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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 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 toxic industrial chemicals).

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

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

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

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

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

(8) Describes procedures for administering the Nerve Agent Pyridostigmine Pretreatment tablets, Nerve Agent Antidote Kit (NAAK) (MARK I) and Antidote Treatment Nerve Agent Autoinjector (ATNAA) and Convulsant Antidote for Nerve Agent (CANA) (Appendix 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 cyanogen (blood) agents 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 toxic industrial 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 eliminating or neutralizing the chemical agent to a point that it no longer presents a health risk. Further the chemical agent hazard on equipment is reduced to a point that allows 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 may be 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 industrial chemical, smoke and incendiary materials.

g. Riot Control Agents. These are chemicals that produce 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. Toxic Industrial Chemicals. Chemical compounds used or produced in industrial processes that are toxic to humans and animals, or cause damage to plants. Examples include fuels, solvents, heavy metals, and chemicals used in manufacturing processes. 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.

Implementation Plan

Participating Service command offices of primary responsibility (OPR) will review this publication, validate the information, reference, and incorporate it in Service and command manuals, regulations, and curricula as follows:

a. Army. The Army will incorporate this publication in United States (US) Army training and doctrinal publications as directed by the Commander, US Army Training and Doctrine Command (TRADOC). Distribution is in accordance with initial distribution number (IDN) 115861, requirements for FM 8-285.

b. Marine Corps. The Marine Corps will incorporate the procedures in this publication in US Marine Corps (USMC) training and doctrinal publications as directed by the Commanding general, US Marine Corps Combat Development Command (MCCDC). Distribution is in accordance with Marine Corps Publication Distribution System (MCPDS).

c. Navy. The Navy will incorporate these procedures in US Navy (USN) training and doctrinal publications as directed by the Commander, Navy Warfare Development Command (NWDC). Distribution is in accordance with MILSTRIP Desk Guide and NAVSOP Publication 409.

d. Air Force. The Air Force will validate and incorporate appropriate procedures in accordance with applicable governing directives. Distribution is in accordance with AFI 33-360.

e. Coast Guard. The Coast Guard will validate and refer to appropriate procedures when applicable. No material contained herein should conflict with Coast Guard regulations or other directives from higher authority, or supersede, or replace any order or directive issued by higher authority.

User Information

a. The US Army Medical Department Center and School developed this publication with the joint participation of the approving Service commands.

b. This publication reflects current Service and joint doctrine on prevention, protection, medical management, and treatment of nuclear and radiological casualties.

c. We encourage recommended changes for improving this publication. Key your comments to the specific page and paragraph and provide a rationale for each comment or recommendation. Send comments and recommendations directly to—

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References

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

PART ONE: CHEMICAL WARFARE AGENT CASUALTIES

CHAPTER 1

INTRODUCTION

1-1. The Threat of Chemical Warfare Agents to US Forces

a. Chemical warfare (CW) agents remain a continuing threat to US forces. Delivery may be accomplished by multiple means, causing extensive injury and contamination. Release of agents may also be due to collateral damage to enemy storage facilities or destruction of their munitions by friendly forces bombing or artillery fire. Traditionally, enemy commanders have regarded CW agents as a part of their conventional arsenal. The Chemical Weapons Convention (CWC), which banned the use of CW agents and was signed by 140 countries in January 1993, will take many years to fully implement. Not all countries have signed the CWC. In spite of the CWC and other diplomatic efforts, CW agents will be available to threat forces in regions where US forces may be deployed.

b. Chemical warfare agents 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. Chemical warfare agents are most effectively employed against untrained or unprotected targets. Civilian fixed sites (airfields, depots, cities, and ports) are especially vulnerable and may be targeted as part of a plan to defeat US force projection. Chemical warfare agents can also be encountered in a variety of situations off the battlefield.

1-2. Military Employment of Chemical Warfare Agents

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

b. Chemical warfare agents can be used in conjunction with other weapons systems or by themselves. These agents may produce temporary incapacitating effects, serious injury, or death. Chemical warfare agents also have the potential for use by saboteurs and terrorists in rear areas against key targets and civilian populations. The scope of CW agents is broad since they target groups rather than individuals and could be directed against civilian populations. Vapors of CW 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 agent operations can create psychological and 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. Service members 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 warfare agents may enter the body by several routes. When inhaled, gases, vapors, and aerosols may be absorbed by 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. Wounds or abrasions are presumed to be more susceptible to absorption than is the intact skin.

1-4. Classification of Chemical Warfare Agents

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

a. Physiological Action.

(1) 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 noncardiogenic pulmonary edema and predispose to secondary pneumonia.

(2) Nerve agents (anticholinesterases), such as tabun (GA), sarin (GB), soman (GD), cyclosarin (GF), and V agents (for example, O-ethyl methyl phosphonothiolate [VX]), inhibit the cholinesterase enzymes. The cholinesterase enzymes hydrolyze acetylcholine, a chemical neurotransmitter. Inhibition of those enzymes creates an accumulation of acetylcholine at cholinergic synapses that disrupts the normal transmission of nerve impulses, causing cholinergic crisis. Cholinergic receptors 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.
- On smooth muscle of the gastrointestinal tract.
- On smooth muscle of the respiratory tract.

(3) Cyanogen (blood) agents include hydrogen cyanide (AC) and cyanogen chloride (CK). These agents are transported by the blood to all body tissues, where they block the oxidative processes, preventing tissue cells from utilizing oxygen. The CNS is especially sensitive to this anoxia, and toxicity with these agents leads to cessation of respiration followed by cardiovascular collapse.

(4) Blister agents (vesicants) include sulfur mustard (HD), nitrogen mustards (HN), arsenicals such as Lewisite (L), and phosgene oxime (CX) (technically an urticant). 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.

(5) Incapacitants are chemicals designed to temporarily disable an individual, but they do not cause permanent injury or death. Although a variety of different types of chemicals are classified as incapacitants, predominant among these are chemicals with anticholinergic properties that block the effect of acetylcholine on receptor sites and at neuronal synapses. As a result, symptoms are exactly the

opposite one would see with nerve agents and include erythema, decreased salivation, urinary retention, mydriasis (dilation of the pupils with decreased visual acuity), hyperthermia, and mental status changes.

b. Military Use.

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

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

1-5. Means of Delivery of Chemical Warfare Agents

Chemical warfare agents can be dispersed by explosive shells, rockets, missiles, aircraft bombs, mines, and spray devices. Water supplies have the potential for contamination by either water-soluble or miscible liquids or solids, although effective concentrations are difficult to maintain. The means of delivery does not in itself help in identifying CW agents. A spray or cloud delivered from an aircraft or by shells and bombs may indicate that a chemical attack is taking place. Vapors delivered from aircraft may not be visible and vapors and sprays may be hidden by atmospheric conditions.

1-6. Diagnosis of Injury from Chemical Warfare Agents

a. Odor. Some agents have odors which may aid in their detection and identification (Table 1-1), but many are essentially odorless. The odor of a CW agent delivered by an explosive shell may be concealed by the odor of the burning explosive. Detection of a CW agent odor is one indication for immediately putting on the mask and wearing it until the "all clear" signal is given. Odor alone must not be relied on for detection or identification of a CW agent. Some CW 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 may be specific to a given state (such as vapor but not liquid, or vice versa) and may indicate agent presence in their immediate area only. They may not cover large areas and thus should not be the sole means on which to base conclusions on the presence or absence of CW agents.

b. Signs and Symptoms. These include the following:

(1) A brief history eliciting symptoms and their progression.

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

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

1-7. Protective Measures and Handling of Chemical Warfare Agent Casualties

a. Depending on the theater of operations, guidance may dictate the assumption of a minimum mission-oriented protective posture (MOPP) level. Mission-oriented protective posture (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 troop movement, once CW agent use has been suspected.

- When casualties are being received from an area where CW agents have reportedly been used. Appendix A provides additional information on recognizing CW agent casualties.

b. The mask should be worn until detection procedures indicate the air is free of CW agent and the "all clear" signal is given by authorized personnel (see FM 3-11.4 for unmasking procedures).

c. It is the responsibility of all individuals to decontaminate themselves or to decontaminate other personnel in their unit. Contaminated casualties may arrive at a medical treatment facility (MTF), presenting a hazard to unprotected personnel. Handlers must wear their individual protective equipment (IPE) while handling these casualties. A casualty decontamination area is located downwind (prevailing winds) of designated MTFs. Contaminated clothing and equipment are placed in plastic bags and removed to a designated dumpsite downwind from the MTF (see Appendices B and C).

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

e. Military commanders, leaders, and medical personnel should be on the alert for the possibility of anxiety reactions (combat/operational stress reactions [COSR]) among personnel during CW 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 (-12.2° C) to the WBGT index. (See FM 3-11.4 for additional guidance on the degradation effects of the protective clothing.)

f. Military commanders, leaders, and medical personnel should be on the alert for unexposed personnel self-administering antidotes. Administration of atropine without exposure to nerve agents can stop the individual's ability to perspire, resulting in potentially severe heat injury.

1-8. Chemical Warfare Agent Contamination Detection and Identification

Identification of CW agents will greatly assist in the diagnosis and treatment of chemical injuries (reference FM 4-02.7). The following are means of detecting and identifying chemical agent contamination:

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

(1) The 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 CW agent vapors. Some solvents and standard decontaminating solutions cause false-positive reactions by the M8 paper.

(2) The M9 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 (HD, HN, and L). The M9 tape does not distinguish between the types of agent; it signifies merely the presence of an agent. Neither will

it detect CW agent vapors. Extremely high temperatures, scratches on the tape, or certain organic liquids cause M9 tape false-positive reactions.

b. Automatic CW agent alarm systems and the improved chemical agent monitor (ICAM) 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 cyanogen 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 medical treatment, decontamination, emergency medical treatment (EMT), advanced trauma management, evacuation, and continuing protection of CW agent casualties (Appendix C).

1-10. Personal Decontamination

Personal decontamination must be carried out immediately. For those individuals who cannot decontaminate themselves, the nearest able person should assist them as the situation permits. Decontamination consists of either agent removal and/or neutralization; agent removal is preferred. Refer to Appendix D for decontamination procedures.

1-11. Casualty Decontamination

Contaminated casualties entering the medical treatment system are decontaminated through a decentralized process. Units should further decontaminate the casualty before evacuation. Casualty decontamination stations are established at all levels of care to decontaminate individuals as required prior to entry into collective protection. Medical supervision is required to prevent further injury to the casualty and to provide 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 casualty decontamination, see FM 4-02.7 , FM 3-5, and Appendix D.)

1-12. First Aid

First aid comprises self-aid, buddy aid, or aid provided by those nonmedical personnel trained as combat lifesavers (Army).

a. Self-Aid. Self-aid consists of measures that service members can apply in helping themselves. These include individual decontamination, administration of CW agent antidotes, and assuming the appropriate level of MOPP.

b. Buddy Aid. Buddy aid consists of emergency actions to restore or maintain vital body functions in a casualty who cannot adminster self-aid. 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 follow these steps in order:

(1) Mask the casualty, if not already masked.

(2) Administer antidotes.

(3) Decontaminate the casualty.

(4) Put remaining protective clothing on the casualty to preclude further absorption of contamination through any exposed skin.

(5) Evacuate the casualty as soon as possible.

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

(1) Administering additional atropine.

(2) Administering additional CANA.

(3) Establishing an oropharyngeal airway.

(4) Starting intravenous (IV) infusions.

1-13. Medical Treatment

Medical treatment consists of those procedures undertaken to return service members 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 CW agent casualties. Specific CW agent treatment procedures are described in the ensuing chapters.

1-14. Medical Evacuation

a. Casualties requiring evacuation should be decontaminated, if possible, before evacuation. In many instances, the casualty must be evacuated to the first level of care before complete decontamination. Ground ambulances are the preferred conveyances to evacuate the casualties in contaminated forward areas, when feasible. This does not mean that rotary-wing air medical evacuation assets should not be used. When used, the numbers of assets committed to evacuation within the contaminated area should be limited; once contaminated, the same evacuation assets should be repeatedly used in the contaminated area until all casualties have been evacuated.

b. During mass casualty situations, commanders may be required to employ nonmedical vehicles/aircraft for casualty evacuation (CASEVAC). En route care is not available for CASEVAC. If medical personnel augmentation is available, limited en route care may be available.

c. For detailed information on medical evacuation see JP 4-02.2, FM 8-10-6, and FM 4-02.7.

1-15. Individual Prescriptions

All Force Health Protection Prescription Products (FHPPP) will be issued under a prescription by qualified personnel who have been instructed on exclusion criteria and other medical guidance applicable to the product. A blanket prescription may be issued by a physician serving as the Assistant Secretary of Defense (Health Affairs) (ASD[HA]), the Surgeon General of the Army, Navy, or Air Force, or the Command Surgeon of a Combatant Command. Although the inclusive list of FHPPP may vary between areas of responsibility based on differing threats, examples of such products include atropine/2-pralidoxime chloride (2-PAM Cl) autoinjectors; certain antimicrobials, including antimalarials; and pyridostigmine bromide. The provision or issuance of FHPPP shall be documented in medical records of the personnel or individuals receiving the FHPPP. For more information, refer to ASD(HA) policy memo, 24 April 2003, and AR XX "Prescription Only Requirements for Individually Issued NBC Medical Defense Items."

1-16. Investigational New Drugs and Off-Label Indications

a. DoDD 6200.2, "Use of Investigational New Drugs for Force Health Protection" directs that when, at the time of the need for a force health protection countermeasure against a particular threat, no safe and effective drug or biological product is available that has been approved by the United States Food and Drug Administration (FDA), the DoD Components may request approval of the Secretary of Defense to use an investigational new drug (IND). An IND is subject to the FDA regulations 21 CFR and includes—

- A drug not approved or a biological product not licensed by the FDA.
- A drug unapproved for its applied use.

Such requests must be justified based on available evidence of the safety and efficacy of the drug and the nature and degree of the threat to personnel.

b. When using INDs for force health protection, the DoD Components will comply with United States Code Title 10 (section 1107) and Executive Order 13139, "Improving Health Protection of Military Personnel Participating in Particular Military Operations," September 30, 1999 and applicable FDA regulations.

c. The Secretary of the Army, as Executive Agent, and in concert with the Commander of the Combatant Command involved and the ASD(HA), will develop a specific treatment protocol for the use of the IND. The protocol will provide for the prior informed consent of members receiving the IND. Under Title 10, only the President may grant a waiver of informed consent to use an IND for force health protection in connection with members' participation in particular military operations and only the Secretary of Defense may request that the President grant such a waiver.

d. When using an IND for force health protection, the DoD components will—

- Inform persons receiving the drug or biological product that it is an IND.
- Explain the reason the IND is being used.
- Provide information regarding the possible side effects of the IND.
- Ensure that medical records of personnel receiving the IND are accurately documented.

Healthcare providers and those in leadership positions will participate in ongoing training and health risk communication in the administration of INDs.

Table 1-1. Summary of Chemical Agent Effects

AGENT	SYMBOL	ODOR	MECHANISM OF ACTION	EYES			NOSE AND THROAT	
				PUPILS	CONJUNCTIVAL	REST OF EYE		
TABUN SARIN SOMAN CYCLOSARIN	GA GB GD GF	NONE, OR FAINT SWEETISHNESS, FRUITY OR PAINT-LIKE	ANTICHOLINESTERASE AGENTS	MIOSIS	REDNESS	PAIN (ESPECIALLY ON FOCUSING), DIMNESS OF VISION, HEADACHE, LACRIMATION	INCREASED SALIVATION, RHINORRHEA	
VX	VX	NONE						
MUSTARD	H HD	GARLIC OR HORSERADISH, 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	
NITROGEN MUSTARD	HN	NONE OR FISHY, IRRITATING						
LEWISITE AND OTHER ARSENICAL VESICANTS	L	FRUITY TO GERANIUM-LIKE, VERY IRRITATING	VESICANTS. ARESENICAL POISONS		PROMPT REDNESS, EDEMA, IRRITATION	IMMEDIATE BURNING SENSATION, IRITIS, CORNEAL INJURY	PROMPT IRRITATION.	
MUSTARD/ LEWISITE MIXTURE	HL	GARLIC-LIKE	LIKE LEWISITE AND MUSTARD	LIKE HD, HN, AND L				
PHOSGENE OXIME	CX	UNPLEASANT AND IRRITATING	POWERFUL VESICANT		VIOLENTLY IRRITATING, REDNESS, EDEMA	LACRIMATION, CORNEAL INJURY WITH BLINDNESS	VERY IRRITATING TO MUCOUS MEMBRANES	
PHOSGENE	CG	GREEN CORN, GRASS, OR NEW-MOWN HAY	LUNG-DAMAGING AGENT		IRRITATION	LACRIMATION (AFTER RESPIRATORY SYMPTOMS)	IRRITATION	
HYDROGEN CYANIDE	AC	FAINT, BITTER ALMONDS	INTERFERES WITH OXYGEN UTILIZATION AT CELLULAR LEVEL					
CYANOGEN CHLORIDE	CK	VERY IRRITATING	LIKE HYDROGEN CYANIDE, LUNG IRRITANT		IRRITATION	LACRIMATION	IRRITATION	
VOMITING AGENTS	DM DA DC	BURNING FIREWORKS, VERY IRRITATING	LOCAL IRRITANT, INDUCES VOMITING		IRRITATION	LACRIMATION	PAIN, RHINORRHEA, TIGHTNESS, SNEEZING	TIGHTNESS
IRRITANT AGENTS	CN CA	IRRITATING	LOCAL IRRITANT		REDNESS, IRRITATION	PAIN, BLEPHAROSPASM, PROFUSE LACRIMATION, PHOTOPHOBIA	IRRITATION, BURNING	TIGHTNESS, BURNING
	CS CR	VERY IRRITATING, PUNGENT, PEPPER-LIKE	LOCAL IRRITANT		INTENSE IRRITATION	PAIN, BLEPHAROSPASM, PROFUSE LACRIMATION, PHOTOPHOBIA	IRRITATION BURNING, TIGHTNESS	TIGHTNESS, BURNING
INCAPACITATING AGENTS	BZ	NONE	ANTICHOLINERGIC	MYDRIASIS		BLURRED VISION	EXTREME DRYNESS	EXTREME DRYNESS
	LSD	NONE	PSYCHOTOMIMETIC	MYDRIASIS				

