dose of mustard.
apnea	Cessation of breathing.
apneic	Without breathing or respirations.
arsenic	A toxic heavy metal found in the vesicant lewisite.
arsenical	Pertaining to or containing arsenic; a reference to the vesicant lewisite.
arsenoxide	Oxophenarsine hydrochloride. An arsenical used as a vesicant such as lewisite.
asphyxiation	Suffocation.
asthma	Difficult breathing associated with bronchial obstruction precipitated by respiratory inhalants, toxins, or allergies. Inhaled chemical agents may cause bronchial spasms or mucous membrane swelling, producing asthma.
ataxia (ataxic)	Incoordination, staggering, muscular discoordination.
atelectasis	Collapse of the alveoli of the lungs secondary to mucous plugs, foreign bodies, and secretion. Frequently associated with pneumonia, best treated by vigorous coughing and breathing exercises, as well as positive pressure breathing with PEEP.
atropine	An anticholinergic used as an antidote for nerve agents to counteract excessive amounts of acetylcholine. It also has other extensive medicinal uses.
atropine sulfate ophthalmic (1 percent) ointment	An ointment applied to the eye to dilate the pupil, used in the relief of pain and to counteract miosis.
atropinization	The effect of treating with sufficient atropine to increase heart rate, stop sweating, dilate the pupils, and produce mild redness to the skin under the influence of atropine. In the case of nerve agent poisoning, it is referred to as sufficient atropine to produce a heart rate above 90.
barbiturate	A group of medications (organic compounds) which produce sedative and hypnotic effects, causing depression of the central nervous system and respiration.
beclomethasone	A glucocorticoid administered by aerosol inhalation and felt to relieve bronchospasm and prevent or ameliorate pulmonary edema following inhalation of chemical warfare agents such as CG.
belladonna alkaloid	An anticholinergic alkaloid (such as atropine, alkaloid hyoscyamine, belladonnine, scopolamine) derived from the belladonna plant and important in specific antidotal properties in counteracting acetylcholine excess in nerve agent poisoning.
betamethasone	A synthetic glucocorticoid, like beclomethasone, when administered by aerosol inhaler is felt to assist in relieving bronchospasm and ameliorate pulmonary edema following inhalation of chemical agents such as CG.
blepharospasm	A twitching or spasmodic contraction of the orbicular oculi muscle around the eye.
blister agent (vesicant)	A chemical warfare agent which produces local irritation and damage to the skin and mucous membranes, pain and injury to the eyes, reddening and blistering of the

	skin, and when inhaled, damage to the respiratory tract. Blister agents include mustards (HD and HN), arsenical (L), phosgene oxime (CX), and mustard and lewisite mixtures (HL).
blood agent (cyanogen)	A chemical warfare agent which is inhaled and absorbed into the blood. The blood carries the agent to all body tissues where it interferes with tissue oxygenation process. The brain is especially affected. The effect on the brain leads to cessation of respiration followed by cardiovascular collapse. Examples of blood agents are AC and CK.
Bowman's membrane	Thin homogeneous membrane separating corneal epitheliums from corneal substance.
bradycardia	Heart rate less than 50.
British anti-lewisite (BAL)	Commercial name for a chemical compound (dimercaprol) which is used as an antidote for heavy metal poisoning—specifically, arsenic (a component of L).
bromobenzylcyanide (CA)	The first tear agent used. It is now obsolete. It produced severe burning to the mucous membranes and irritation and tearing to the eyes. It was used as a riot control agent.
bronchiectasis	Saccular dilatation of the terminal bronchi, resulting in chronic low-grade pulmonary infection with acute exacerbations. May be acquired as a result of past pulmonary disease or injury, or may be congenital.
bronchitis	Inflammation of the mucous membrane of the bronchial tubes, producing chronic cough.
bronchoconstriction	Constriction of the bronchial tubes which tends to trap air in the lungs.
bronchopneumonia	Inflammation of the terminal bronchioles and alveoli, causing edema and consolidation of alveoli.
Burow's solution	A solution of aluminum acetate used to treat certain forms of dermatitis.
calcium hypochlorite	Calcium combined with the salt of hypochlorous acid. Used in diluted strength for decontamination of patients and equipment.
cannabinols	An alkaloid derived from the hemp plant. (See cannabis.)
cannabis	The upper portion of the hemp plant, used as a hallucinogenic. It is known as hashish and marihuana. (See cannabinols.)
carbamate	Any ester of carbamic acid. Can be used to protect acetylcholinesterase from nerve agents.
carbon monoxide	A colorless, tasteless, odorless poison gas which gives no warning of its presence. It is found in the fuel exhaust from all internal combustion engines and fossil fuels. It results from inefficient and incomplete combustion of these fuels. It is found in enclosed spaces with poor ventilation such as closed garages, inside crew compartments of vehicles, cellars, mines, and tunnels. (The field protective mask does not protect against carbon monoxide.)
carbon tetrachloride (pyrene)	Used as a solvent in industry. Its vapors are toxic and must be used cautiously. It causes liver and kidney degeneration.