Table 1-1. Summary of Chemical Agent Effects, Continued

AGENT	RESPIRATORY TRACT	SKIN	GI TRACT	CARDIOVASCULAR SYSTEM	GENITOURINARY SYSTEM
TABUN SARIN SOMAN CYCLOSARIN VX	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
MUSTARD NITROGEN MUSTARD	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	
LEWISITE AND OTHER ARSENICAL VESICANTS	RAPID IRRITATION, HOARSENESS, APHONIA, COUGH. PNEUMONIA, FEVER, PULMONARY, EDEMA IN SEVERE CASES, PLEURAL EFFUSION	PROMPT BURNING. REDNESS WITHIN 30 MINUTES. BLISTERS ON 1ST OR 2D DAY. PAIN WORSE AND NECROSIS DEEPER THAN H	DIARRHEA, NAUSEA, VOMITING, HEPATIC FAILURE	SHOCK AFTER SEVERE EXPOSURE. HEMOLYTIC ANEMIA. HEMOCONCENTRATION	RENAL FAILURE
MUSTARD/ LEWISITE MIXTURE	LIKE HD, HN, AND L				
PHOSGENE OXIME	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)			
PHOSGENE	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	
HYDROGEN CYANIDE	DEEP RESPIRATION FOLLOWED RAPIDLY BY DYSPNEA, GASPING, THEN CESSATION OF RESPIRATION	INITIALLY PINKER THAN USUAL; MAY CHANGE TO CYANOSIS	NAUSEA	PROFOUND HYPERTENSION	
CYANOGEN CHLORIDE	IRRITATION, COUGH, CHOKING, DYSPNEA: PULMONARY CAN BE RAPID	LIKE AC			
VOMITING AGENTS	TIGHTNESS AND PAIN, UNCONTROLLABLE COUGHING	STINGING (ESPECIALLY OF FACE), OCCASIONAL DERMATITIS	SALIVATION, NAUSEA, VOMITING		
IRRITANT AGENTS	TIGHTNESS AND IRRITATION IF CONCENTRATION IS HIGH	STINGING (ESPECIALLY OF FACE), OCCASIONAL DERMATITIS, MAY BLISTER	OCCASIONAL VOMITING		
	TIGHTNESS IN CHEST AND DIFFICULTY BREATHING	STINGING, OCCASIONAL DERMATITIS, MAY BLISTER	NAUSEA AND VOMITING		
INCAPACITATING AGENTS BZ		DRY, FLUSHED	CONSTIPATION	TACHYCARDIA, ELEVATED BLOOD PRESSURE	URGENCY, URINARY RETENTION
INCAPACITATING AGENTS LSD		SWEATY PALMS, COLD EXTREMITIES		TACHYCARDIA	

Table 1-1. Summary of Chemical Agent Effects, Continued

AGENT	CENTRAL NERVOUS SYSTEM	OTHER	DECONTAMINATION	TREATMENT
TABUN SARIN SOMAN CYCLOSARIN VX	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 FOR SOMAN POST-EXPOSURE THERAPY: (1) CHOLINERGIC BLOCKADE—ATROPINE (2) ENZYME REACTIVATION—OXIMES (2 PAM C1) (3) ANTICONVULSANT—DIAZEPAM (CANA) (4) ASSISTED VENTILATION (5) SUCTION FOR RESPIRATORY SECRETIONS
MUSTARD NITROGEN MUSTARD	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 FIELD MTF, WITH A SODIUM BICARBONATE OR SALINE EYEWASH. REMOVE CONTAMINAED 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
LEWISITE AND OTHER ARSENICAL VESICANTS	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
MUSTARD/ LEWISITE MIXTURE	LIKE HD, HN, AND L			LIKE SULFUR MUSTARD, NITROGEN MUSTARD AND LEWISITE
PHOSGENE OXIME	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
PHOSGENE	ANXIETY, DEPRESSION			CORTICOSTERIODS IV AND BY INHALATION PROMTPLY MAY BE LIFESAVING. REST, OXYGEN, ANTIBIOTICS
HYDROGEN CYANIDE	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
CYANOGEN CHLORIDE	LIKE AC			LIKE AC AND CG
VOMITING AGENTS	SEVERE HEADACHE, MENTAL DEPRESSION	MAY CAUSE DESIRE TO REMOVE PROTECTIVE MASK		WEAR MASK IN SPITE OF SYMPTOMS. SPONTANEOUS IMPROVEMENT
IRRITANT AGENTS	HEADACHE		WASH EYES WITH COPIOUS AMOUNTS OF WATER	SPONTANEOUS IMPROVEMENT. ANALGESIC EYE AND NOSE DROPS IF NECESSARY
	HEADACHE		WASH EYES WITH COPIOUS AMOUNTS OF WATER	SYMPTOMS DISAPPEAR RAPIDLY IN FRESH AIR
INCAPACITATING AGENTS — BZ	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
INCAPACITATING AGENTS — LSD	MENTAL EXCITATION, POOR CONCENTRATION, TREMOR, INDECISIVENESS, INABILITY TO ACT IN A SUSTAINED OR PURPOSEFUL MANNER. HALLUCINATIONS	PYREXIA		REASSURANCE, RESTRAINT, PROMPT EVACUATION, DIAZEPAM (CANA)

CHAPTER 2

LUNG-DAMAGING AGENTS (CHOKING AGENTS)

2-1. General

Chemical agents that primarily cause pulmonary edema by attacking lung tissue have traditionally been classified as lung-damaging agents (choking agents), or pulmonary edematogenic agents. They include phosgene (CG), diphosgene (DP), chlorine, and chloropicrin (PS). Best known of these agents is CG. There are also numerous toxic industrial chemicals (TICs) or products of combustion that pose a primary threat similar to lung-damaging agents. Smokes are covered in Chapter 8 and TICs, including chlorine and oxides of nitrogen, are covered in Chapter 10. Agents causing pulmonary edema by damaging capillary endothelia in alveolar septa are also called peripheral pulmonary agents because they affect the peripheral compartment (those airways distal to the terminal bronchioles). Central pulmonary agents are compounds that irritate and damage the larger, or central, airways. The terms lung-damaging agents, choking agents, and respiratory irritants are sometimes ambiguous and are not so specific as the terms centrally acting pulmonary agents and peripherally acting pulmonary agents (pulmonary edematogenic agents). Pure central and pure peripheral effects represent two ends of a spectrum; some agents, such as chlorine, exhibit central and peripheral effects in roughly equal proportions.

a. Central pulmonary agents.

(1) *The central compartment of the respiratory tree.* The central compartment, or tracheobronchial region, of the respiratory tract can be defined physiologically as that portion of the airways in which bulk air flow—flow with appreciable velocity—occurs. This includes the trachea, bronchi, and bronchioles down to the level of respiratory bronchioles.

(2) *Factors characterizing agents.* These agents tend to be very soluble in water and other aqueous media and very chemically reactive. They dissolve in and react with the first moist tissue they encounter, the tissue of the central compartment. At low doses, they may be essentially consumed by dissolving into and reacting with tissue in the central compartment; at high doses, they can reach the peripheral compartment as well.

(3) *Representative agents.* Strong acids and bases such as hydrogen chloride, hydrogen fluoride, acetic acid, and ammonia (NH_3) act as central agents. Agents that are intermediate in solubility and reactivity tend to affect both central and peripheral compartments relatively equally. Sulfur mustard, even though officially classified as a vesicant, can be regarded as the prototypical central pulmonary CW agent.

(4) *Pathophysiology.* After dissolving in aqueous solutions, central pulmonary agents typically release hydrogen ions. These ions kill the delicate epithelial cells that line the airways of the central compartment. The necrotic epithelium may slough off and can occlude airways. Alternately, the epithelium may be released in membrane-like sheets. These sheets are not true membranes but rather pseudomembranes, of the type seen in diphtheria; and they can also obstruct airways. Effects on the peripheral airways may be seen with central pulmonary agents, but chiefly at high doses. At these doses, the generation of oxygen free radicals may predominate over release of hydrogen or ions.

(5) *Clinical presentation.* Identification of a particular CW agent is important mainly as a means of predicting, identifying, and managing central vs. peripheral pulmonary damage. Central pulmonary agents produce irritation (a symptom) and noise (a sign). The clinical hallmark of central damage to the central compartment is airway noise. Casualties may cough, sneeze, become hoarse, exhibit inspiratory stridor, or develop coarse rhonchi or wheezing. In severe cases, irritation may lead to

apnea from reactive laryngospasm. For most central pulmonary agents, irritation and noise occur relatively soon after exposure, although these effects may be delayed with slowly dissolving but extremely reactive agents such as sulfur mustard.

(6) *Management.* Management should be primarily focused on the type of damage to the airway rather than on the agent since agents in different doses may produce only one kind of effect or both kinds of effects. Treatment of central pulmonary damage involves administration of warm, moist air, treatment of bronchoconstriction with bronchodilators in the case of irritative bronchospasm or in those with underlying reactive airways, and removal of necrotic debris by percussion, postural drainage, and, if available, bronchoscopy. Administration of supplemental oxygen is recommended, especially in cases in which the estimated inhaled dose raises the suspicion of eventual peripheral-compartment effects in addition to central-compartment effects.

b. *Peripheral pulmonary agents.*

(1) *The peripheral compartment of the respiratory tract.* The peripheral compartment, or gas-exchange region, of the respiratory tract can be defined physiologically as that portion of the airways in which bulk air flow is absent during each breath. This comprises the respiratory bronchioles, alveolar ducts, alveolar sacs, and alveoli, that is, the portion of the respiratory tract distal to the terminal bronchioles.

(2) *Factors characterizing agents.* Peripheral pulmonary agents tend to be relatively insoluble in water and other aqueous media and are chemically unreactive. At high doses, both compartments of the airway can be affected either by a central or a peripheral pulmonary agent

(3) *Representative agents.* The World War I agents CG and DP are relatively insoluble and chemically unreactive and exhibit peripheral effects at low to moderate concentrations. Perfluoroisobutylene (PFIB) (a high-temperature combustion product of polytetrafluoroethylene, or Teflon®); isocyanates; oxides of nitrogen; and hexachloroethane, grained aluminum, and zinc oxide (HC) smoke also exhibit peripheral effects. Chloropicrin, chlorine, chloramines, and to some extent ozone are intermediate in aqueous solubility and chemical reactivity and tend to produce central and peripheral effects in roughly equal proportions. Lewisite (L) has irritative central effects similar to those of HD but also damages pulmonary endothelial cells and leads to peripheral-compartment effects as well. Phosgene can be regarded as the prototypical peripheral pulmonary CW agent.

(4) *Pathophysiology.* Peripheral pulmonary damage is characterized by reactions of carbonyl groups (as in CG) with tissue in the endothelial cells lining pulmonary capillaries. These capillaries begin to leak fluid into the normally thin alveolar septa separating the capillaries from the alveolar spaces, and the septa expand from the influx of fluid. Fluid eventually seeps into the alveoli, tracks up respiratory and terminal bronchioles, and may spill over into even large bronchi. The term for this type of effect is “noncardiogenic pulmonary edema,” or “dry-land drowning”; peripherally acting pulmonary agents are therefore often called pulmonary edematogenic agents. At high doses, other reactions, such as liberation of hydrogen ions, can also cause irritation and damage to tissue in the central compartment. Oxides of nitrogen and HC smoke appear to have an additional immunological component leading in many cases to apparent recovery of acute effects followed by extensive and in some cases irreversible pulmonary fibrosis (cryptogenic organizing pneumonia).

(5) *Clinical presentation.* Identification of a particular CW agent is important mainly as a means of predicting, identifying, and managing central vs. peripheral damage. The clinical hallmark of damage to the peripheral compartment is dyspnea (shortness of breath) which results from fluid expansion of alveolar septa. This dyspnea usually occurs only after an hours-long clinically asymptomatic period that is inversely proportional to dose, and it can be brought on earlier by exertion. Because the hallmark of peripheral pulmonary damage is a symptom (delayed dyspnea) rather than a sign (noise), the absence of abnormal signs on clinical examination should not be used to exclude damage to the peripheral

compartment; neither should the initial absence of dyspnea. Irritation may be absent or so mild that victims of low doses may not be aware of being poisoned. With higher doses, initial irritation may present as coughing or sneezing; however, these signs usually subside after several minutes at most. Thus, disappearance of initial signs of irritation should not be used to exclude peripheral pulmonary damage. Eventually, crackles, decrease in arterial oxygen saturation, radiological indications of pulmonary edema, and dullness to percussion will be evident, but diagnosis before the occurrence of these relatively late signs is crucial. Most patients who survive the episode of pulmonary edema will recover without sequelae, but those exposed to oxides of nitrogen or HC smoke are at risk of late-onset pulmonary fibrosis heralded by cough, fever, chills, dyspnea, cyanosis, and radiological evidence of cryptogenic organizing pneumonia.

(6) *Management.* Management should be primarily focused on the type of damage to the airway rather than on the agent since agents in different doses may produce only one kind of effect or both kinds of effects. Management includes enforced rest (exertion leads to earlier appearance of effects and more severe effects), administration of supplemental oxygen, observation of clinically asymptomatic individuals, early evacuation of victims with relatively early-onset symptoms or with a significant likelihood of developing early-onset symptoms, and treatment of pulmonary edema in a pulmonary-intensive-care-unit setting. Antibiotics should not be used prophylactically but should be reserved for treatment of infections with culture-positive organisms. Bronchodilators and other treatments for central-compartment effects may be used as clinically indicated since high doses of peripheral pulmonary agents may also produce central effects; however, pulmonary edema by itself is not a usual indication for bronchodilator therapy. Steroids have not proven beneficial in most cases of agent-induced pulmonary edema. Nevertheless, their use in cases of poisoning by oxides of nitrogen or HC smoke should be considered since these agents appear capable of inducing late-onset pulmonary fibrosis by immunological means.

2-2. Protection

The protective mask or a collective protection system gives protection against military lung-damaging agents. High concentrations of certain lung-damaging industrial chemicals (such as ammonia and carbon monoxide) may defeat the mask.

2-3. Properties of Phosgene

Phosgene is the prototypical peripherally acting pulmonary agent and the one with the most extensive battlefield history. At ordinary temperatures and atmospheric pressure, CG is a colorless gas. The boiling point of CG is 47° Fahrenheit (F) (8.3° Celsius [C]), and 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 that some have likened to that of newly mown hay. Phosgene is readily soluble in organic solvents and fatty oils. In water, CG is rapidly hydrolyzed with the formation of hydrochloric acid (HCL) and carbon dioxide.

2-4. Pathology

Aside from mild conjunctival irritation with moderate doses, 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 severe CG poisoning is massive pulmonary edema. The trachea and large bronchi are usually normal in appearance, although with higher doses, damage to bronchiolar epithelium may be seen in association with patchy areas of emphysema. This contrasts with the findings in chlorine and PS poisoning in which not only is pulmonary edema present, but both the trachea and the large bronchi 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 victim survives, resolution commences within 48 hours, and in the absence of complicating infection, there may be little or no residual damage. This contrasts with exposure to oxides of nitrogen and HC smoke, either of which can result in apparent recovery for two to five weeks followed by cough, dyspnea, and radiological and pathological evidence of pulmonary fibrosis (cryptogenic organizing pneumonia).

2-5. Symptoms

During and immediately after exposure, there may either be no symptoms at all or, at moderate to high doses, coughing, choking, a feeling of tightness in the chest, nausea, occasionally vomiting, headache, and lacrimation. The presence or absence of these symptoms is of little value in immediate prognosis since some patients with severe coughing fail to develop serious lung injury, while others with little sign of early respiratory tract irritation develop fatal pulmonary edema. Nevertheless, the appearance of severe coughing should always raise the suspicion of a high inhaled dose of agent. 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. The larger the dose, the sooner the symptoms will appear; onset of dyspnea (shortness of breath) within four hours of exposure is usually a grave prognostic indicator. The clinically asymptomatic phase is replaced by signs and symptoms of pulmonary edema, beginning with dyspnea (the clinical hallmark of incipient pulmonary edema), cough (occasionally substernally painful), rapid shallow breathing, and cyanosis. Nausea and vomiting may appear. As 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.

2-6. 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. Noncardiogenic pulmonary edema is similar to 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. A high index of suspicion and the early generation of a presumptive clinical diagnosis of possible CG exposure may mean the difference between life and death for a victim.

2-7. Prognosis

During the acute phase, prognosis should be guarded because of the progressive nature of the effects. The most important prognostic indicator is the length of the latent, or clinically asymptomatic, period. Victims with dyspnea occurring within the first four hours of exposure may well be expectant. Exertion after exposure will worsen the prognosis. Most deaths occur within the first 48 hours. The few that 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 the development of chronic bronchitis and bronchiectasis. Long-term pulmonary effects are generally the result of intercurrent infection or other exposures.

2-8. 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. The victim should be evaluated by medical staff familiar with the presentation of noncardiogenic pulmonary edema. Victims with no initial difficulty breathing may still become fatalities, and if there is reason to suspect significant CG exposure, affected soldiers should be kept at rest, evaluated, and promptly evacuated if the operational situation permits.

c. If potentially affected service members develop dyspnea (shortness of breath) either on exertion or at rest, they should be evaluated clinically as soon as possible. In the event of a suspected CW agent release, clinical judgment should be made concerning the likelihood of exposure to CG and the inhaled dose (taking into account that higher doses produce shorter latent periods), and those soldiers who are at high likelihood of exposure should be kept at rest, observed, and promptly evacuated even if they are not yet clinically symptomatic.