carcinogen	Any cancer-causing substance.
cardiopulmonary resuscitation	The act of restoring heartbeat through chest compression and mouth-to-mouth breathing after apparent death.
catheter	A tube passed into the body for evacuating or injecting fluids into blood vessels, organs, or other parts of the body.
cerebral edema	Swelling of brain cells which, because of limited space inside the skull, may create brain compression.
chemical contamination	The deposition of chemical agents on personnel, clothing, equipment, structures, or areas. Chemical contamination mainly consists of liquid, solid particles, and vapor hazards. Vapor hazards are probably the most prevalent means of contaminating the environment, although they are not necessarily a contact hazard.
chemical decontamination	The process of sufficiently reducing the hazard caused by chemical agents in order to allow the mission to be continued. Decontamination can be done by individual service members, unit decontamination teams, or chemical units. Generally, methods used for skin decontamination include removal and/or chemical neutralization of agent(s); removal of clothing for medical examination; for equipment, the methods used are removal, destruction, covering, weathering, and chemical neutralization.
chemical pneumonitis	Inflammation of the lungs from any one of several sources, such as inhaling chemical vapors or smoke, with injury to the bronchial system as well as the alveoli.
chemical warfare agent (chemical agent)	A chemical substance which, because of its physiological, psychological, or pharmacological effects, is intended for use in military operations to kill, seriously injure, or incapacitate humans (or animals) through its toxicological effects. Excluded are riot control agents, chemical herbicides, and smoke and flame materials. Chemical agents are nerve agents, incapacitating agents, blister agents (vesicants), lung-damaging agents, blood agents, and vomiting agents.
chemosis	Edema of the conjunctival about the cornea.
Cheyne-Stokes respiration	A common and bizarre breathing pattern characterized by a period of apnea lasting 10 to 60 seconds, followed by gradually increasing respirations, and then a return to apnea. This condition can be caused by exposure to a nerve agent.
chloral hydrate	A sedative or hypnotic medication used to induce sleep. It is not felt to be a depressant. Usually administered orally.
chlorine	A gas that is used to treat drinking water. It is a highly irritating gas that is destructive to the mucous membranes of the respiratory passages; excessive inhalation may cause death. Chlorine was the first CW agent used in World War I.
chloroacetophenone	A riot control agent.
chloroform	Originally used in vapor form as an anesthetic agent, no longer used for that purpose. It is a clear, colorless liquid used in laboratory procedures.
chloropicrin (PS)	A riot control agent. It is an irritant which produces severe sensory irritation in the upper respiratory passages. Also used in industry as a disinfectant and fumigant. It is a potent skin irritant as well. May produce nausea and vomiting.
chlorosulfonic	An irritant war gas and lacrimator used widely as an intermediate in chemical synthesis.

chlorpromazine	A medication used as a minor tranquilizer and antiemetic agent. Proprietary name is Thorazine™. May be used orally, IM, or IV.
choking agent	See lung-damaging agent.
cholinergic	Referring to acetylcholine or nerve endings which liberate acetylcholine. Acetylcholine transmits the nerve impulse across the neuromuscular junction.
cholinesterase	The abbreviated term for acetylcholinesterase, which is an enzyme that hydrolyses acetylcholine to acetic acid and choline upon the chemical transmission of a nerve impulse across the neuromuscular junction.
ciliary spasm	Spasm of the muscles of the eyelids which is usually painful and may interfere with fi.mctioning of the eyelid.
codeine	An analgesic obtained from opium, acceptable for the relief of moderate pain and used to suppress coughing.
collagen	Protein substance of connective tissue.
conjunctival	The delicate membrane that lines the eyelids and covers the exposed surface of the sclera.
conjunctival	Pertaining to the conjunctiva.
conjunctivitis	Inflammation of the conjunctiva.
conventional military chemicals	These are chemical substances used within the military for day-to-day operations as well as in combat. Included in this group are chemical herbicides, insecticides, and smoke and incendiary materials.
conventional weapons	Weapons that do not employ the use of chemical, biological, or nuclear munitions.
corium	The layer of the skin under the epidermis. It contains the hair follicles, sweat glands, and sebaceous glands.
cornea/corneal	The clear, transparent anterior portion of the eye, comprising about one-sixth of its surface through which light passes to transmit images to the retina. It is continuous at its periphery with the sclera and composed of five layers.
corticosteroid (steroid)	A group of hormones derived from the adrenal gland, primarily anti-inflammatory in nature but also associated with sexual hormones and electrolyte balance with profound effects upon the body.
cricothyroid membrane	A small circular area of the thyroid cartilage which can be readily entered with a needle to establish an airway.
cricothyroid needle	A hollow needle specifically designed to pierce the cricothyroid membrane and to permit the flow of air.
cuboidal epitheliums	Cuboidal refers to cells that are shaped like a cube. Cells lining the surfaces of organs and the body are known as epitheliums.
cutaneous	Pertaining to the skin.
cyanide	The broad term used for any cyanide, which includes hydrogen cyanide and cyanogen chloride.

cyanogen chloride (CK)	A blood CW agents. Acts similar to cyanide in depriving cells of oxygen.
cyanogens	Current NATO generic term for blood agents that includes hydrogen cyanide and CK. See blood agent.
cyanosis	Slightly bluish, grayish, slate-like, or dark purple discoloration of the skin due to reduction of oxygen in the blood.
cyclitis	Inflammation of the ciliary body of the eye.
d-amphetamine (dextroamphetamine sulfate)	A medication that is a CNS stimulant. Frequently used in drug abuse, a common isomer of amphetamine sulfate.
DECON-1 packet	A comuonent of the M258A1 Skin Decontamination Kit (3 packets in total). Each packet contains a wipe prewetted with hydroxyethane 72 percent, phenol 10 percent, sodium hydroxide 5 percent, and ammonia 0.2 percent, and the remainder water.
DECON-2 packet	Another component of the M258A1 Skin Decontamination Kit (3 packets in total). Each packet contains a wipe impregnated with chloramine B and sealed glass ampules filled with hydroxyethane 45 percent, zinc chloride 5 percent, and the remainder water.
dermatitis	An inflammation or infection of the skin.
dexamethasone-21-isonicotinate	A glucocorticoid used in several forms, specifically in the treatment of mild mustard conjunctivitis in topical ointment form.
diathesis	Conditions that predisposes the body toward or causes it to have a tendency to develop certain diseases.
dibenzoxazepine (CR)	Similar to CS but minimum effective concentration is lower and LCt 50 is higher. Symptoms and treatment are similar to CS.
dichloroarsine	An arsenical vesicant such as phenyldichloroarsine and chlorovinyldichloroarsine (L).
dirnercaprol	See British anti-lewisite.
dimethyl sulfate (H-agent simulant)	A chemical used as a simulant for mustard (H). Also has been used as an industrial poison and war gas, causing nystagmus, convulsions, and death from pulmonary complications.
diphenylaminearsine chloride (Adamsite, DM)	A vomiting agent.
diphenylchloroarsine (DA)	A vomiting agent.
diphenylcyanoarsine (DC)	A vomiting agent.
d-lysergic acid diethylamide (LSD)	A hallucinogenic drug subject to abuse. Creates bizarre behavior, psychosis. No legitimate use now, but has been used experimentally in the study of mental disorders.
dorsum	The back or posterior surface of the body.
dysarthria	Garbled speech as a result of muscular impairment.

dyspnea	Labored breathing resulting from an increased need for oxygen or inadequate air exchange in the lungs.
eczematoid dermatitis	Superficial skin condition with inflammatory component and crusting.
edema	Excess fluid buildup in the tissues causing swelling.
emphysema	Process of trapping air in the alveoli, associated with loss of elasticity of the lung tissues and resulting in being unable to completely exhale.
endemic	A low level but continuous incidence of a disease in a given population.
endotracheal	Placing a device through the lumen of the trachea, such as an endotracheal tube.
endotracheal tube	A tube placed through the lumen of the trachea to maintain a patent airway and prevent aspiration by inflating a cuff that surrounds the tube after the tube is in place.
epidemiological	The study of diseases.
epigastric	Upper middle abdomen, especially that portion located in the sternal area.
epilepsy	Usually a convulsive disorder precipitated by a massive brain electrical discharge, altered consciousness, with bursts of motor activity. There may be a significant difference between types of epilepsy.
epinephrine	A fight or flight hormone from the adrenal medulla produced by stress or pain. Increases heart rate, dilates pupils, and increases respiratory rate. Also known as adrenaline. Used as a medication to relieve bronchial constriction.
epinephrine hydrochloride	A drug used to relieve bronchospasms or constrictions, such as when exposed to HC mixture. It is administered by IM injection.
epistaxis	Nosebleed.
eructation	Belching.
erythema	Red area of the skin, caused by heat or cold injury, trauma, or inflammation. May be localized or generalized.
ethyldichloroarsine	A chemical warfare agent related to L used as a vesicant. May be a respiratory tract irritant and cause pulmonary edema.
fasciculation	Localized contraction of muscle fibers, usually visible through the skin.
fibrosis	Scar tissue, replacement by fibrous tissue.
flaccid paralysis	Loss of muscle tone and capability to function. Nerve agents cause this condition.
fluocinolone acetonide	One of the components of a topical steroid used in treatment of skin erythema and edema after exposure to certain riot control chemical agents.
fluorescein dye	Used to make foreign bodies in the eye fluoresce.
flurandrenolone	One of the components of a topical steroid used in the treatment of skin erythema and edema after exposure to certain riot control chemical agents.