2-9. 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, if operational situation permits. 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. Evacuation by litter in cases of significant respiratory involvement is strongly advised.

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], continuous positive airway pressure [CPAP] mask, positive end-expiratory pressure [PEEP] mask, or, if necessary, intubation 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 culture-positive 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 for victims of CG poisoning. Steroids have been demonstrated to be useful only for treatment of oxides of nitrogen and HC smoke. 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. Steroid dosage requirements are much greater than those used to treat asthma. Two regimes are used: 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:

(*a*) Treatment should start at the earliest possible moment with the inhalation of the steroid from an inhaler. This must be done in a CW agent vapor-free environment. Treatment may be required for five days or longer.

(*b*) Systemic steroids should be administered according to a tapering-dose regimen. Beginning with day six, the dose of systemic steroids should be reduced as soon as possible, provided that the chest radiograph remains clear. If further early systematic treatment is necessary, epinephrine (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 one puff should be given hourly for the next 24 hours for as long as inhalational therapy is considered necessary (at least five days). Systemic therapy is needed even, for precautionary treatment, during the first 24 hours and should commence as soon as possible with the intravenous (IV) injection of 20 mg of betamethasone or the equivalent dose of another systemic steroid. This dose should be repeated intravenously or intramuscularly every six hours for at least the first 24 hours. During the next five days, inhalation therapy should be continued but systemic therapy may be reduced based on clinical response and improvement on chest radiographs.

(*b*) Pulmonary fibrosis is typical of damage caused only by oxides of nitrogen and HC smoke. Definitive treatment may call for longer periods of systemic therapy. Prednisolone, betamethasone, and 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.

2-10. 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 encouraged to resume physical exertion as soon as possible.

CHAPTER 3

NERVE AGENTS

Section I. INTRODUCTION

3-1. General

a. Nerve agents are a group of highly toxic organophosphorous compounds. They are similar in action to organophosphate insecticides but are more potent, longer-acting, and tend to be irreversible after a time that varies with the agent.

b. Nerve agents are among the deadliest of CW agents and may produce symptoms rapidly. They include the G- and V-agents. Examples of G-agents are tabun (GA), sarin (GB), soman (GD), and cyclosarin (GF). A V-agent is VX. (Detailed descriptions of nerve agents are found in FM 3-9.) Non-traditional agents exist that have similar modes of action.

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. The symptoms and treatment of poisoning by these compounds are similar to those of poisoning by nerve agents.

3-2. Physical and Chemical Properties

Nerve agents are colorless to light brown liquids. Some are volatile, while others are relatively non-volatile at room temperature. Most nerve agents are odorless; a few have a faint fruity odor. Aqueous solutions of nerve agents are tasteless. The G-agents tend to be nonpersistent, whereas the V-agents are persistent. Thickening substances may be added to nonpersistent agents, reducing volatility and allowing these mixtures to remain in the environment for extended periods of time.

3-3. 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, it 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. Skin exposure produces localized sweating and/or muscular twitching. Local effects after vapor or liquid exposure to the eye include miosis and often, conjunctival hyperemia. Effects of liquid on mucous membranes include twitching or contracting of the underlying muscle and glandular secretions. 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 is absorbed through the skin very slowly, so proper masking may provide some protection against the effects of low vapor concentrations.

To prevent inhaling an incapacitating or lethal dose, one should stop breathing immediately and don the mask within nine seconds at the first warning of a nerve agent presence.

c. Liquid nerve agents rapidly penetrate ordinary clothing. Although absorption through the skin usually requires least several minutes (and for low doses this may take up to 18 hours), the process begins almost immediately after contact with the liquid agent. The effects may be reduced by quickly removing contaminated clothing and neutralizing liquid nerve agent on the skin (washed off, adsorbed through blotting, or wiped away). Prompt decontamination of the skin is imperative. Decontamination of nerve agents on the skin within one minute after exposure is ten times more effective than if delayed five minutes. Nerve agent on the skin can be removed effectively by using a skin decontaminating kit (SDK) (such as M291 SDK [Appendix D]). Liquid nerve agent in the eye is absorbed faster than on the skin; contaminated eyes should be immediately irrigated with copious amounts of saline or uncontaminated water.

d. The mission-oriented protective posture ensemble (chemical protective overgarment, impermeable protective gloves, and overboots) and the patient protective wrap (PPW) protect the skin against nerve agents in liquid, aerosol, and vapor forms. The protective capability of the MOPP ensemble is enhanced by use of the Skin Exposure Reduction Paste Against Chemical Warfare Agents (SERPACWA). See Appendix D for discussion on use of SERPACWA.

3-4. Effects of Nerve Agents

a. Mechanism of Action. Nerve agents (Table 3-1) inhibit cholinesterase enzymes throughout the body. Since the normal function of these enzymes is to hydrolyze acetylcholine, such inhibition results in the accumulation of excessive concentrations of acetylcholine at its various sites of action. These include the synapses 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 synapses of sympathetic nerves to the sweat glands (Figure 3-1). Accumulation of acetylcholine at these sites results in characteristic signs and symptoms (Table 3-1) at muscarinic receptors in smooth muscle and glands. 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 3-1). Finally, accumulation of excessive acetylcholine in the brain and spinal cord results in characteristic CNS symptoms (Table 3-1). The total picture of signs and symptoms so produced is called cholinergic crisis. The inhibition of cholinesterase enzymes by nerve agents may be irreversible and the effects prolonged; therefore, treatment should begin promptly. Until the tissue cholinesterase enzymes are restored to normal activity, which may take months, there is a theoretical period of increased susceptibility to the effects of another exposure to any nerve agent and the effects of repeated exposures are cumulative.

Table 3-1. 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, drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, ataxia.

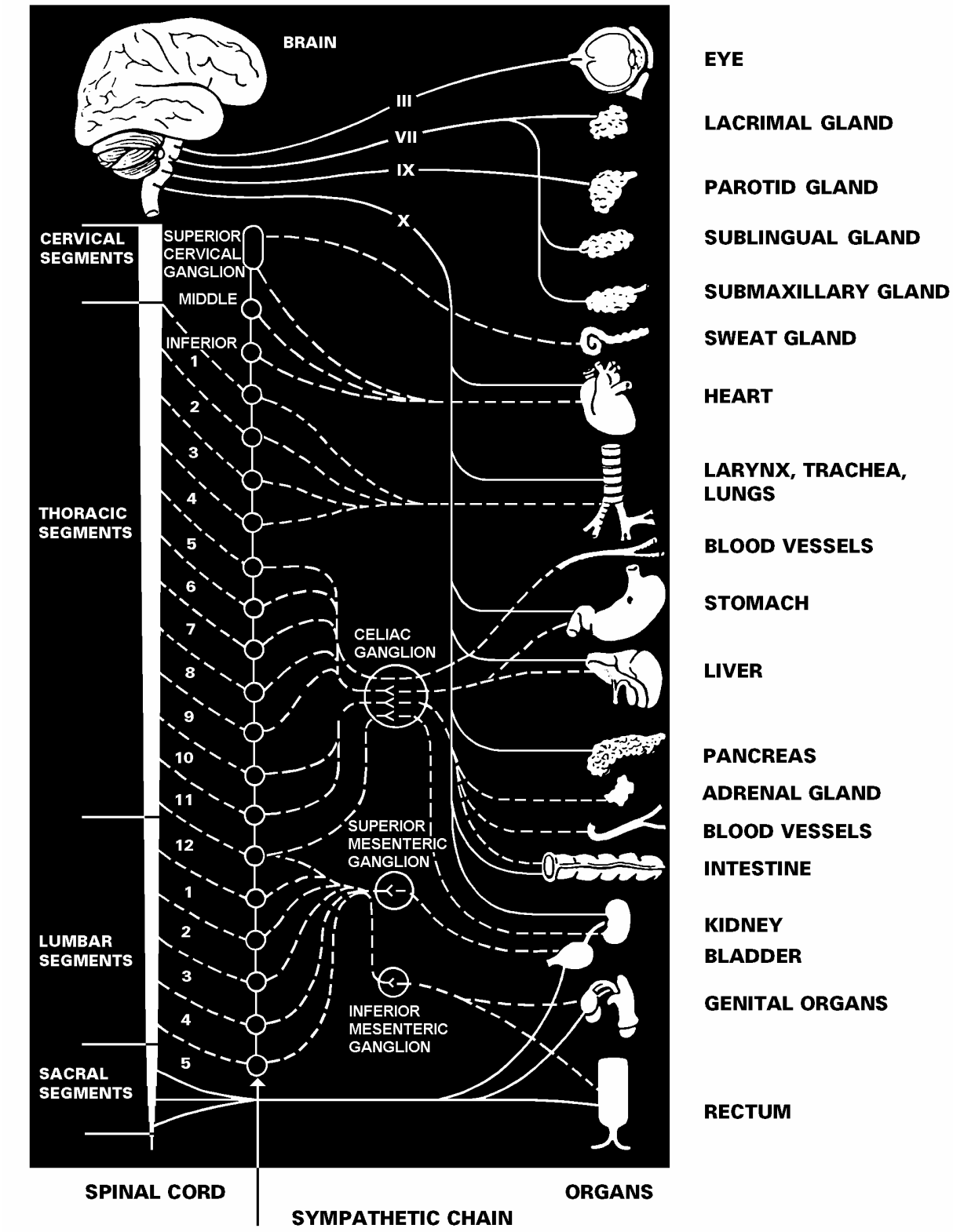


Figure 3-1. Autonomic nervous system.

b. Pathology. Aside from the decrease in the activity of cholinesterase enzymes throughout the body (this decrease 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 airways and the eyes absorb nerve agents rapidly. Results include miosis (contraction of the pupil), bronchial constriction, and excessive secretions in the upper and lower airways. High vapor exposures lead to rapid absorption of agent from the lungs into the general circulation; widespread systemic effects may appear in less than one minute.

(1) *Local ocular effects*. These effects begin within seconds or minutes after exposure and before there is any evidence of systemic absorption. Miosis is an invariable sign of ocular exposure to enough vapor to produce other symptoms. It is also the last ocular manifestation to disappear and may persist for up to weeks to months. The pupillary constriction may be different in each eye. Within a few minutes of exposure, there may be reddening of the eyes due to conjunctival hyperemia; the casualty may also experience a sensation of pressure with heaviness in and behind the eyes. Usually vision is not grossly impaired, although the casualty may complain of dim or dark vision. (This may be from less light entering the eye, but in cases with systemic distribution of agent, may also be secondary to direct effects of nerve agent on the brain.) Exposure to a low level results in miosis; pain in and behind the eyes (attributable to ciliary spasm), especially on focusing; some difficulty of accommodation; and frontal headache. 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. The conjunctival erythema, eye pain, and headache may last from 2 to 15 days depending on the dose; paralysis of accommodation can persist for weeks to months.

(2) *Local respiratory effects*. Earliest effects on the respiratory tract are watery nasal discharge, nasal hyperemia, sensation of tightness in the chest, and occasionally, prolonged wheezing expiration suggestive of bronchoconstriction, or increased bronchial secretion. Rhinorrhea usually lasts for several hours after minimal exposure and for about one day after more severe exposure. Respiratory symptoms may last hours to days.

(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, these effects are more properly considered local effects of nerve agents on exposed respiratory epithelium and musculature. Systemic manifestations are 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 3-2. The systemic effects may be considered to be nicotinic, muscarinic, or due to 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. A sensation of chest tightness is an early local symptom of respiratory exposure. This symptom increases as the nerve agent is absorbed into the systemic circulation, regardless of the route of exposure. After 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 secretions, bronchoconstriction, or both. Some pain may occur in the lower thorax, and salivation increases. Secretions may be thick and tenacious. If postural drainage or suction is not employed, these secretions may add to the airway obstruction. Laryngospasm and collapse of the airway 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, laryngospasm, or collapse of the airway musculature, 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 collapse and lose consciousness. 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. 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 (belching). Abdominal cramps, profuse sweating,vomiting, diarrhea, tenesmus, increased lacrimation, and urinary incontinence may occur. Cardiovascular effects may include early bradycardia, transient tachycardia and/or hypertension followed by hypotension, and cardiac arrhythmias. The casualty may go into cardiorespiratory arrest and die.

Table 3-2. 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.

(b) Nicotinic effects. Increased fatiguability and generalized weakness are followed by scattered muscular fasciculations, involuntary twitching, and occasional 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 may dominate; however, because of the opposing effects of nerve agent at nicotinic receptors in autonomic ganglia and at muscarinic receptors in the heart, the heart rate can be low, normal, or high in a nerve agent casualty and must not be used to gauge the severity of the exposure. Early on, tachycardia is more frequent in casualties than is bradycardia. As the absorbed dose increases, fasciculations (which usually appear first in the eyelids and in the facial and calf muscles) become generalized. 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 may contribute to respiratory depression. Central respiratory depression may be a major cause of respiratory failure.

(c) Central nervous system effects. 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. The casualty may exhibit cofusion and ataxia (difficulty with balance) and have changes in speech, including slurring and difficulty in forming words. The casualty may then become comatose, reflexes may disappear, and Cheyne-Stokes respirations may be seen. Finally, generalized seizures may ensue; in a paralyzed casualty, they may not be observable. With the appearance of severe CNS symptoms, central respiratory depression will occur and may progress to respiratory arrest. 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 [L]). Bloody tears have been reported.

(2) *Local skin effects*. Following cutaneous exposure, there is localized sweating at and near the site of exposure and localized muscular twitching and fasciculation. These may not be noticed; and since nerve agents are colorless and are not irritating to skin, skin absorption may 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, they more properly represent a local effect upon respiratory tissues. Gastrointestinal symptoms are usually the first systemic effects seen after ingestion or after absorption through the skin or through wounds. Following comparable degrees of exposure, respiratory manifestations are most severe after inhalation, and gastrointestinal symptoms may be most severe after ingestion, percutaneous absorption, or entry via wounds. 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 3-2. The latency between exposure and onset and progression of signs and symptoms is dependent on both dose absorbed and route of exposure. The first sign of a massive exposure may be sudden collapse with apnea and convulsions; the difference is that the collapse will be essentially immediate after inhalation of vapor but will be preceded by a clinically asymptomatic, or latent, period following liquid exposure. Most fatal liquid exposures will have a latent period of 30 minutes or less, although mild effects from a tiny drop of VX may take up to 18 hours to appear.

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 persist for up to three months.

g. Mechanism of Death. Death is due to respiratory depression caused by four mechanisms: bronchoconstriction; increased respiratory secretions obstructing airways; paralysis of respiratory muscles, especially the diaphragm; and most importantly, central apnea, or failure of the respiratory center in the brain. When overwhelming doses of the agent are absorbed quickly, death occurs rapidly without an orderly progression of symptoms.

3-5. Clinical Presentation and 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 pinpoint. 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 or only 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 CW agent produces muscular twitching and fasciculations, rapidly developing pinpoint pupils, or the characteristic train of muscarinic, nicotinic, and CNS manifestations. Both cyanide and nerve agents (as well as hydrogen sulfide) can lead to rapid collapse with apnea and convulsions; fine distinctions involving the presence or absence of miosis, secretions, or cyanosis may be difficult to make in this situation. For this reason, when a casualty suddenly collapses, stops breathing, and begins to convulse, nerve agent antidotes should be administered immediately; if the casualty fails to respond, a trial of cyanide antidotes should be considered.

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) (paragraphs 3-9).

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

- Unexplained runny nose.
- Unexplained sudden headache.
- Sudden drooling.
- Difficulty in seeing (dimness of vision and miosis).
- Tightness in the chest or difficulty in breathing.

- Wheezing and coughing.
- Localized sweating and muscular twitching in the area of the contaminated skin.
- Stomach cramps.
- Nausea with or without vomiting.
- 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:

- Confused behavior.
- Increased wheezing and increased dyspnea (difficulty in breathing).
- Severely pinpoint pupils.
- Red eyes with tearing.
- Vomiting.
- Severe muscular twitching and general weakness.
- Involuntary urination and defecation.
- Convulsions.
- Unconsciousness.
- Respiratory failure.
- Bradycardia.

NOTE

Casualties with severe symptoms will not be able to treat themselves and must receive prompt buddy aid (paragraphs 3-9*b*), combat lifesaver aid (paragraphs 3-14*c*), and prompt follow-on medical treatment (paragraphs 3-16) if they are to survive. The first indication of severe exposure may be sudden loss of consciousness with or without apnea and convulsions; that is, there may not be an orderly progression from mild to severe effects.

c. The progress of symptoms from mild to severe indicates either inadequate treatment or continuing exposure to the agent.

Section II. PREVENTION AND TREATMENT OF NERVE AGENT POISONING

3-6. 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 or ATNAA (paragraphs 3-10, and 3-11) as soon as any signs or symptoms are noted.

c. Administering the CANA to severely poisoned casualties or those obviously seizing (paragraph 3-12).

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 an open airway (for example, with a cricothyroidotomy or endotracheal tube) and administering assisted ventilation, if required. Airway resistance from bronchospasm may frustrate attempts at mechanical ventilation of a severely exposed casualty until atropine takes effect.

g. Administering supplemental oxygen as available.

3-7. 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 death. 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 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 milligrams (mg) without nerve agent exposure and will degrade an individual's ability to perform duties in a hot environment because of an inability to sweat. Atropine is rapidly used up in the treatment of nerve agent poisoning, and repeated doses may be necessary.

c. Pyridostigmine bromide (PB), when given as a pretreatment, affords some protective effects against GD. See Section V for a complete discussion on PB.

d. Nerve agents (liquid or vapor) can poison food and water. For details on management and decontamination of food and water, see FM 4-02.7/NTRP 4-02.7/AFTTP(I) 3-2.47/MCRP 4-11.1F.