fog oil	A smoke made from a special petroleum oil.
G-agent	A nerve agent such as GA, GB, GD and GF.
gangrene	A death of a body part, usually due to deficient or absent blood supply.
glottic edema	Swelling of the larynx due to exposure to chemical agents. It may result in a voice change or loss of voice.
grand mal	The most pronounced form of epilepsy, usually preceded by an aura, then a loss of consciousness and tonic clonic movements.
granulocytopenia	Absence of white cells of the granulocyte series in blood stream.
H-agent simulant	A chemical, isoamyl salicylate, used as a simulant for mustard vesicant. It is one of a family of vesicant simulants.
hallucinogen	A drug which produces visual, auditory, and olfactory imaginary sensations. Such drugs are cannabinols, LSD, peyote, and alcohol.
HC mixture	A special smoke made from petroleum oil. It is a mixture of grained aluminum, zinc oxide, and hexachloroethane.
hematopoietic	Pertaining to the production and development of blood cells.
hemoconcentration	A relative increase in the number of red blood cells, resulting from a decrease in the volume of plasma.
hemolysis	Separation of the hemoglobin contents of the red blood cell from the red blood cell membrane as a result of injury or aging of the red blood cell.
hemolytic anemia	Anemia caused by increased destruction of red blood cells where the bone marrow is not able to compensate for it.
histamine	A substance found in most body tissues (particularly in most cells) which causes vasodilatation, increased gastric secretion, increase in heart rate, and hypersensitivity reactions.
hydrocarbon	Any compound made up of hydrogen and carbon, either as a long chain (aliphatic) or in ring form (aromatic or cyclic).
hydrochloric acid	A strong acid in the form of an aqueous solution of hydrogen chloride. Also known as muriatic acid.
hydrogen cyanide (AC) (hydrocyanic acid)	A CW agent, extremely poisonous, which blocks the uptake of oxygen by tissue cells (suppresses cellular respiration). Produces rapid onset of symptoms from toxic effects including tachypnea, dyspnea, paralysis, and respiratory arrest.
hydrogen sulfide	A noxious chemical with a strong odor of rotten eggs.
hydrolytic	Process of changing the characteristics of a chemical by subjecting it to water with the production of a hydroxyl group and a hydrogen atom.
hyperemia	Increased redness of the skin which usually disappears with pressure or increased blood flow to a body part.
hypertension	High blood pressure, usually greater than 140 systolic and 90 diastolic.

hypertonic	Greater than normal physiologic concentration. A solution having a greater tonicity than a normal solution of particular body fluids.
hyperventilation	Excessive breathing (too rapid and/or too deep) with a resultant decrease in carbon dioxide tension and respiratory alkalosis.
hypopyon	Pus in the anterior chamber of the eye.
hypotension	Less than "normal" blood pressure within the vascular system. An insufficient blood pressure to adequately perfuse the body. If blood pressure is markedly low, then it is termed shock.
hypovolemic shock	Insufficient blood volume to maintain adequate tissue oxygenation and aerobic metabolism.
hypoxemia (hypoxia)	Insufficient oxygen in the circulatory system to adequately supply tissue cells. May be caused by lack of oxygen, inadequate hemoglobin to carry oxygen, or interference with transfer of oxygen to the cells.
incapacitating agent	A CW agent which produces a temporary disabling condition that persists for hours to days after exposure has ceased. Generally, CNS depressants and CNS stimulants are the two types that are likely to be encountered in military operations. Examples are cannabinols and phenothiazine compounds.
incendiary agent	A warfare agent used to burn supplies, equipment, and structures. The main groups are thermite, magnesium, WP, and combustible hydrocarbons (including oils and thickened gasoline).
individual protective equipment (IPE)	Individual protective equipment includes the chemical protective overgarment, mask with hood, rubber butyl gloves, and booties.
integrated battlefield	Warfare and/or contingency operations where nuclear, biological, and/or chemical weapons are being employed or have a high probability of being employed in addition to conventional weapons.
intermittent positive pressure breathing (IPPB)	A method of ventilating a patient with pressure greater than atmospheric during the inspiratory phase of breathing.
iritis	Inflammation of the iris with accompanying pain, photophobia, lacrimation, and diminution of vision. Treated with atropine to dilate the pupils, systemic steroids are frequently used.
irritant agent	A tear agent or lacrimator which, in very low concentrations, acts primarily on the eyes, causing intense pain and lacrimation. Higher concentrations cause irritation in the upper respiratory tract and the skin, and sometimes nausea and vomiting. Examples of irritant agents are CN, CNC, CA, and CS.
ischemic necrosis	Death of body tissue (or cells) due to lack of blood supply.
lacrimation	Secretion and discharge of tears.
lacrimator	A substance which induces the secretion of tears.
laryngitis	Swelling, redness, and inflammation of the larynx.
laryngoscope	A lighted instrument for visualization of the larynx.