3-8. Effects of Nerve Agent Antidotes

a. General.

(1) *Atropine*. Atropine sulfate remains the principal drug in the treatment of nerve agent

poisoning. It blocks the effects of acetylcholine at muscarinic receptors and produces relief from these symptoms. If given in large doses, some therapeutic effects are also produced within the CNS, although atropine does not penetrate the blood-brain barrier as readily as does diazepam (paragraph 3-8*a*(3)), and central muscarinic receptors are thought not to be identical with those in the periphery. Atropine is thought to counteract the respiratory depression in the medulla oblongata. More importantly, it probably has a role in preventing the activation of additional neurotransmitters important in the later, more refractory, stages of seizures induced by nerve agents. Used alone, it will not prevent or reverse muscle weakness, paralysis, or apnea and therefore must be supplemented by 2-PAM Cl and by attention to the basics of airway, breathing, and circulation. The combination of adequate atropine plus assisted ventilation is several times more effective in saving lives than assisted ventilation alone and has saved lives even without the second antidote

(2) *2-pralidoxime chloride*. 2-pralidoxime chloride is an oxime that blocks the nerve agent inhibition of cholinesterase by breaking the initial bond between the nerve agent and cholinesterase. Clinically, its effects are more prominent on muscle weakness associated with nerve agent effects at nicotinic sites; thus, its clinical effects are complementary to those of atropine. 2-pralidoxime chloride blocks and reverses the bonding of the nerve agent to the acetylcholinesterase. After a time that is dependent on the specific nerve agent used, a process known as aging strengthens the agent-cholinesterase bond to such an extent that the oximes may no longer be effective. Since the half-times of aging for most nerve agents are hours to days, aging is not clinically relevant for most nerve agents. Almost all of the complex of GD and cholinesterase has aged within 10 minutes of binding. This renders 2-PAM Cl ineffective against GD exposure unless administration occurs relatively early.

NOTE

Other countries field other oximes for this purpose. Their mode of action is identical to that of 2-PAM Cl.

(3) *Diazepam*. Diazepam, the active ingredient in CANA, is the only FDA-approved anticonvulsant that is effective against the seizures caused by nerve agents.

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 duration of action of the antidote may be significantly shortened. More frequent doses of atropine will be required to achieve and maintain the desired clinical effect.

(2) *2-pralidoxime chloride*. Depending on the degree of intoxication, a 600-mg injection will be effective in six to eight minutes and will maintain peak effectiveness for one hour or more. If the system is unchallenged by a nerve agent, this dose will remain in the circulatory system for several hours without apparent adverse 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 rate of distribution in individual patients may vary substantially. The concentrations will then decline over a prolonged period. Early administration via CANA after nerve agent exposure will 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 injections (a total of 30 mg diazepam) to control convulsions; additional IM or IV doses may be given by qualified medical personnel.

c. Symptoms Produced by Antidotes.

(1) *Atropine*.

(a) The administration of a single dose of 2 mg (one autoinjector) of atropine to an individual who has absorbed little or no nerve agent produces minimal to no symptoms. If symptoms occur, they may include dryness of the skin, mouth, throat, and 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 drowsiness, slowness of memory, and diminished 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. 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 increase. After repeated injections of atropine, heat-stressed individuals will become casualties (3-8c(1)(*b)* below). A third 2-mg dose of atropine (again without nerve agent challenge) administered within an hour will incapacitate most people. severe incapacitating symptoms of atropine overdosage in the absence of nerve agent 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 a severe overdose. Overdosage 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 since 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 interfering 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 will sharply reduce combat efficiency, and three doses 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. Near vision may be impaired for as long as 24 hours.

(c) Experience in chemical operations has shown that when troops become alarmed, some believe they have been exposed to more CW 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 and where they are may not need any more atropine injections. If the symptoms do recur, additional atropine, up to two more injections for a total of three, can be administered to these casualties. A service member must consult with a buddy to determine if he needs

additional injections of atropine. If an individual's breathing appears normal, bronchial secretions have diminished, and the skin is dry, the individual does not need any more atropine at that 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 (paragraph 3-5) disappear, or if breathing becomes easier and secretions diminish, no further injections should be administered. These casualties should remain under observation without further injections of atropine unless signs of nerve agent intoxication reappear.

(d) Patients with severe symptoms due to systemic absorption of a nerve agent have increased tolerance for atropine. Multiple doses may be required before airway resistance and secretions diminish. Most cases of nerve agent poisoning should not require a total dose of more than approximately 20 mg of atropine in the first few hours or 50 mg of atropine in a 24-hour period. This contrasts with the often heroic doses (up to 1 to 2 grams [gm]) that may be required in patients poisoned by ingestion of organophosphorous ("organosphosphate") pesticides. The absence of increased tolerance for atropine indicates that nerve agent poisoning probably is not present or is mild. More than three injections of atropine will be administered only by the combat lifesaver or medical personnel.

(2) *2-pralidoxime chloride*. Blurred vision, nausea, vomiting, vertigo, and, most significantly, elevations of heart rate and blood pressure may occur after overdosage with 2-PAM Cl. After the administration of three injections of 2-PAM Cl via MARK I or ATNAA autoinjectors, repeat doses may be given as needed of atropine alone. The additional IM doses of 2-PAM Cl should normally be separated by approximately 60 to 90 minutes.

(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 may have impaired decision-making functions, reduced alertness, and breathing difficulties. For this reason, casualties should be lying on their sides 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 TRAUMA SPECIALIST/CORPSMAN/AIR FORCE MEDIC (4N0 CAREER FIELD) TREATMENT

3-9. Principles of Self-Aid and Buddy Aid

a. Don the protective mask and hood 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. Wear the mask and protective clothing continually until the "all clear" signal is given.

b. Immediately mask any casualty who does not have a mask on if the atmosphere is still contaminated.

c. The appearance of severe (see paragraph 3-5*b*(2)) nerve agent poisoning symptoms calls for the immediate IM injection of the nerve agent antidote and CANA (paragraphs 3-10, 3-11, 2-12, 3-13, and 3-14).

d. Promptly remove any liquid nerve agent on the skin or on the clothing. Remove agent in wounds and eyes by irrigation.

(1) If a liquid nerve agent gets on the skin, decontamination should ideally be accomplished within one minute (see Appendix 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 saline or 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 the first set of atropine and 2-PAM Cl 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 given two additional doses of atropine, two additional doses of 2-PAM Cl, and one injection of CANA by a buddy, in accordance with the provisions of paragraph 3-12. If symptoms still persist, bronchial secretions persist, or the skin remains moist, then the service member can be administered additional atropine injections by medical personnel (who carry additional atropine for the treatment of nerve agent patients) to counteract the nerve agent. Trauma specialists/corpsmen/Air Force medics (4N0 career field) (paragraph 3-13) also carry extra CANA for administration to nerve agent patients. Trauma specialists/corpsmen/Air Force medics (4N0 career field) can administer additional CANA up to a maximum of three before evacuating the patient. Evacuate the service member to a 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. Medical personnel may treat these symptoms with atropine ointment.

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 ATNAA (or three sets of the MARK I) and one CANA, the airway must be secured and assisted ventilation must be started by medical personnel, if a resuscitation device is available. Assisted ventilation should be continued until normal breathing is restored.

3-10. The Nerve Agent Antidote Kit, MARK I

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

a. Description. The MARK I kit consists of four separate components: the atropine autoinjector in a short tube, the 2-PAM C1 autoinjector in a longer tube, 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 that 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 MARK I 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 MARK I 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 US Army, each person is authorized to carry three MARK I kits for the treatment of nerve agent poisoning. The US Navy, the US Air Force, and US Marine Corps, however, do not use the MARK I; rather, its antidote components are issued as three separate atropine and three separate 2-PAM Cl autoinjectors per person.

c. Protection Against Freezing. The atropine and the 2-PAM Cl solutions freeze at about 30° F (-1.1° C). Therefore, when the temperature is below freezing, the 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 injectors become frozen, they can be thawed and used. Allowing the autoinjector to freeze can prevent an individual from having the nerve agent antidote immediately available for use.)

3-11. Antidote Treatment, Nerve Agent, Autoinjector

The ATNAA (Figure E-1) is another nerve agent antidote device that will be used by the Armed Forces in the treatment of nerve agent poisoning.

a. Description. The ATNAA is a multi-chambered device that consists of three components. The autoinjector tube, a spring-activated needle, and a safety cap. The device is packaged in a chemically hardened pouch.

(1) The autoinjector outer cylinder is natural polypropylene consisting of two chambers (one chamber contains 2.1 mg of atropine injection; the second chamber contains 600 mg of 2-PAM Cl injection). It has a pressure-activated coiled spring mechanism which triggers the needle for injection of the antidote solutions. The third component is a safety cap.

(2) The label is white with black lettering; there are two colored stripes on the end of the label (one is tan and the other is yellow). The safety cap is gray plastic. The needle end is green plastic.

(3) The chemically protected pouch is amber and black in color. The end of the pouch that covers the atropine (needle end of the autoinjector) is solid black, the remainder of the pouch is amber. The lettering on the pouch is black.

b. Issue to Service Members. Each service member will be issued and will carry three ATNAA for the treatment of nerve agent poisoning. These devices are for use as the initial treatment of nerve agent poisoning (self-aid or buddy aid).

NOTE

For self-aid or buddy aid, the ATNAA will replace the MARK I and the separately packaged autoinjectors when all stocks of the MARK I and the separately packaged autoinjectors have been exhausted or the device's shelf life expires. Separately packaged atropine autoinjectors will still be available for medical personnel.

c. Protection from Freezing. The atropine and the 2-PAM Cl solutions freeze at about 30° F (-1.1° C). Therefore, when the temperature is below freezing, the ATNAA should be protected from freezing. Normally, the ATNAA issued to service members is 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 ATNAA become frozen, it can be thawed multiple times, if necessary, and used.) Allowing the device to freeze will delay your ability to administer the antidote when needed, which could lead to increased injury from exposure to a nerve agent.

3-12. Convulsant Antidote for Nerve Agent, Autoinjector

The CANA is another nerve agent antidote device that will be used by the Armed Forces in the treatment of nerve agent poisoning.

a. Description. The CANA autoinjector consists of a light gray plastic tube with two flanges and is labeled with directions; the lettering is black. The CANA is packaged in an easy-to-open clear plastic package with a single injector inside. The safety cap is gray plastic on the end of the autoinjector. The needle end is the black plastic end which, when pressure is applied, activates the spring mechanism.

(1) The autoinjector contains 10 mg of diazepam injection. It has a pressure-activated coiled spring mechanism which triggers the needle for injection of the antidote solution. The third component is a safety cap.

(2) The label has black lettering. The safety cap is gray plastic. The needle end is black plastic.

(3) The chemically protected pouch is clear plastic. The pouch has easy-to-tear notches on all sides. The lettering on the pouch is black.

b. Issue to Service Members. Each service member will be issued and will carry one CANA for use in the treatment of nerve agent poisoning. These devices are for use in buddy aid.

NOTE

The CANA is not for use as self-aid. If you know who you are and where you are, you most likely do not need CANA. Seek buddy aid if you feel that you need CANA.

The use of the CANA by or upon persons to whom it has not been prescribed (such as contractors, displaced persons, civilian casualties of terror or combat actions) is enabled by a DOD policy that empowers Health Care Providers and other first responders and service members to use these medications in an emergency outside of an MTF, as an element of pre-hospital or on-site emergency medical actions.

c. Protection from Freezing. The diazepam solutions freeze at about 30° F (-1.1° C). Therefore, when the temperature is below freezing, the CANA should be protected from freezing. Normally, the CANA issued to service members is 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 CANA become frozen, it can be thawed multiple times, if necessary, and used.) Allowing the device to freeze will delay your ability to administer the antidote when needed, which could lead to increased injury from exposure to a nerve agent.

3-13. Principles in the Use of the MARK I and Antidote Treatment Nerve Agent Autoinjector

The following are principles to be followed in the administration of the nerve agent antidotes.

a. Self-Aid. If you experience most or all of the mild symptoms of nerve agent poisoning (paragraph 3-5*b*(1)), you should immediately hold your breath (do not inhale) and put on your protective mask. Then administer one ATNAA or one set of MARK I injections into your lateral thigh muscle (or buttocks). (Self-aid procedure for administering the autoinjectors is found in Appendix 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 may 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 ATNAA or 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 (paragraph 3-5*b*(2)), render the following aid:

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

(2) Administer, in rapid succession, three ATNAAs or sets of the MARK I. Follow administration procedures outlined in Appendix E.

NOTE

Use the casualty's own antidote autoinjectors when providing first 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 combat lifesaver must check to verify if the individual has received three ATNAAs or sets of the MARK I. If not, the combat lifesaver performs first aid as described for buddy aid above. If the individual has received the initial three ATNAA or sets of MARK I, then the combat lifesaver may administer additional atropine injections at approximately 10-minute intervals until breathing becomes easier and secretions are reduced. Administer additional atropine at intervals as needed (to reduce airway resistance and secretions and to maintain the heart rate above 90) or until the casualty is placed under the care of medical personnel. Request medical assistance as soon as the tactical situation permits.

d. Trauma Specialist/Corpsman/Air Force Medic (4N0 Career Field). If a patient has received three ATNAAs or sets of MARK I but is not yet medically stable, then administer additional atropine at approximately 10-minute intervals until breathing becomes easier and secretions are reduced. Administer additional atropine at intervals as needed (to reduce airway resistance and secretions and to maintain the heart rate above 90) or until the patient is evacuated to an MTF. Provide assisted ventilation for severely poisoned patients, if equipment is available. Monitor the patient for development of heat stress.

3-14. Principles in the Use of Convulsant Antidote for Nerve Agents

The following are principles to be followed in the administration of **CANA** (Figure 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 or ATNAA, seek assistance from a buddy.

b. Buddy Aid. When giving all three doses of MARK I or ATNAA antidotes at once as buddy aid, and/or if the casualty is also convulsing, administer the CANA.

(1) Mask the casualty, if necessary.

(2) Administer the CANA with the third MARK I or ATNAA 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 Trauma Specialist/Corpsman/Air Force Medic (4N0 Career Field). The combat lifesaver or trauma specialist/corpsman/Air Force medic (4N0 career field) should administer additional CANA to patients 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 combat lifesaver or trauma specialist/corpsman/Air Force medic [4N0 career field]).

Section IV. TREATMENT IN MEDICAL TREATMENT FACILITY

3-15. Administration of the Nerve Agent Antidotes

Upon arrival at the MTF, a patient may still have signs/symptoms of nerve agent poisoning (paragraph 3-5). The patient may have received self-aid, buddy aid, combat lifesaver care, or treatment by the trauma specialist/corpsman/Air Force medic (4N0 career field), or other medical personnel in the field before and during evacuation. Additional injections of the nerve agent antidote(s) must be administered at the MTF.

a. Atropine. Decreased airway resistance and secretions 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 airway resistance decreases (that is, the patient can breathe easily or can be ventilated adequately) and until secretions are reduced.

(2) Severe symptoms should be treated by administering 2 mg of atropine IM or IV as available as frequently as required until airway resistance decreases (that is, the patient can breathe easily or can be ventilated adequately) and until secretions are reduced. Doses of 2 mg of atropine (without 2-PAM Cl) can be injected every 10 to 30 minutes as long as needed.

b. 2-pralidoxime chloride. Specifically as an adjunct to atropine, 2-PAM Cl is used to break the bond between the nerve agent and cholinesterase if aging has not yet occurred (paragraph 3-8). Clinically, 2-PAM Cl reduces muscle twitching, weakness, and paralysis (nicotinic effects) and is thus complementary to the muscarinic effects of atropine. 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) Severe symptoms should have been treated by administering three 600-mg IM or IV injections of 2-PAM Cl. Repeat the dose at least every hour if respiration has not improved.

c. Diazepam. Diazepam (CANA) is used specifically as a treatment for convulsions in nerve agent poisoned casualties. If brain damage is to be prevented in to severe nerve agent poisoned casualties, CANA must be administered early. Convulsions (seizures) should be anticipated in all severe cases and treated with the CANA, repeated as necessary. Whenever a patient is affected enough to require the administration of three MARK I kits or three ATNAA autoinjectors at the same time, CANA must be concurrently administered.

3-16. 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. Patients must be decontaminated before entering MTFs. Modifications of these procedures may be used in a contaminated environment although an increase in exposure will occur. If this is not done, the patient may die.

a. Administration of Additional Atropine. For patients who are in severe respiratory distress or are convulsing, all three ATNAA or three sets of their MARK I autoinjectors should have been given. (Convulsions are treated with diazepam, as described in 3-16*d* below.) If relief does not occur and if airway resistance remains high (tightness in the chest in a conscious patient or difficulty in ventilating an apneic patient), if bronchial secretions and salivation do not decrease, or if the heart rate is less than 90 beats per minute, administer additional atropine IM or IV 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 the muscarinic effects of the nerve agent for as long as necessary. Patients who are sufficiently recovered to be able to treat themselves but who are not yet stable for discharge/evacuation may self-administer MANAA under medical supervision.

b. Management of Increased Airway Resistance. In an unconscious and apneic patient, airway resistance may be so high that attempts at artificial ventilation (manually or with a mechanical ventilator) may be unsuccessful. This underscores the need for immediate atropine administration in an unconscious and apneic patient even before intubation and ventilation are attempted. Atropine must be repeated as long as increased airway resistance impedes effective ventilation.

c. 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. An oropharyngeal airway may then 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, administer additional atropine and insert an endotracheal tube.

d. Management of Convulsions. Convulsions are a prominent feature of nerve agent poisoning. Patients who develop convulsions usually progress rapidly to unconsciousness and generalized muscular weakness or flaccid paralysis, at which point external evidences of convulsions cease. Administer CANA or IV diazepam until convulsions are controlled.

e. Treatment of Ocular Symptoms. Ocular symptoms produced by local absorption of a nerve agent do not respond to the systemic administration of atropine. 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 one to three 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.

f. 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. It is important to anticipate increased airway resistance and to administer atropine, preferably before cricothyroidotomy or intubation, to minimize this problem. Cricothyroidotomy should not be deferred if required merely because atropine is not available. When a casualty reaches an MTF where oxygen and a positive pressure ventilator are available, these should be employed continuously until adequate spontaneous respiration is resumed. Endotracheal intubation will most likely be required.

NOTE

Treatment outlined in paragraphs 3-15 and 3-16 is based on the US Army doctrine on the use of the ATNAA or MARK I and CANA.

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 BROMIDE PRETREATMENT FOR SOMAN NERVE AGENT POISONING

3-17. Purpose

a. This section prescribes the use of soman nerve agent pyridostigmine bromide pretreatment (SNAPP) as an adjunct to the MARK I or ATNAA for GD nerve agent poisoning. When PB is used in conjunction with the atropine and 2-PAM Cl (paragraphs 3-10 and 3-11, and Appendix E), the survivability of GD nerve agent-poisoned casualties may be enhanced. Also covered in this section are the individual, unit, and command responsibilities for the pretreatment regimen.

b. The FDA has approved 30-mg SNAPP tablets as a pretreatment against GD nerve agent poisoning. Therefore, SNAPP is no longer considered investigational when used as a GD nerve agent pretreatment.

c. Approval was based on animal studies of how well SNAPP works (efficacy). It is not ethical to test SNAPP in humans because studies would have to expose people to the deadly effects of nerve agent, risking poisoning or killing them. The results of the animal studies establish that PB is reasonably likely to produce clinical benefit in humans.

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

e. Minimal detrimental effects are expected at the recommended dosages. Adverse effects and contraindications are described in paragraph 3-22 below.

3-18. The Soman Nerve Agent Pyridostigmine Bromide Pretreatment Tablet Set

a. The SNAPP tablet blister pack (Figure 3-2 and Figure 3-3) contains the pretreatment medication to be taken within 8 hours prior to exposure to GD nerve agent at which time the atropine is used. The blister pack contains 21 tablets. Each tablet consists of 30 mg PB. Each blister pack contains enough tablets for seven days (one taken every 8 hours).

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

c. Orders to start taking SNAPP will be issued by the proper line authority within the chain of command. It is not a medical decision (paragraph 3-24).

Front of a Cardboard Sleeve Containing 1 Blister Pack of 21 Tablets

21 TABLETS
PYRIDOSTIGMINE BROMIDE USP 30 mg
(Soman Nerve Agent Pre-Treatment Tablets)
NSN 6505-01-178-7903

Rx only

Directions for use:
1. START TAKING ONLY WHEN ORDERED BY YOUR COMMANDER
2. TAKE ONE (1) EVERY EIGHT (8) HOURS
3. IT IS DANGEROUS TO EXCEED THE STATED DOSE

ICN Canada Limited, Montreal, Quebec H4M 1V1

Lot No.: XXXX
Expiration Date: XXXX
DISCARD CONTENTS 3 MONTHS AFTER ISSUE

Back of a Cardboard Sleeve Containing 1 Blister Pack of 21 Tablets

Before using, **READ** enclosed **INFORMATION.**

PB is indicated for pre-treatment against Soman nerve agent.

PB is taken before potential exposure to Soman. If you are exposed to nerve agent and have symptoms, you must use your nerve agent antidotes (atropine and pralidoxime provided in the MARK I Nerve Agent Antidote Kit or the ATNAA). **Do NOT take PB after exposure to nerve agents.**

Warning: If you have asthma, are pregnant, are allergic to bromide, or are taking medicine for high blood pressure or glaucoma, see your unit doctor before taking PB.

Pyridostigmine may cause stomach cramps, diarrhea, nausea, frequent urination or headaches, dizziness, shortness of breath, worsening of peptic ulcer disease, and lacrimation (eye tearing). Seek medical attention if these or other symptoms persist or worsen.

Figure 3-2. Pyridostigmine bromide tablet cardboard sleeve labels.

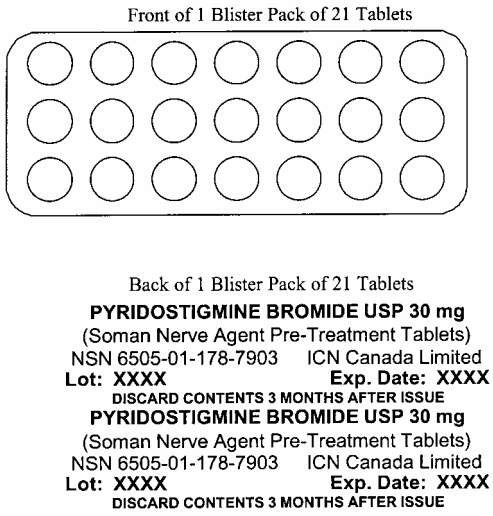


Figure 3-3. Pyridostigmine bromide blister pack front and back label.

3-19. Effects of Pyridostigmine Bromide

a. Pyridostigmine bromide protects the acetylcholinesterase enzyme in the body from the action of the nerve agent GD. Nerve agents irreversibly block acetylcholinesterase, resulting in an excessive accumulation of acetylcholine at the neuromuscular junction, which results in nerve agent poisoning and its accompanying symptoms. When enough PB is given to bind temporarily with a certain percentage of the acetylcholinesterase in the body before nerve agent exposure, the bound enzyme is thus converted into a "reserve force" that is protected against the initial onslaught of nerve agent but that can then be freed up (as the PB eventually leaves the enzyme naturally) to help counteract the excess acetylcholine.

b. Pyridostigmine bromide is not a "true" pretreatment. A true pretreatment would, by itself, provide some protection directed specifically against a nerve agent. Though not providing protection by itself, PB significantly enhances the efficacy of the ATNAA or MARK I within one to three hours after taking the first tablet. Maximal benefit develops with time and is reached when a tablet is taken every eight hours.

3-20. Principles in the Use of Pyridostigmine Bromide

a. To be maximally effective, one SNAPP tablet should be taken every eight hours on a continuous basis prior to exposure to a GD 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 SNAPP 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 eight hours, the beneficial effect of SNAPP as a pretreatment significantly diminishes after eight hours from the last tablet.

b. Antidotes are still required in individuals who have received SNAPP prior to exposure.

CAUTION

Do not attempt to give a SNAPP tablet to a casualty with nerve agent symptoms. SNAPP must not be taken after exposure to GD. If SNAPP is taken immediately before exposure or at the same time as poisoning by GD, it is not expected to be effective and may make the effects of a sub-lethal exposure to GD worse.

c. At times, a commander may defer administration of SNAPP 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 (paragraph *d* below). In any case, the benefits versus the risks should be carefully weighed before a decision is reached.

d. As long as the risk is elevated, 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 could be hazardous. (Examples: Troops are operating at night without lights or are in a CW 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. Pyridostigmine bromide should be used during pregnancy only if clearly needed.

3-21. Administration of Pyridostigmine Bromide 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 eight hours as directed by your commander. If a dose is missed, do not make it up. Do not take two tablets at once because of a missed dose—merely start again with one tablet every eight hours. Taking two tablets at once could result in adverse side effects. Taking more than one tablet at a time does not provide additional protection—and increases the risk of side effects. To make it easier to track the number of pills taken during the course of a day, the first three pills should be taken from a row of three against one of the ends of the packet. Additional pills should then be taken as a total of one three-pill row per day.

3-22. Signs and Symptoms of Pyridostigmine Bromide Overdose, Adverse Reactions, and Contraindications

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. The most commonly expected side effects will be diarrhea and increased urinary frequency. In most patients, these improve after the first day or two on SNAPP.

c. Contraindications.

(1) Since PB may increase bronchial secretions and aggravate bronchiolar constriction, caution should be used in its administration to personnel with bronchial asthma.

(2) Pyridostigmine bromide is contraindicated in mechanical intestinal or urinary obstructions.

(3) Pyridostigmine bromide should not be administered to personnel with known hypersensitivity to anticholinesterase agents.

(4) Additional relative contraindications include hyperthyroidism, sensitivity to bromide, peptic ulcer disease, and low serum acetylcholinesterase.

(5) Personnel who are self-administering PB while handling or working around insecticides containing organophosphorus compounds should use additional precautions, including the use of personal protective equipment, since any effects of exposure to these compounds will be exacerbated by PB.

WARNINGS

1. Pyridostigmine bromide may increase bronchial secretions and aggravate bronchiolar constriction; thus, caution should be used in its administration to individuals with bronchial asthma.

2. Pyridostigmine bromide should also be used with caution in individuals with hyperthyroidism, sensitivity to bromide, peptic ulcer disease, and low serum acetylcholinesterase

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

3-23. Emergency Medical Treatment for Pyridostigmine Bromide Adverse Side Effects, Allergic Reactions, and Overdose

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

a. Emergency treatment for an overdose of PB requires the administration of atropine in adequate doses to overcome the cholinergic crisis. Initially, the 2 mg of atropine found in the MARK I kit or ATNAA should be used. In most cases, this will be sufficient. Further administration of atropine may be necessary to control the cholinergic effects of PB. 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 eight hours as directed.

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

3-24. Responsibilities

a. The corps/division/wing/fleet commander will—

(1) Decide whether to begin, continue, or discontinue the administration of SNAPP based on the threat. The intelligence officer, chemical officer, and the surgeon serve as advisors to the commander to assist him in determining if a chemical nerve agent threat exists (for example, the presence of nerve agents in the combat zone or a high probability of their use). Since SNAPP is a prescription drug, the command surgeon or another physician should be personally involved in the decision to issue and use SNAPP. After three days of self-administration of SNAPP by the service member, combat conditions should be reevaluated by the commander and his staff to determinate whether to continue the medication or not. Orders to discontinue the pretreatment can 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 seven days (21 tablets) and is

issued the second pack on the seventh day. Administration of the medication beyond 14 days is not recommended without a thorough evaluation of the situation and recommendation of the medical authority. The magnitude of the threat may outweigh any possible adverse side effects and indicate continuance of the pretreatment.

(2) Train the service members to take SNAPP as directed to enhance their survivability if they are exposed to GD. Service members must be trained to take SNAPP 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 SNAPP blister pack during personnel decontamination and overgarment exchange.

b. Units will—

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

(2) Maintain at least a two-week supply of SNAPP per member of the unit. One SNAPP blister pack is issued to each member of the unit. An additional week's supply of SNAPP 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 SNAPP for individual issue and request replacements as the items are issued, or as they exceed their labeled shelf life. Pyridostigmine bromide tablets should be stored (refrigerated) in temperatures ranging from 35° to 46° F (2° to 8° C). If the medication is removed from refrigeration for more than three months, do not issue to the individual service member. Once issued to the individual service member, SNAPP must be replaced every three months.

(4) Issue SNAPP 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 PB overdose, adverse reactions, and side effects (paragraph 3-22 above) for determining, on an individual basis, whether or not a service member is to continue SNAPP 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 SNAPP as directed and in accordance with the provisions of paragraph 3-20 above.

(2) Cease taking SNAPP if exposed to nerve agent until directed to resume self-administration by higher authority.

(3) Secure SNAPP supplies against loss and freezing

CHAPTER 4

CYANIDE COMPOUNDS ("BLOOD AGENTS")

4-1. General

Cyanide compounds are taken up by the blood or lymphatics and systemically distributed to all tissues and organs of the body. Hence, they were historically called "blood agents." The subsequently introduced blister agents, nerve agents, and incapacitating agents are also absorbed into the bloodstream and systemically distributed and are in that sense as much "blood agents" as are the cyanides. The term "blood agents" may promote the incorrect idea that the main action of the cyanides is in the blood. In fact, these agents produce their effects by interfering with oxygen utilization at the cellular level. The term "blood agents" is still in use, but it should be considered an obsolete term to be replaced by "cyanide compounds." Hydrogen cyanide (AC) and cyanogen chloride (CK) are the important agents in this group. Cyanogen chloride also produces central and peripheral pulmonary effects on the respiratory tract because of its chlorine component (paragraph 7-1). These agents can be dispersed by artillery shell, mortar shell, rocket, aircraft spray, and bomb. All cyanide compounds are nonpersistent.

4-2. Protection

The protective mask with a fresh filter gives protection against field concentrations of cyanide. For protection, MOPP 4 is needed when exposed to or handling liquid AC.

4-3. 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 (21.1° C) and freezes at 7° F (-13.9° C). It is highly soluble and stable in water. It has a faint odor, somewhat like peach kernels or bitter almonds, but the ability to detect this odor is conferred by a single gene present in only 40 to 60 percent of the population. Moreover, olfactory accommodation to the odor of cyanide compounds is rapid. Because AC is highly volatile, AC vapor and gas dissipate quickly in the air. It is the only CW agent lighter than 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 (15.0° C) and freezes at 20° F (-6.7° C). Although only slightly soluble in water, CK dissolves readily in organic solvents. The vapor of CK is heavier than air and is very irritating to the eyes and mucous membranes. The pungent, biting odor of CK may be masked by its irritating and lacrimatory properties. Although nonpersistent, CK vapor may remain in the jungle and forest for up to hours under suitable weather conditions.

4-4. Pathology

a. Hydrogen cyanide is thought to act by combining with cytochrome oxidase, an enzyme located within mitochondria in cells and essential in the electron-transport system of oxidative phosphorylation, or cellular respiration. Blockage of this enzyme results in failure of the cell to use presented oxygen from the blood and produce energy and package it as adenosine triphosphate (ATP). Hydrogen cyanide poisoning causes cells to switch to anaerobic metabolism, with a buildup of lactic acid resulting in lactic acidosis. This can be measured by medical laboratories. The CNS (particularly the respiratory center) is especially susceptible to this effect, and central apnea is the usual mechanism of death. Hydrogen cyanide in high concentrations may cause death within a few minutes without

anatomical changes. After longer exposure to lower concentrations, there may be small areas of hemorrhage and softening in the brain that are more pronounced in delayed deaths. Because the ability of cells to extract oxygen from blood is impaired in cyanide victims, venous blood may be as red as arterial blood; and cyanosis is not classically associated with cyanide poisoning. In fact, the skin may have a pink color similar to that seen in carbon monoxide (CO) poisoning. The cherry-red coloration seen in CO poisoning results from the intrinsic color of carboxyhemoglobin (COHb), whereas the pink tinge to the skin in cyanide poisoning reflects the high oxygen content of capillary and venous blood.

b. Cyanogen chloride acts in two ways. Its systemic effects are similar to those of AC, but because of its chlorine component, it also has local irritant effects on the eyes and in the upper (central) respiratory tract and in the peripheral compartment of the respiratory tract (pulmonary edema). 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.

4-5. Symptoms

a. The symptoms of AC depend upon the agent concentration and the duration of exposure. Exposure to high concentrations of cyanide gas can produce fatalities within minutes, whereas exposure to lower concentrations may produce symptoms gradually. At high exposures, death usually occurs rapidly or there is prompt clinical recovery after removal of the victim from the toxic environment. In animals, relapse and death have occurred hours after apparent recovery; observation for 24 hours is therefore recommended for cyanide casualties. High concentrations induce increased rate and depth of breathing (gasping) within seconds. This gasping reflex may be so powerful that casualties cannot voluntarily hold their breath. Unconsciousness and violent convulsions may occur after as little as 20 to 30 seconds, with cessation of respiration within one minute. Cardiac failure follows shortly thereafter. Following moderate exposure, weakness of the legs, vertigo, nausea, and headache appear very early. These may be followed by convulsions and coma that 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 that may be manifested by irrationality, altered reflexes, and unsteady gait that may last for several weeks or longer. Temporary or permanent nerve deafness has 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 those produced by chlorine, which is a combination central/peripheral pulmonary agent. Initially, CK, like AC, stimulates the respiratory center and then rapidly paralyzes it. In high concentrations, however, its local irritant action may produce 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, heralded by dyspnea and eventually with persistent cough, production of frothy sputum, and marked cyanosis.

4-6. 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. Sudden collapse with loss of consciousness, apnea, and convulsions is consistent both with nerve agent exposure and cyanide poisoning.

b. In casualties exposed to CK, the diagnosis is further suggested by the rapid onset of cyanide effects together with the intense irritation characteristic of exposure to chlorine.

c. In theory, miosis, twitching, hypersalivation, and cyanosis should be more prominent in nerve agent casualties; in practice, it may be difficult to distinguish between nerve agent exposure and cyanide exposure in this situation. Casualties that present with these signs and that are unresponsive to nerve agent antidotes should be considered for a trial of cyanide antidotes.

4-7. Prognosis

a. Hydrogen Cyanide. Death may occur rapidly. Occasionally, when there is prolonged tissue anoxia, residual injury of the CNS may persist for weeks; some of this damage may be permanent. Many casualties recover within hours without sequelae.

b. Cyanogen Chloride. Prognosis is similar to that for AC. Recovery from the systemic effects is usually as prompt as in AC poisoning. A higher incidence of residual damage to the CNS should be expected. Depending on the concentration of CK to which the casualty has been exposed, the pulmonary effects may develop immediately (suggestive of central pulmonary damage) or may be delayed (consistent with peripheral pulmonary damage) until the systemic effects have subsided. Thus, prognosis must be guarded.

4-8. Self-Aid

a. Hydrogen Cyanide. If you get a sudden stimulation to breathe or detect a bitter almond odor during a chemical attack, put on your mask immediately. Speed in masking is absolutely essential since the effects of this agent are so rapid that within a few seconds you will not be able to put on your mask. Stop breathing until the mask is on, if 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.