larynx	The voice box located in the thyroid cartilage.
latent period	Specifically in the case of mustard, the period between exposure and onset of signs and symptoms; otherwise, an incubation period.
leukemia	Cancer of the white blood cells.
leukocytosis	Increase above normal of white blood cells.
leukopenia	Less than the normal number of white blood cells.
lewisite (chlorovinyldi-chloroarsine)	A fast-acting vesicant, lacrimator, and lung irritant.
lipoid pneumonia	A pneumonia caused by the inhalation or ingestion of petroleum oils or fats.
liquefaction necrosis	Death of tissue, with softening to the point that tissue becomes at least partially liquefied.
lung-damaging agent	A chemical warfare agent, also known as a "choking agent" which produces irritation to the eyes and upper respiratory tract and damage to the lungs, primarily causing pulmonary edema. Examples of lung-damaging agents are CG, DP, chlorine, PS, and CK.
lymphocytopenia	An absolute decrease in the presence of lymphocytes in the blood, usually less than 1500 per mm^3.
M256 Chemical Agent Detector Kit	A kit that detects and identifies vapor concentrations of nerve, blister, and blood agents.
M258A1 Skin Decontamination Kit	A kh used for performing emergency decontamination of the skin and selected small equipment, such as the protective gloves, mask, hood, and individual's weapons. Each kit contains three DECON-1 WIPES and three DECON-2 WIPES. This kit is being replaced by the M291 Skin Decontaminating Kit. When replaced the M258A1 will ONLY be used for decontamination of individual equipment.
M291 Skin Decontaminating Kit	A kit to perform emergency decontamination of the skin and mask. The kit contains six decontamination packets.
M295 Decontamination Packet, Individual Equipment (DPIE)	A kit (similar to the M291 Skin Decontaminating Kit) used to decontaminate equipment, such as the weapon, helmet, and other gear, that is carried by the service member. Although similar to the M291, this kit is not FDA-approved for use on the skin.
M9E1 Chemical Agent Detector Paper	A paper that detects the presence of liquid nerve agents (V and G) and blister agents (H, HN, and L). This paper does not distinguish between the types of agent involved, only that an agent or agents may be present. Neither will it detect chemical agent vapor.
maceration	Destruction of soft tissue, usually associated with prolonged immersion in water or wetness and may, in some cases, have been associated with trauma.
magnesium	An element which, in metal form, burns readily at high temperatures, splatters readily upon burning, and may cause severe bums.

malathion	An organophosphate insecticide currently in wide usage.
MARK I	See Nerve Agent Antidote Kit, MARK I.
methemoglobin	A reduced form of hemoglobin, no longer capable of oxygen transport. May be caused by medications. The iron in the hemoglobin is oxidized from ferrous to ferric. Cyanide is attracted to methemoglobin. Sodium nitrite is administered to form the methemoglobin in the blood to sequester the cyanide.
methyldichloroarsine	One of a group of vesicant chemical warfare agents.
methylene blue solution	An organic compound which prevents the formation of methemoglobin. However, oxygen should be used in most instances rather than methylene blue. Has been used as an antidote for cyanide poisoning (not recommended).
methylprednisolone	A steroid medication derived from prednisolone, anti-inflammatory in nature, and used to prevent or lessen the severity of pulmonary edema.
micturition	The act of emptying the bladder of urine.
miosis	Pinpoint or small pupils.
mission-oriented protective posture	A flexible system for protection against NBC contamination. This posture requires personnel to wear only that individual protective clothing and equipment consistent with the threat, work rate imposed by the mission, temperature, and humidity. There are five levels of MOPP (zero through 4). MOPP 4 offers the greatest protection but also degrades mission performance the most.
morbilliform	Description of a specific rash that is red, blotchy, and generalized in character.
morphine	A potent narcotic used in the control of pain, derived from opium. Readily abused. Continues to be the analgesic of choice for initial pain control in the combat-wounded service member.
muscarinic	A specific type of poisoning affecting the postganglionic parasympathetic neural-muscular junction, resulting from excess acetylcholine due to inhibition of acetyl-cholinesterase. The result is a decrease in heart rate, bronchoconstriction, and salivary and lacrimal gland stimulation.
mustard (HD)	A vesicant chemical warfare agent which has been used extensively in warfare. Creates destruction of epidermis, eye and pulmonary injury, and, in high doses, bone marrow depression.
myasthenia gravis	A disease characterized by either lack of acetylcholinesterase or excess of acetyl-choline in which the patient has disabling muscular weakness and severe fatigability. Treated by such medications as pyridostigmine.
mydriasis	Large or dilated pupils.
mydriatics	Substances that produce mydriasis such as atropine or homatropine.
narcosis	To be under the influence of narcotics.
necrosis	Death of tissue.
necrotic	Pertaining to necrosis, end result of necrosis.