4-9. Buddy Aid

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

4-10. Treatment

a. In AC or CK poisoning, if the patient's respirations are feeble or have ceased, immediately begin assisted ventilation, provide oxygen if available, start an IV, administer amyl nitrite if available, and begin IV administration of sodium nitrite and sodium thiosulfate (paragraph 4-10*b* and 4-10*c* below). Before the treatment is rendered, either remove the patient from the contaminated environment or mask the patient. Continue assisted ventilation until spontaneous breathing returns or until 10 minutes after the last evidence of heart activity has occurred.

b. If amyl nitrite is available and the environment is uncontaminated, hold one ampule, or capsule (0.2, 0.3, or 0.35 mL, depending upon the formulation), close to a breathing patient's nose, crush the amputee, and allow the patient two to six breaths (15 seconds) from the ampule. In a contaminated environment, it is not advised to break the seal of the patient's mask in order to introduce a crushed ampule. For an apneic patient, crush one ampule in an Ambu bag and ventilate the patient. The dose may be repeated in three to five minutes.

c. Intravenously inject one vial (10 ml of a 3 percent solution, or 300 mg) of sodium nitrite over a period of three minutes. Immediately after completion of the sodium nitrite injection, intravenously inject one bottle (50 ml of a 25 percent solution, or 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 any remaining free cyanide to form thiocyanate that is excreted from the body.

d. Caution should be exercised when giving methemoglobin formers such as sodium nitrite when there are other reasons for low oxygen saturations (such as if the casualty has been in a fire) even if cyanide intoxication is suspected because neither methemoglobin nor carboxyhemoglobin carries oxygen.

CAUTION

Administer sodium nitrite and sodium thiosulfate ONLY intravenously. Intramuscular administration will cause severe tissue necrosis.

e. The decrease in blood pressure following sodium nitrite injections is usually not clinically significant 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.

f. The lung irritant effects of CK are treated according to the presence of pulmonary effects, as in chlorine poisoning.

CHAPTER 5

BLISTER AGENTS (VESICANTS)

Section I. INTRODUCTION

5-1. 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 and "dusty" blister agents will contaminate terrain, ships, aircraft, vehicles, or equipment and present a persistent hazard. Dusty mustard refers to a form of sulfur mustard (HD) developed as a dry powder. Vesicants include HD, nitrogen mustards (HN), Lewisite (L) (this may be used in mixtures with HD), and halogenated oximes (example, phosgene oxime [CX]). The properties and effects of halogenated oximes are different from those of the other vesicants and, strictly speaking, CX is a corrosive and an urticant (producing wheals or hives) rather than a vesicant. It is usually grouped with the true 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 have delayed effects on blood-forming organs. Lewisite causes pain within minutes of exposure and CX causes immediate pain on contact, but 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. Vesicants damage the respiratory tract when inhaled and cause vomiting and diarrhea when ingested.

c. Some vesicants have a faint odor; others are odorless.

d. Vesicants can 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. The severity of systemic effects from mustard is not well correlated with the percentage of body surface area burned. This may be due to factors such as agent concentration on the skin and concomitant inhalational exposure.

5-2. Self-Aid

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

b. Immediately decontaminate the eyes or the skin if exposed to liquid or vapor agents. Follow decontamination procedures as outlined in Appendix D.

5-3. 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 must be decontaminated outside the MTF to prevent vapor accumulation indoors. Contaminated casualties should be separated from clean (uncontaminated) casualties until decontamination is completed. Contaminated litters, blankets, and equipment should be kept outdoors. All equipment, vehicles, watercraft, and aircraft that have been used to transport contaminated casualties should be limited; once contaminated, the same evacuation assets should be repeatedly used in the contaminated area until all casualties have been evacuated. All evacuation assets used must be decontaminated before return to full service. See Appendix B for further information on decontamination.

c. Mustard present on casualties' skin surface can present a hazard to individuals receiving or treating these casualties even after several hours, but mustard that has been absorbed into the skin will not be a surface contact hazard.

5-4. 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.

c. Skin Exposure Reduction Paste Against Chemical Warfare Agents (SERPACWA) is a topical skin protectant that provides added protection for selected skin areas. SERPACWA is to be used with the overgarment, not as a replacement for it. Use SERPACWA at potential points of exposure such as the wrists, ankles, armpits, groin, and waistline. See Appendix D.

Section II. SULFUR MUSTARD

5-5. Sulfur Mustard

a. Physical Properties. Sulfur mustard (commonly referred to simply as mustard) occurs principally as a solid (below 58° F [14.4° C]), as an oily liquid ranging from colorless when pure (neat) to dark brown when impure, and as HD vapor released from liquid. Mustard "gas" exists only above 423° F (217.2° C). Mustard is heavier than water, but small droplets may float on water surfaces and present a special hazard in contaminated areas. Mustard is not related to the mustard plant but gets its name from its odor, which resembles that of mustard, garlic, onions, or horseradish. Distilled HD, the most common form of HD, is only slightly soluble in water, which gradually destroys it, but undissolved HD 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 HD. Mustard disappears from contaminated ground or materials through evaporation or hydrolysis.

b. Persistence. The persistence of a hazard from HD vapor or liquid depends on the degree of contamination by the liquid, type of HD, nature of the terrain and soil or material contaminated, 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 CW agent detector kits should be used to detect the presence of HD vapor in the field.

c. Cumulative Effect. Repeated exposures to HD produce cumulative effects. For example, repeated exposures to vapors from spilled HD can kill or produce disability by irritating the lungs and causing a chronic cough and pain in the chest.

5-6. Effects of Sulfur Mustard on the Eyes

a. Pathology, Symptoms, and Prognosis. The eyes are more susceptible to HD than either the respiratory tract or the skin. Figures 5-1 through 5-3 show effects of HD on the eyes. Conjunctivitis follows an exposure time of about one hour to a concentration barely perceptible by odor. A latent period of 4 to 12 hours follows mild exposure, after which there is lacrimation and a sensation of grit in the eyes. The conjunctivae and the lids become red and edematous. Heavy exposure irritates the eyes after one to three hours and produces some severe lesions. Functional "blindness" results from blepharospasm and pain, causing casualties to shut their eyes and keep them closed; permanent blindness, from agent damage to the cornea or the globe, can also occur. Casualties should 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 one to two weeks.

(2) Severe conjunctivitis with minimal corneal involvement (15 percent of the cases in World War I). Blepharospasm, edema of the lids, and conjunctivae occur, as may orange-peel roughening of the cornea. Recovery takes two to five 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 may require two to three months of hospital convalescence.

(4) Severe corneal involvement (about 0.1 percent of HD casualties in World War I). Ischemic necrosis of conjunctivae may be seen.

(5) In a small number of cases, delayed-onset keratitis may occur from as early as eight months to decades after exposure; this can progress to erosions and ulcerations.

SOURCE: PHOTOGRAPH PROVIDED BY THE IMPERIAL WAR MUSEUM, LONDON.

Figure 5-1. Sick call line of service members suffering from mustard eye irritation.

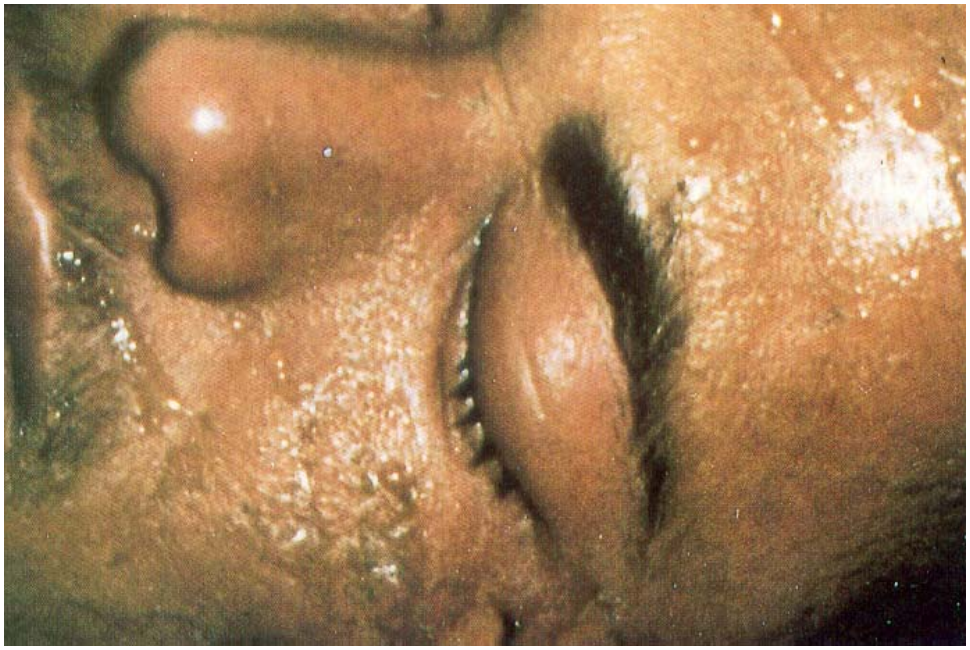


Figure 5-2. Casualty showing eye effects of mustard vapor.

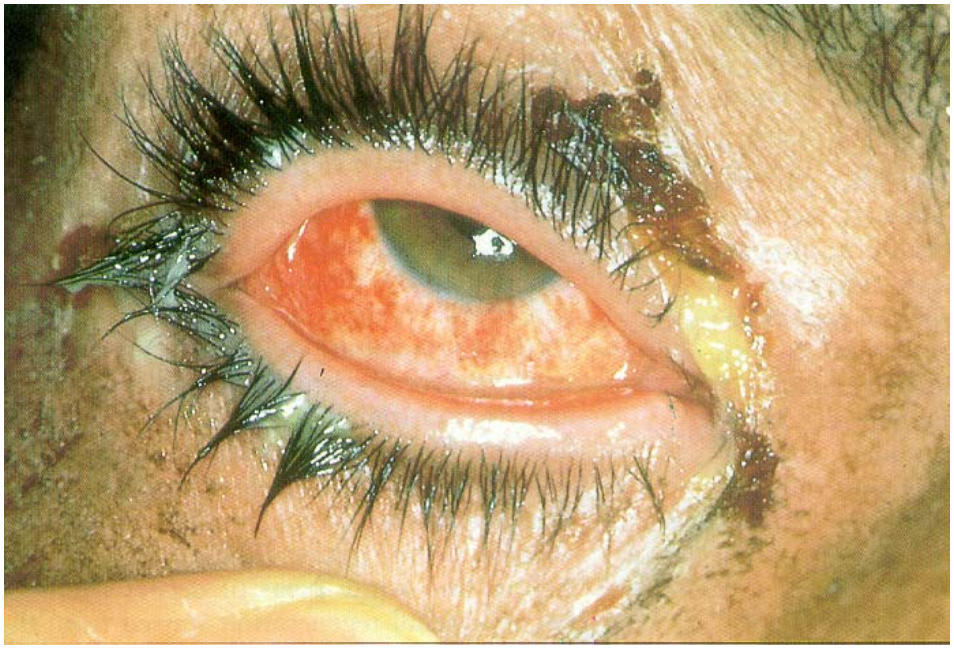


Figure 5-3. Casualty showing effects of mustard conjunctivitis.

b. Treatment.

(1) *Self-aid.*

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

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

(2) *Treatment of mustard conjunctivitis.*

(*a*) Mild lesions require little treatment. The lesions may become secondarily infected, and a combination eye ointment, such as tobramycin with dexamethasone, can be applied. Ophthalmic ointments will provide lubrication and minimal antibacterial effects. The application of sterile petroleum jelly or a sterile antibiotic ointment between the eyelids will provide additional lubrication and prevent the eyelids from sticking together.

(*b*) More severe injuries will cause enough edema of the lids, photophobia, and blepharospasm to obstruct vision. This obstruction of vision alarms patients. The lids may be gently opened to assure the patients that they are not blind.

(*c*) The best pain control is the use of 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 10 percent solution of sodium sulfacetamide should be instilled every four hours. Other antibacterial ophthalmic preparations may be substituted for sodium sulfacetamide, which produces a burning sensation on application.

(*d*) The eye must not be bandaged or the lids allowed to stick together. Prevent the eyelids from sticking together as described in 5-7b(2)*(a)* above. The accumulation of secretions in the conjunctival sac or pressure on the eye predisposes to corneal ulceration. To prevent complications, the patient should be 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 alleviate photophobia.

(3) *Treatment of infected mustard burns of the eye.* Secondary infection is a serious complication and increases the amount of permanent corneal scarring. If infection develops, initial treatment should be carried out with several drops of a 10 percent sodium sulfacetamide solution every two hours. After appropriate cultures, specific antibacterial preparations may be applied. Irrigation should be gentle and employed only to remove accumulated exudate. Control pain as described in (2)(*c*) above. Refer patients with secondary infection or other complications to an ophthalmologist. Local anesthetics should not be used.

5-7. Effects of Sulfur Mustard 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 HD. 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. In temperate weather the latent period for most vapor exposures is usually 6 to 12 hours.

(2) *Erythema.* Erythema gradually appears (2 to 48 hours post-exposure) and becomes brighter, resembling sunburn (Figures 5-4 and 5-5). 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, increased areas of pigmentation are left; this sequence is reminiscent of that seen in sunburn.

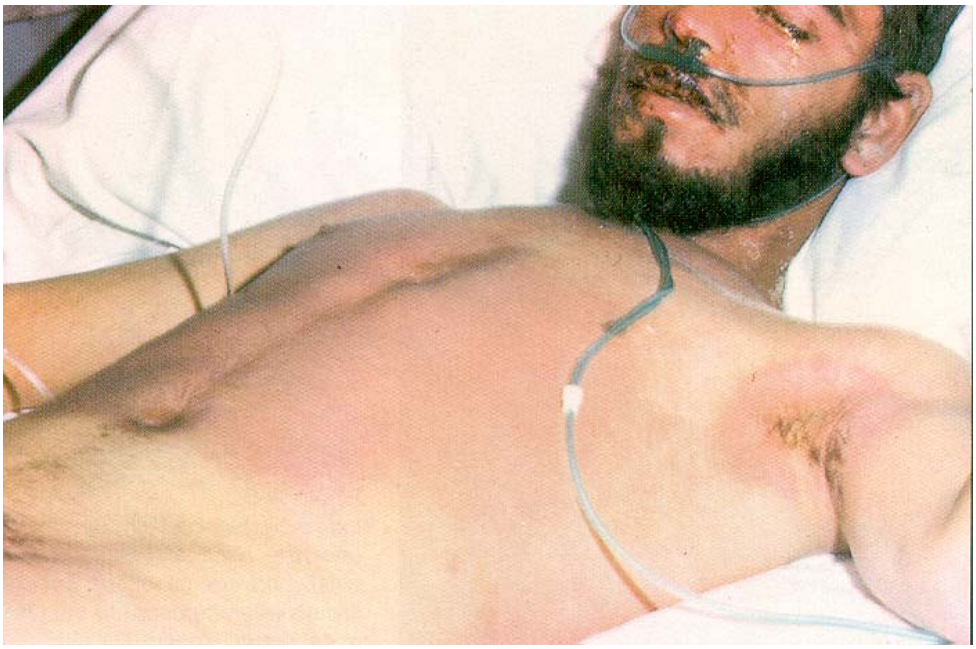


Figure 5-4. Mustard casualty showing less erythema inferior to navel, where clothing was tightly wrapped around waist. The sensitivity of warm, moist, and oily skin such as that of the axilla to mustard burns is also evident.

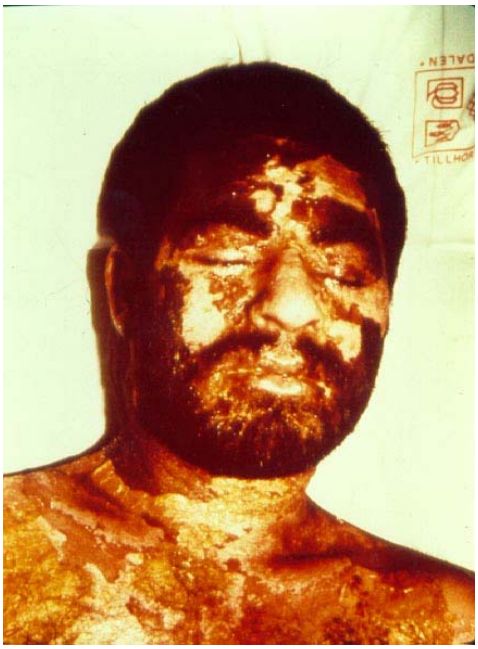


Figure 5-5. Casualty with generalized erythema and systemic intoxication.

(3) *Vesication.* Except with mild vapor burns, erythema is followed by vesication (Figures 5-6, 5-7, 5-8, 5-9, and 5-10). This is caused by the progressive development of liquefaction necrosis of the cells in the lower layers of the epidermis. Exudation of tissue fluid into the spaces so formed results in an intraepidermal vesicle. Clinically, multiple pinpoint lesions may arise within the erythematous skin; these enlarge and coalesce to form the typical blisters and bullae (which are unusually large, domed, thin-walled, and yellowish, and may be surrounded by erythema). The blister liquid is clear or slightly yellow and tends to coagulate. The blister fluid does not contain free (unfixed) HD and is not a vesicant. Liquid contamination of the skin classically results in a ring of vesicles surrounding a gray-white area of skin which, although necrotic, does not vesicate (see Figure 5-6). This pattern is often not present, and blisters may arise indiscriminately in the affected area (see Figure 5-7). As noted in paragraph 5-3*c* above, unreacted vesicant on contaminated patients may pose a hazard to other individuals coming in contact with them.