neostigmine	An anticholinesterase agent used in medical conditions to enhance acetylcholine action.
nerve agent	The most toxic of CW agents. It is an organic ester of phosphoric acid which has physiological effects (inhibition of cholinesterase). Nerve agents are absorbed into the body by breathing, by injection, or through the skin, and affect the nervous and the respiratory systems and various body functions. They include the G- and V-agents. Examples of G-agents are Tabun (GA), Sarin (GB), Soman (GD), and V-agent (VX).
Nerve Agent Antidote Kit (NAAK)	The nerve agent antidote used by the U.S. Armed Forces in the treatment of nerve agent poisoning. The kit consists of four separate components: the atropine auto-injector, the pralidoxime chloride autoinjector, the plastic clip, and the foam carrying case. Also called the MARK I.
Nerve Agent Pyridostigmine Pretreatment (NAPP) Tablet Set	A blister pack containing a pretreatment medication to be used with NAAK. The pack consists of twenty-one 30-mg pyridostigmine bromide tablets. When used in conjunction with the MARK I, this medication may enhances the service member's survivability when exposed to nerve agents.
nicotinic	Referring to the toxic effect of nicotine on autonomic ganglia, initially stimulating, then inhibiting neural impulses at the ganglia level as well as the neuromuscular junction.
nitric acid	A caustic and corrosive acid widely used in industry and chemical laboratories.
nitric oxide	An unstable chemical compound formed by passing air through an electric arc. Converts to nitrogen dioxide when exposed to air. Like other nitrogen compounds (nitrogen dioxide), it is extremely hazardous to breathe. Self-contained masks plus adequate ventilation are mandatory when exposed to even small amounts.
nitrogen dioxide	An irritating gas, one of several oxides of nitrogen, usually formed from nitrogen tetroxide or by the reaction of certain metals with nitric acid.
nitrogen mustard	A vesicant which attacks deoxyribonucleic acid (DNA). Is also used as a antineoplastic agent (classed as an alkylating agent). Several were developed as CW agents. Also produces pulmonary injury and bone marrow depression.
nitrogen tetroxide	An unstable compound that readily decomposes to nitrogen dioxide.
N-methylglucarnine	A chemical compound used as a V-agent simulant, with significant irritant properties.
nonpersistent agent	A chemical agent that disperses or vaporizes rapidly after release and presents an immediate short duration hazard. These agents are generally released as aerosols, gases, vapors, liquids, or solids.
norepinephrine	An epinephrine-like hormone secreted by the adrenal medulla, with primary effect as vasoconstrictor as compared to epinephrine (which primarily increases heart rate and cardiac output).
noxious chemicals	Included in this category are gases such as carbon monoxide (CO), oxides of nitrogen, chlorine vapor, hydrogen sulfide, and ammonia.
O-chlorobenzylidene malononitrile	A tear gas used primarily as a riot control agent. Potent eye, throat, and skin irritant, but incapacitation 'is short-lived.
octamethyl pyrophosphoramide (OMPA)	An organophosphate insecticide. Like organophosphates in general, it inhibits acetylcholinesterase.