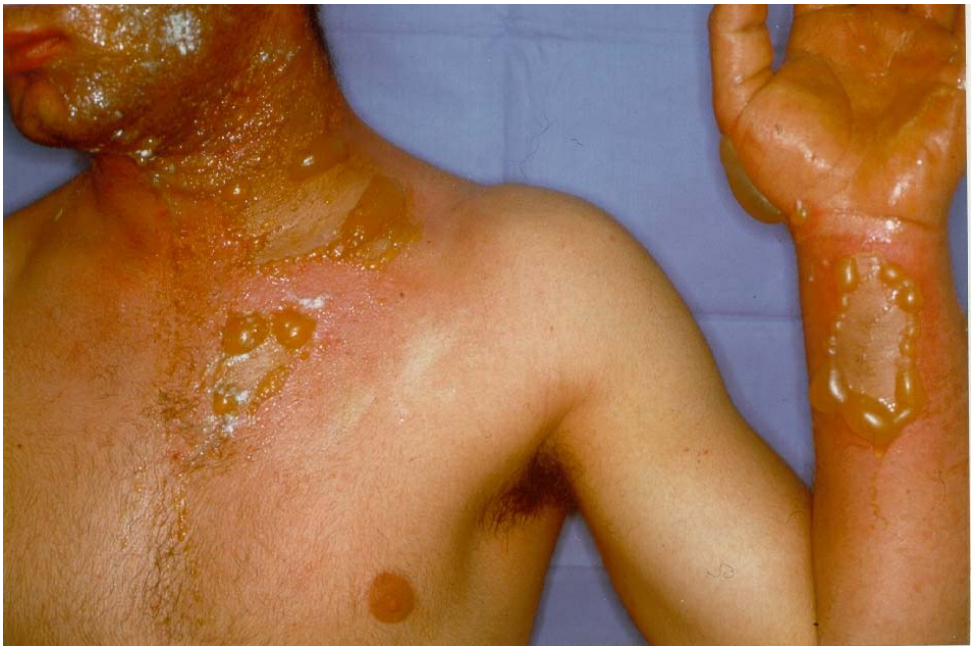
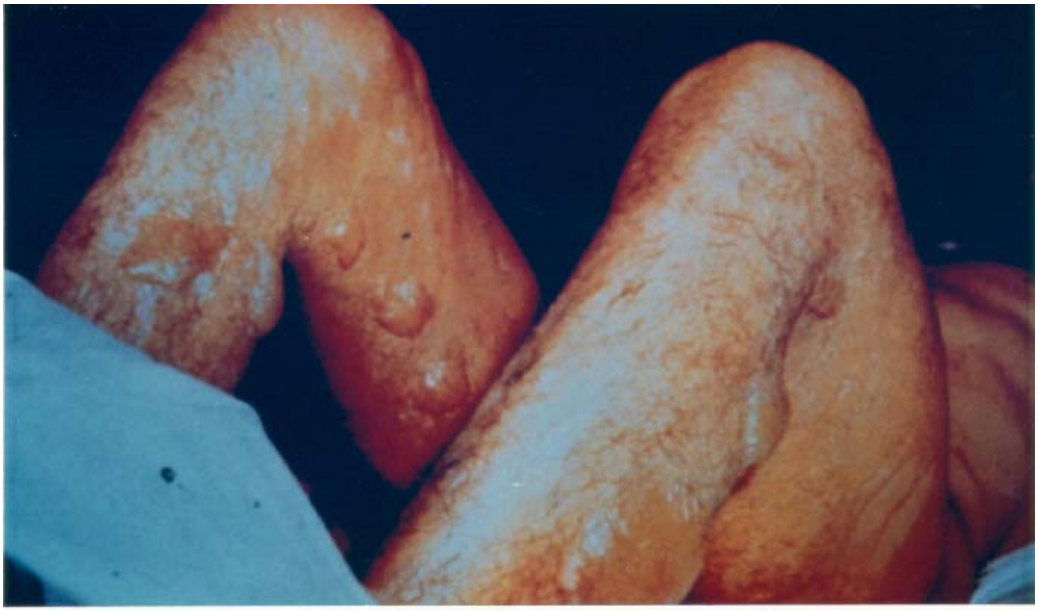
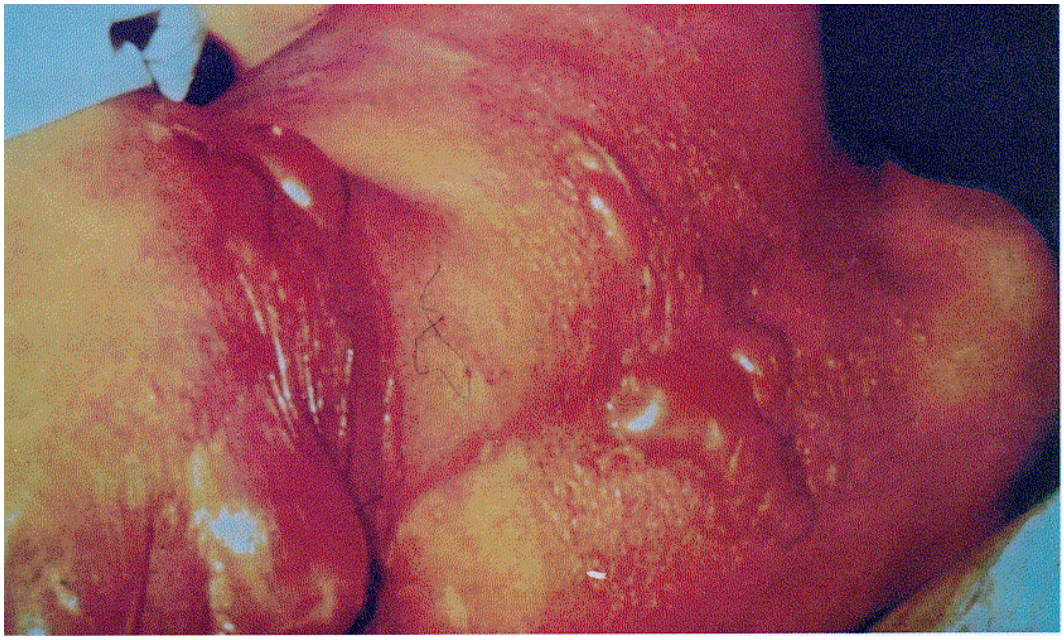


Figure 5-6. Casualty with classical rings of vesicles.



SOURCE: PHOTOGRAPH PROVIDED BY IRANIAN NEWS AGENCY.

Figure 5-7. Casualty with extensive mustard blisters without a ring-like distribution.



SOURCE: PHOTOGRAPH PROVIDED BY IRANIAN NEWS AGENCY.

Figure 5-8. Casualty with severe blisters on the back.

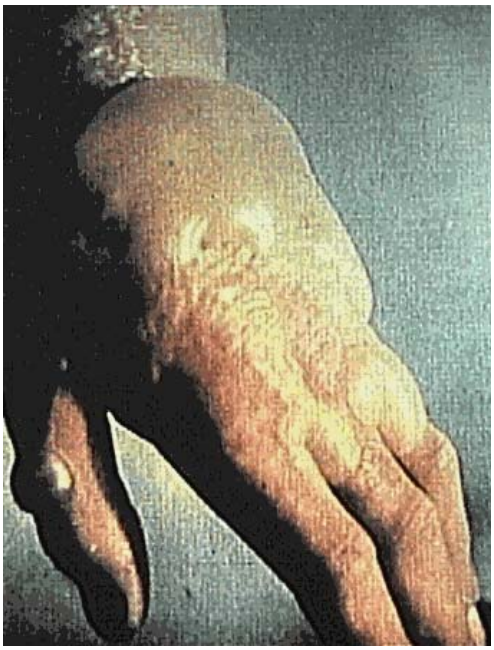


Figure 5-9. Large bulla from mustard exposure to dorsum of hand.

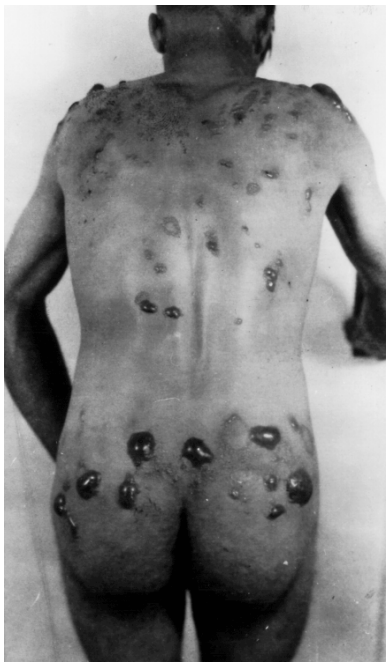
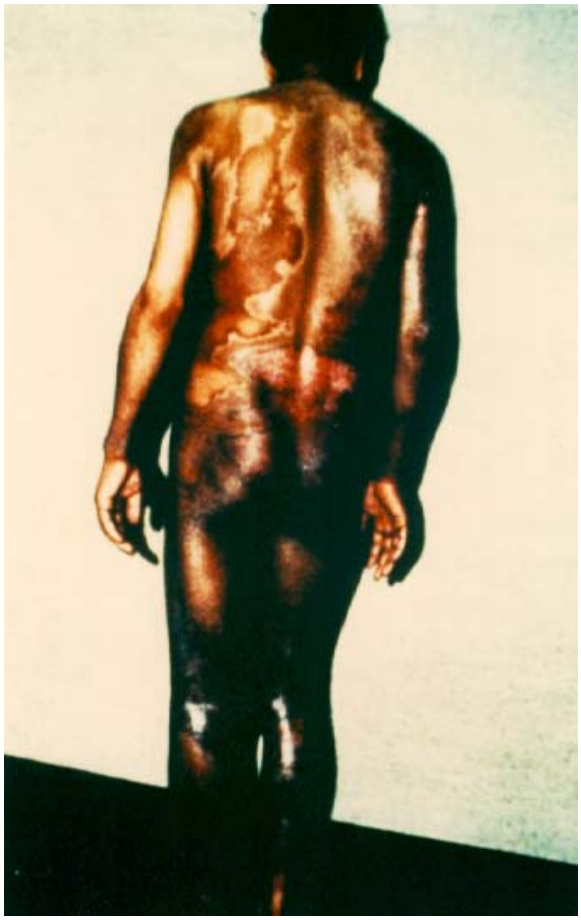


Figure 5-10. Casualty with widespread vesication.

(4) *Resorption.* If the blister does not rupture, resorption takes place in about a week. The roof forms a crust beneath which reepidermalization 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 dermis 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 (Figure 5-11). Classic "salt-and-pepper" pigmentation seen in some healing patients reflects epithelial regeneration arising from hair follicles and gradually spreading to confluence.



SOURCE: PHOTOGRAPH PROVIDED BY IRANIAN NEWS AGENCY.

Figure 5-11. Casualty with typical hyperpigmentation.

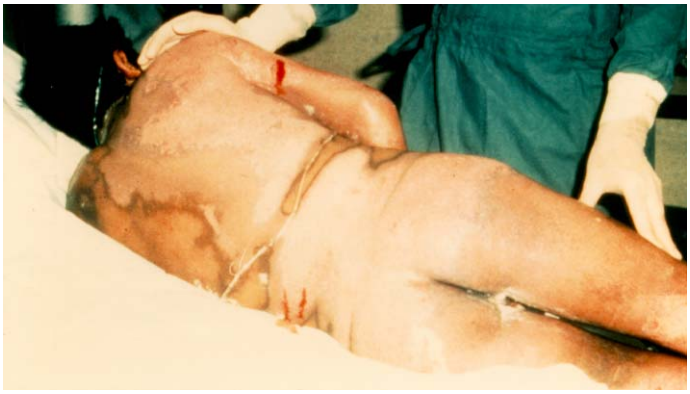
(7) *Hypersensitivity.* Mustard burns may lead to skin hypersensitivity to subsequent exposures.

b. Symptoms and Prognosis.

(1) A notable characteristic of the action of HD is its insidiousness. Exposures to HD are not accompanied by immediate cutaneous 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.

(2) Mustard erythema resolves at about the same rate as sunburn of like severity. Healing times for HD blisters vary widely with both severity and anatomical location. Areas of multiple pinpoint vesication usually heal, with skin peeling, in 1 to 2 weeks. Blisters of the face usually heal in 1 to 2 weeks. Blisters located in other areas may take up to 2 to 4 weeks to heal. If cutaneous injury results in full-thickness coagulation necrosis, skin grafting may ultimately be necessary. An HD burn of the skin is usually limited to the epidermis and does not require grafting.

(3) Moderate contamination of HD skin lesions with saprophytic bacteria, which cause no appreciable inflammatory reaction, does not seem to delay the HD burn healing. Active infection, with inflammation and purulent exudation, may increase the severity of the lesions and delay healing (Figures 5-12 and 5-13).



SOURCE: PHOTOGRAPH PROVIDED BY IRANIAN NEWS AGENCY.

Figure 5-12. Casualty with extensive mustard burns.

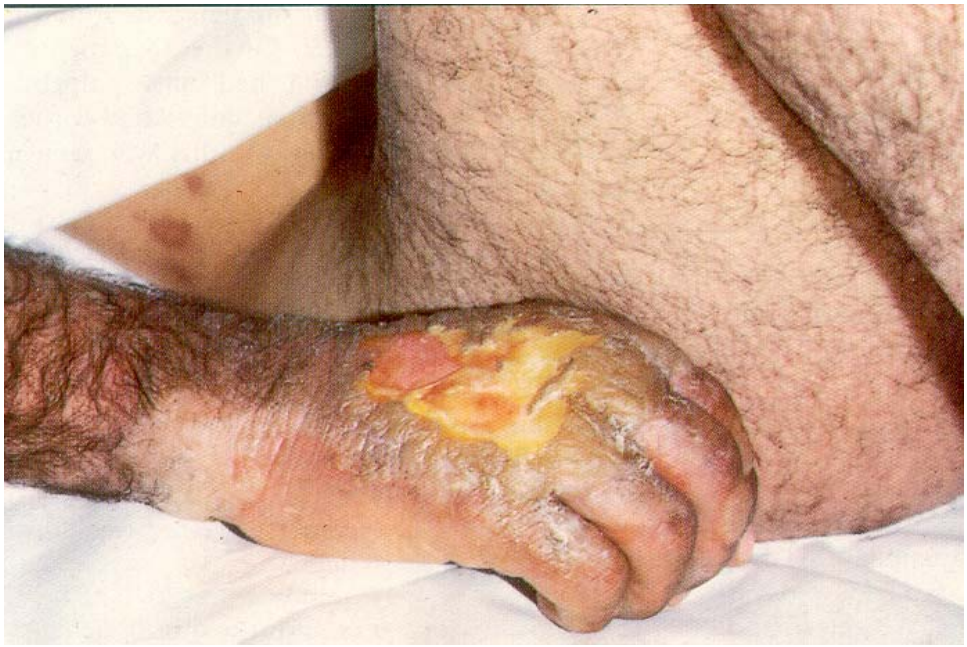


Figure 5-13. Purulence at base of disrupted mustard bulla on dorsum of hand.

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

(1) History of exposure.

(2) Absence of pain or discomfort at the time of contamination (L is irritating and painful within a few minutes of exposure).

(3) A zone of erythema surrounding blisters (not prominent with arsenicals). 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. The skin lesions of plant contact, however, are on exposed skin and tend to be linear in configuration. The earliest affected areas of skin from mustard are typically the skin folds, groin, and inner aspects of the extremities.

d. Decontamination of Casualties. Casualties who have liquid HD on skin or clothing and who have not been promptly decontaminated in the field will seldom be received by an MTF 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. Even if late decontamination fails to prevent the eventual development of blisters, it can still be life-saving by preventing continued absorption. It also can prevent spread of agent to other sites on the casualty or to personnel and equipment at the MTF. Promptly remove contaminated clothing from casualties outside the MTF to prevent more severe burns and to lessen the vapor hazard to patients and attendants. Cut away and discard hair contaminated with liquid HD. Decontaminate the exposed scalp and exposed skin with the M291 SDK. If short of these substances, use water with soap or shampoo for decontamination of skin and hair.

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; associated weeping and maceration may ensue. Treatment of such lesions with exposure to the air may be desirable. Care must be taken so that secondary infection of tissue does not occur.

f. Treatment of Mustard Blisters.

(1) *Forward Field Treatment*. Unless painful, leave the blister intact. In a clean environment, the blister may be antiseptically drained. Once the blister has broken, the antiseptic removal of the ragged roof will decrease the possibility of secondary infection. Application of burn creams or antibiotic ointments are best left to the hospital environment. Sterile dressings are applied to protect the open areas.

(2) *Field Hospital and Higher Levels*. Mustard blisters or deep lesions can be handled in several ways depending on severity, preferences, and available facilities:

- Leave small blisters, 1 cm or less in diameter, intact. Larger blisters that will likely rupture can be unroofed with subsequent cleansing and the application of an antibiotic cream or ointment.

- Larger blisters can be aspirated using a sterile needle, leaving the blister roof as a sterile dressing.

- The blister roof can be removed and artificial skin, cultured skin, or pig skin placed as a temporary dressing (skin). Infection negates this treatment and requires open care as initially described.

g. Treatment of Denuded Areas.

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

(2) Wounds that 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. Fluid Therapy. Mustard burns are associated with less fluid loss than are thermal burns of corresponding degree and area, and strict application of standard burn fluid-replacement protocols such as the Brooke and Parkland formulas may lead to fluid overload in an HD patient. Fluid replacement should be governed by clinical judgment.

i. 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.

5-8. Effects of Sulfur Mustard on the Respiratory Tract

a. Pathology.