opacification	The condition of blocking the transmission of light as opacification of the cornea or lens of the eye.
ophthalmic	Pertaining to the eye.
ophthalmologist	A physician who specializes in the diagnosis and medical and surgical treatment of diseases and defects of the eye and related structures.
opiate	A derivative of opium or containing opium.
organophosphate	A compound with a specific phosphate group which inhibits acetylcholinesterase. Used in CW and as an insecticide.
oropharyngeal airway	A short airway inserted into the oropharynx to prevent the tongue from obstructing the airway.
oropharynx	That portion of the pharynx associated with and posterior to the mouth, namely from the soft palate to the epiglottis.
overatropinization	Too much atropine; may cause psychosis.
oxime	A compound that can remove the cholinesterase inhibition from the cholinesterase providing that aging has not occurred. Oxime is used in the therapy of nerve agent poisoning.
pannus	A covering over the cornea of the eye, usually from superficial vascular tissue, producing a cloudy vascular film. Seen in some diseases or as a result of irritation.
paralyzing agent	Any agent that prevents the use of certain muscles or groups of muscles.
parasympathomimetic	The effects obtained from stimulation of the parasympathetic portion of the autonomies nervous system, causing cholinergic effects.
parenchyma	The functioning part of an organ as contrasted to its structural parts. Parenchyma of the stomach are the secreting glands which produce acid, mucous, and so forth, as contrasted to the stomach wall which provides structure.
pathognomonic	A sign or symptom specifically distinctive of a disease.
percutaneous	Through the skin, such as applying an ointment with medication or injection by needle.
peristalsis	A wave-like contraction in an organ, such as the intestines, which propels the contents.
pernicious anemia	One form of anemia associated with a lack of vitamin B,, and other factors. Usually responds to B_{12} and iron diet.
persistent agent	A chemical agent that continues to present a hazard for considerable periods after delivery by remaining as a contact hazard and / or by vaporizing very slowly to produce a hazard by inhalation. Generally, may be in a solid or liquid state.
phagocytosis	The engulfing of microorganisms and foreign particles by cells called phagocytes.
pharyngitis	Inflammation of the pharynx.
pharynx	The air passageway from the posterior nose to the trachea.

phenol	An ingredient used in calamine lotion (1 percent).
phenothiazine	A group of psychotherapeutic medications with a phenothiazine structure which act by adrenergic blocking. They have antiemetic, antihistaminic, and antispasmodic activity in addition to CNS effects.
phenyldichloroarsine	A vesicant of the L group.
phosgene	Carbonyl chloride, a chemical warfare agent used in World War I (was leading cause of death). Causes severe pulmonary irritation and injury.
phosgene oxime	Dichloroformoxime. A vesicant, as well as a lung irritant, used as a chemical warfare agent.
phosphoric acid	A tribasic acid.
photophobia	Literally, fear of light. Occurs when light becomes painful to the eyes.
physical characteristics of chemical agents	Chemical agents cover the whole spectrum of physical properties. Their physical state may be aerosol, gaseous, liquid, or solid under normal conditions. Their vapor pressure (the force exerted by the vapor when in equilibrium with the liquid or solid at a given temperature) may be high or negligible. Their vapor density varies from slightly lighter than air to considerably heavier than air. Their range of odors varies from none to highly pungent. They may be soluble or insoluble in water, fats, or organic solvents. The physical characteristics may give an indication of the behavior of the agents in the field with regard to vapor hazard, persistency, decontamination methods required, and personal and subsistence protection required.
physostigmine	A reversible anticholinesterase permitting an accumulation of acetylcholine (cholinergic). It readily crosses the blood-brain barrier. It improves the tone and action of skeletal muscles, increases intestinal peristalsis, acts as a miotic in the eye, and is used in treatment of BZ.
physostigmine salicylate	See physostigmine.
pneumonia	Inflammation of the lungs, usually caused by an infective agent. May be secondary to injury to the lungs.
Positive end-expiratory pressure (PEEP)	A method of ventilating a patient where positive pressure is maintained in the lungs at the end of the expiatory cycle, thus maintaining a higher pressure than the pulmonary circulation which reduces the pooling or shunting of blood in the lungs.
pralidoxime	An oxime used in the treatment of organophosphate insecticides and nerve agent poisoning to block the inhibition of acetylcholinesterase by nerve agents.
prednisolone	A steroid (glucocorticoid) used in the treatment of choking agents over a course of several days.
preganglionic	The nerve fiber leading to a ganglion.
pruritus	Itching.
pulmonary edema	Swelling of the cells of the lungs, associated with an outpouring of fluid from the capillaries into the pulmonary spaces, producing severe shortness of breath. In later stages, produces expectoration of frothy pink serous fluid and cyanosis.

reserpine	A medication used in the treatment of high blood pressure.
rhinitis	Inflammation of the nasal mucosa.
rhinorrhea	Thin watery discharge from the nose.
riot control agent	A chemical which produces transient effects that disappear within minutes of removal from exposure and very rarely require medical treatment. Riot control agents are effective in quelling civil disturbances and in some military operations, to preclude unnecessary loss of life.
saprophytic	Pertaining to deriving its growth from other living or dead matter.
Sarin	A nerve agent of the organophosphate group which inhibits acetylcholinesterase.
smokes	An obscurant system in which one or more solids are dispersed in a vapor or gas. Smokes are made from special petroleum oils such as SGF2, HC, FM, FS, and WP.
sodium bicarbonate	Commonly called baking soda. Has many uses, including use in irrigating solutions, especially for the eyes.
sodium carbonate	An antacid. Also used as a solution for decontaminating the skin to remove irritants. Can be used as a detergent.
sodium hypochlorite	Bleach, a source of chlorine, with decontamination and disinfectant properties.
sodium nitrite	A hypotensive agent and methemoglobin former, used as an antidote for cyanide poisoning to sequester the cyanide agent.
sodium sulfacetarnide	A medication used either as an ointment or solution in the eye. It is a mild anti-bacterial agent.
sodium sulfite	A compound used (when in solution) as a decontaminant for skin irritants.
sodium thiocyanate	The metabolize formed by the action of sodium thiosulfate on cyanide as an antidote, which is then excreted from the body.
sodium thiosulfate	An antidote for cyanide or as a source of sulfhydryl groups for other actions in the body. If used for cyanide poisoning, it should be preceded with sodium nitrite.
Soman	A nerve agent member of the organophosphate group; inhibits acetylcholinesterase. Used as a chemical warfare agent.
sternutator	An agent which induces coughing and sneezing.
steroid	See corticosteroid.
substernal	Under the sternum.
sulfur trioxide-chlorosulfonic acid solution	An obscurant usually dispensed from aircraft, forms hydrochloric and sulfuric acid on contact with moisture. Is irritating to the eyes, respiratory tract, and skin.
sulfuric acid	An acid from sulfim, oxygen, and hydrogen used in industry. It is caustic and corrosive.
synechia	Adhesion of parts, especially adhesion of the iris to the lens and cornea.

systemic poison	A poison that affects the whole body.
Tabun	A nerve agent member of the organophosphate group which inhibits acetylcholinesterase. Is used as a chemical warfare agent. Is the least toxic of the nerve agents but can cause death rapidly.
therrnite	Incendiaries that are a mixture of powdered iron oxide, powdered aluminum, and other materials.
thrombocytopenia	An absolute decrease in the circulating platelets in the blood.
titanium dioxide	A breakdown product of FM which can be irritating to the eyes and skin.
titanium oxychloride	One of the three components of FM.
titanium tetrachloride	A petroleum base oil that is converted into smoke for battlefield obscuration. May be irritating to eyes and respiratory tract.
tracheobronchial	Pertaining to the portion of the airway starting at the neck and passing into the lungs.
tracheotomy	An opening made into the trachea to permit air to flow directly into the trachea, and bypassing the nose and mouth.
tranquilizer	A medication used in the treatment of various psychoneurotic, neurotic, and psychotic disorders. Major tranquilizers are used for psychoses and include phenothiazines, thioxanthenes, and butyrophenones. Minor tranquilizers are used for treatment of neuroses and anxiety states and include certain barbiturates, the benzodiazepines, and other drugs.
triamcinolone	A steroid used for many purposes, including as an anti-irritant following exposure of the skin to CN and CA agents.
ulceration	Breaking down of a surface (such as the skin or mucous membrane) to form an ulcer.
urticant	A skin irritant which causes itching or a raised red area (wheal).
US Army Field Medical Card (DD Form 1380)	A card used to record the medical diagnosis, medication, and treatment given for all illnesses or injuries (including chemical agent injuries) and, if known, the contaminating agent. It is also used to record the disposition of casualties who are dead on arrival at the battalion aid or division clearing station or who died of wounds, injury, or illness.
vacuolation	Formation of a space.
V-agent (VX)	A nerve agent of the organophosphate group that inhibits acetylcholinesterase.
vascularization	Development of new blood vessels in a structure.
vasoconstriction	Diminution of the interior size of a blood vessel with resultant decrease in blood flow.
vertigo	Dizziness, where space seems to move around.
vesicant	A chemical blister agent which injures the eyes and the lungs and burns or blisters the skin. Examples are HD, L, and CX.
vesication	Blistering.

vesiculation	The process of blistering.
vomiting agent	DA, DM, and DC.
wheezing	A whistling sound made in breathing, usually cause by partial obstruction of the airways.
white phosphorus (WP)	A form of phosphorus which creates spectacular bursts when used in artillery shells. Is very damaging to the skin since it continues to burn upon exposure to oxygen.

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