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

(2) In the more severe cases, the pulmonary parenchyma shows congestion, mild patchy edema, and focal atelectasis. These changes may be sufficient to cause hypoxia and are frequently complicated by bacterial infection of the lungs, resulting in suppurative bronchitis and bronchopneumonia. In the preantibiotic era, the latter was responsible for almost all deaths following vapor exposures. Pulmonary edema is not the primary effect of low to moderate doses of HD but may be seen after massive exposures. The early mortality from HD among American troops in World War I (slightly more than 2 percent) was due almost entirely to such pulmonary complications following inhalation of vapor.

b. Symptoms and Prognosis. Respiratory tract lesions develop slowly and do not reach maximal severity for several days. Symptoms begin with hoarseness, which may progress to loss of voice. A cough (worse at night) appears early and later becomes productive. Fever, dyspnea, rhonchi, and moist rales may develop. Patients who develop pulmonary signs or symptoms within four hours of exposure to HD may have a grave prognosis. The incidence of bronchopneumonia is high. Convalescence is slow; the cough may persist a month or longer. Milder symptoms, such as hoarseness, last only one or two weeks.

c. Treatment of Respiratory Tract Injury Due to Mustard. Mild respiratory tract injury, with hoarseness and sore throat only, usually requires no treatment. Cough may be relieved by codeine-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. In severe cases, intubation may be required to ensure a patent airway, improve oxygenation, and aid in removal of secretions. If evidence of brochospasm is present, bronchodilators may be of benefit. 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. Prophylactic antibiotics in the absence of culture results are not recommended.

5-9. Systemic and Gastrointestinal Effects of Sulfur Mustard

a. Pathology.

(1) Ingestion of HD produces vacuoles and nuclear swelling of the epithelial cells of the gastrointestinal tract, with eventual necrosis and desquamation with hemorrhage. Absorption of HD from the intestinal lumen, or systemic distribution of large doses from any route of exposure and absorption, results in damage to the blood-forming organs mentioned in paragraph 5-10*a*(2) below.

(2) With lesser skin or respiratory exposures to HD, systemic distribution may occur without the development of grossly apparent acute systemic lesions. With absorption and systemic distribution of 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 usually subsequent lymphocytopenia, but there may be initial lymphocytosis.

(3) Mustard also damages deoxyribonucleic acid (DNA), is mutagenic, and is classified by the International Agency for Research on Cancer (IARC) as a Group 1 carcinogen (carcinogenic to humans). The incidence of cancers of the nasopharynx, larynx, and lung is increased following chronic occupational exposure to HD and theoretically could be elevated following a single acute exposure.

b. Symptoms.

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

(2) Exposure of only the skin to HD 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 HD 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.

(3) Sufficiently high doses of HD lead to bone marrow suppression and consequent pancytopenia. This tends to occur between 7 and 21 days after exposure in most cases. The first blood cell fraction to drop is the lymphocytes; relative lymphopenia is a warning sign of impending pancytopenia. Such patients are at high risk for sepsis.

c. Prognosis.

(1) With mild to moderate field exposures to HD vapor, deaths rarely occur from systemic effects of absorbed HD. Death may occur from prolonged exposures to high concentrations of HD vapor or, in instances of extensive liquid contamination of the skin, where decontamination is neglected or unduly delayed. The percentage of body surface area involved in skin contamination is not correlated with mortality, probably because of factors such as agent concentration, permeability characteristics of involved skin, and concomitant vapor exposure. Nevertheless, skin contact with more than about 1 teaspoon (5 mL) of liquid HD is likely to cause fatal systemic effects. This would be roughly equivalent to 20 percent of the body surface area. 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.

(2) Ingestion of HD is rare but can cause severe injury, including death.

d. Self-Protection. Never drink water that has been subjected to chemical attack until it has been certified as fit to drink by the Medical Department. Never eat foods that have been exposed to liquid

vesicants, unless in sealed cans or aluminum-laminated pouches (meal, ready to eat [MRE] pouches), until examined by US Army veterinary personnel and certified as safe to eat. Refer to FM 3-5, FM 4-02.7, and TB MED 577 for additional information.

e. Treatment of Systemic Effects of 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.

(2) Sulfur donors such as sodium thiosulfate decrease systemic effects and elevated the lethal dose for 50 percent of those exposed (LD50) when given before exposure or within 20 minutes after exposure in experimental animals (it has been theorized that the time during which it is effective correlates with the time that systemically absorbed HD remains in the circulation). Its efficacy is very doubtful if given later.

(3) One study in non-human primates demonstrated that granulocyte colony stimulating factor (G-CSF) reduced the severity and duration of HD-induced pancytopenia.

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

f. Secondary Bacterial Infection in Blister Agent Burns.

(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 HD lesions is remarkably low, according to observations made at experimental installations.

(2) Secondary infection becomes manifest several days after injury. Infection is particularly disabling when it involves the feet, hands, genitals, or tissue overlying the joints of the limbs.

(3) 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.

(4) Secondary infection is an uncommon complication of mild HD conjunctivitis and normally will not prevent an individual from continuing duty.

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

g. Long-term Sequelae from Acute Exposure to Mustard.

Exposure to HD has been reported to be associated with a variety of chronic diseases affecting especially the lungs, skin, eyes, and the hematopoietic system. For further information, see Borden Textbook of Military Medicine.

Section III. NITROGEN MUSTARDS

5-10. 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 encountered 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 HD. 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.

5-11. Clinical Presentation and Management

Clinical presentation and management of HN casualties are identical to that of HD casualties.

Section IV. ARSENICAL VESICANTS

5-12. Properties

a. These agents are organic dichloroarsines. The main ones are phenyldichloroarsine (PD) and chlorovinyldichloroarsine, or L. Ethyldichloroarsine (ED) and methyldichloroarsine (MD) 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. Vapors of arsenical agents are toxic, but they are so initially irritating to the eyes and the respiratory tract that eye closure and avoidance of further inhalation when possible will tend to limit vapor damage. 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 as long as the warning irritation is heeded and further inhalation is avoided.

5-13. Effects of Arsenical Vesicants on the Eyes

a. Pathology, Symptoms, and Prognosis. Arsenical vesicants cause severe damage to the eye. Pain and blepharospasm occur within seconds to minutes of contact. Edema of the conjunctivae 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,

may induce pannus formation, or may 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 rapidly produce a gray scarring of the cornea, like an acid burn, at the point of contact. Necrosis and sloughing of both bulbar and palpebral conjunctivae may follow very heavy exposure. All injured eyes are susceptible to secondary infection. Mild conjunctivitis due to arsenical vesicants heals in a few days without specific treatment. Severe exposure may cause permanent injury or blindness.

b. Treatment. Treatment is largely symptomatic. In severe cases, the systemic use of morphine may be necessary 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 solutions (example, normal saline). Occlusive dressings and pressure on the globe must be avoided.

5-14. 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 and then by vesication that 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. Classically, an L blister arises as a single lesion in the center of an area of erythema and expands outward rather than forming the ring-like distribution around a central grayish area as seen with HD. 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 and may be vesicating. Within the dermis and subcutaneous tissue, there is deeper injury to the connective tissue and muscle, greater vascular damage, and more severe inflammatory reaction than is observed in mustard burns. In large, deep, arsenical vesicant burns, there may be considerable tissue necrosis, gangrene, and slough. Lewisite damages capillary endothelium, and the resulting increase in capillary permeability leads to local edema at the site of skin contact.

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 or very shortly after contact with liquid arsenical vesicants usually gives sufficient warning to allow for prompt decontamination and avoidance of deep burns in conscious victims. After about five minutes of contact, there appears a gray area of dead epithelium resembling that seen in corrosive burns. Erythema is like that caused by mustard but is more painful. Local edema may be prominent. 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. Pain from blisters will diminish after 48-72 hours.

c. Prognosis. The erythema of arsenical vesicants usually resolves more rapidly, and with less pigmentation, than that due to mustard. 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) The treatment of arsenical skin and eye injury is entirely supportive and similar to that

of HD. The antidote British anti-Lewisite (BAL) is not available.

(2) Some blistering is inevitable in most arsenical vesicant cases that arrive at medical treatment facilities. 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. The fluid loss from L is greater than that from a corresponding mustard blister because of the additional effect of L to damage capillary endothelium and thus cause capillary leakage. 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 mafenide acetate burn cream, or silver sulfadiazine topical burn cream.

5-15. Effects of Arsenical Vesicants on the Respiratory Tract

a. Symptoms. The vapor of arsenical vesicants is so irritating to the respiratory tract that a conscious casualty will tend immediately to put on a mask. Severe respiratory injuries are likely to occur only among the wounded who cannot put on masks and those who are caught without masks. The respiratory lesions are similar to those produced by mustard except that the propensity of Lewisite to damage capillary endothelia in the lung means that pulmonary edema, sometimes accompanied by pleural effusion, is to be expected after high doses of the agent.

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 begins with that for mustard respiratory injury (see paragraph 5-9*c*) plus preparation for pulmonary edema. See also recommendations (paragraph 5-17*c*) for the treatment of systemic effects of arsenical vesicants.

5-16. Systemic Effects of Arsenical Vesicants

a. Pathology and Symptoms. Absorbed arsenical vesicants 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, low blood pressure, and hypovolemic shock in animals.)

b. Prognosis. Burns severe enough to cause shock and other systemic effects 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.* Following prompt decontamination with the M291 SDK or with soap and water, follow treatment guidelines for mustard with the addition of attention to the development and treatment of pulmonary edema.

5-17. 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 they tend to confuse and make diagnosis difficult.

Section V. PHOSGENE OXIME

5-18. Properties

a. Phosgene oxime (chemical name dichloroformoxime) is an example of the class of CW agents called urticants (or nettle agents). These agents primarily irritate and corrode the skin and mucous membranes. They differ from mustard by producing an immediate sensation of pain, by producing wheals or hives instead of true blisters, and by producing severe tissue necrosis. The 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, crystalline solid readily soluble in water, as a liquid (between 104° F [40.0° C] and 129° F [53.9° C]), or as a gas (above 129° F [53.9° C]). Phosgene oxime has a significant vapor pressure. It is especially effective as a liquid.

5-19. 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 that 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 30 minutes. At about 24 hours, the original blanched area acquires a brown pigmentation. At one week, an eschar forms in the pigmented area; and at about three 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 two months.

5-20. 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.

5-21. 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 SDK for skin decontamination. If the M291 kit is not available, flush the contaminated area as rapidly as possible with copious amounts of soap and water to remove any CX which has not yet reacted with tissue.

5-22. Treatment for Phosgene Oxime Injury

Treat as any other ulcerated necrotic skin lesion with due consideration of other supportive measures, as with HD. Debridement and excision may be needed.

CHAPTER 6

INCAPACITATING AGENTS

6-1. General

a. An incapacitating agent is a CW agent that produces temporary disabling conditions that persist for hours to days after exposure (unlike that produced by riot control agents, which are usually momentary or fleeting in action). Medical treatment, while not essential, may facilitate rapid recovery. The term "incapacitating agents" includes 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) Unlikely to produce permanent injury in concentrations that are militarily effective.

b. Incapacitating agents are not considered to include—

(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 that 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 constraints 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, only two general types of incapacitating CW agents are likely to be encountered in military use. The two types of incapacitating agents of military relevance are as follows:

(1) *Central nervous system depressants*.

(*a*) These compounds produce their effects by interfering with cholinergic synapses in the central nervous system. An example of this type of agent is 3-quinuclidinylbenzilate (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.

(c) Opioid narcotics have multiple central nervous system effects, including CNS depression. In sublethal doses, these narcotics can cause listlessness, significant sedation, and affect alertness, attention, and problem solving. Fentanyl derivatives are alleged to have been used to break the siege of a Moscow theater in 2003.

(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 that appears to act in this manner is d-lysergic acid diethylamide (LSD); similar effects are sometimes produced by large doses of amphetamines.

d. This chapter focuses on the glycolate anticholinergic class of incapacitating agents.

6-2. Diagnosis

Currently, field laboratory methods do not permit isolation and identification of specific agents in environmental or body fluids (blood, urine, or cerebrospinal fluid). Diagnosis rests almost entirely upon clinical presentation, combined with whatever field intelligence or detector system data that may be available. Following a suspected incapacitating agent attack, the following steps should be taken:

a. 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 6-1.

Table 6-1. 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 (SUCH AS BZ), INDOLES (SUCH AS LSD), CANNABINOLS (SUCH AS MARIJUANA), ANXIETY REACTION, OTHER INTOXICATIONS (SUCH AS ALCOHOL, BROMIDES, BARBITURATES, LEAD)
DRYNESS OF MOUTH, TACHYCARDIA AT REST, ELEVATED TEMPERATURE, FLUSHING OF FACE; BLURRED VISION, PUPILLARY DILATION; SLURRED OR NONSENSICAL SPEECH, HALLUCINATIONS THAT ARE EASILY DESCRIBED AND DECREASING IN SIZE OVER TIME, DISROBING, MUMBLING AND PICKING BEHAVIOR, STUPOR AND COMA.	ANTICHOLINERGICS
INAPPROPRIATE SMILING OR LAUGHTER, IRRATIONAL FEAR, DISTRACTIBILITY, DIFFICULTY EXPRESSING SELF, PERCEPTUAL DISTORTIONS (INCLUDING ABSTRACT AND DIFFICULT-TO-DESCRIBE HALLUCINATIONS); 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. Characteristics common to all or most casualties, rather than atypical features, should be identified. 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). Presence of these symptoms in some casualties 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. Hallucinations produced by psychedelic indoles (such as LSD) are different from those produced by glycolate anticholinergic compounds such as BZ. Hallucinations from indoles tend to be abstract and geometric and are associated with synesthesia (sensory crossover) and a sense of oneness with the universe. Subjects may believe that they have special insights into reality; however, these insights are ineffable, that is, difficult to describe to others. In contrast, anticholinergic hallucinations tend to be easily described, although often odd. They are often Lilliputian, that is, the objects described tend to decrease in size over time. Both indole and anticholinergic glycolate casualties may remain quite aware of their environments and may comprehend quite well, although they may react inappropriately. Patients with anticholinergic exposure may in fact realize their hallucinations and illusions are unreal but be unable to rid themselves of these abnormal perceptions.

d. Anticholinergic glycolates block the action of acetylcholine at muscarinic sites in the peripheral nervous system as well as in the CNS and cause peripheral effects that in general are the opposite of those produced by nerve agents. This constellation of signs and symptoms constitutes a characteristic anticholinergic toxidrome. Pupillary dilation (mydriasis) and paralysis of accommodation is classically described as the patient's being "blind as a bat." Lack of cholinergic activation of sweat

glands leads to anhidrosis (a patient who is "dry as a bone") and a resulting rise in core temperature; thus, the patient is "hot as a hare." In an attempt to dissipate the extra heat, superficial blood vessels in the dermis dilate, leading to flushing, or a patient who is "red as a beet" (the so-called "atropine flush" named for the prototypical anticholinergic). Although tachycardia is the usual response to anticholinergics, BZ is often associated with a rebound after a day or two to a normal heart rate or even bradycardia. The CNS component of the anticholinergic toxidrome consists of the characteristic hallucinations described in paragraph 6-2*c* along with semiautonomous behavior such as plucking or picking at imaginary objects (so-called "phantom behavior" or "woolgathering") and disrobing, mumbling, social disinhibition, lethargy progressing through stupor to coma, and paranoia as CNS symptoms resolve. Patients with the CNS component are sometimes referred to as being "mad as a hatter," although this description originally referred to supposed mercury intoxication in hatters working mercury into felt. Identification of the combination of the peripheral-nervous-system signs and symptoms ("blind as a bat," "dry as a bone," "hot as a hare," and "red as a beet") with the CNS symptoms ("mad as a hatter") is helpful in the clinical confirmation of exposure to anticholinergic compounds.

e. Since atropine is also an anticholinergic compound, overdose may produce a similar signs and symptoms and may be confused with other glycolate anticholinergic poisoning.

6-3. 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 inhalation of the agent. With some agents, the percutaneous route may be used (especially with lipophilic solvents as adjuvants); 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 SDK can be used (Appendix 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 (the minimal latent period is probably 20 to 30 minutes after inhalational exposure). 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) Weapons and other potentially harmful items should be removed from the possession of suspected casualties. These include cigarettes, matches, medications, sharp objects (including autoinjectors), and small items that might be accidentally ingested. Delirious casualties have been known to attempt to eat items bearing only a superficial resemblance to food.

(2) 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.

(3) If the body temperature is elevated above 102° F (38.9° 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 (22.3 to 23.9° 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.

(4) Reassurance and a firm, but friendly, attitude by personnel administering first aid will be beneficial. Even if a casualty is incoherent and may not understand what is being said, reassurance should always be attempted; however, prompt and vigorous restraint and early evacuation to an MTF

remains paramount.

(5) 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 due to paralysis of the bladder smooth muscle. Moistening the mouth with an astringent swab may be comforting and will reduce the foul breath associated with membrane parching. Rehydration, orally or parenterally, should be instituted if clinical signs of dehydration occur.

6-4. Treatment

a. Anticholinergics. Certain cholinesterase inhibitors (such as physostigmine) are highly active antagonists of the centrally active anticholinergics. Neostigmine and pyridostigmine are ineffective because they ordinarily do not cross the blood-brain barrier. In contrast, physostigmine readily passes into the brain. Treatment with 2 to 3 mg of physostigmine salicylate IM will be required to alleviate symptoms. Repeated injections at intervals of approximately 15 minutes to 1 hour may be required to produce a sustained level in tissues. Once a desirable effect is achieved, it should be maintained by oral administration, slow intravenous (IV) injection, or infusion. Physostigmine is a reversible anticholinesterase compound and controls signs and symptoms of anticholinergic poisoning only as long as its inhibition of cholinesterase lasts. Therefore, doses of 2 to 4 mg every one to two 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 days. Physostigmine does not shorten the clinical course of anticholinergic poisoning but only controls the symptoms during the course of the poisoning. Oral dosing should replace IV therapy as soon as possible (2 to 5 mg every one to two hours).

NOTE

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

2. An overdose of physostigmine can result in cholinergic toxicity up to and including muscle weakness, increased secretions, temporary apnea, and seizures. Hypertension, dysrhythmias, and hallucinations have also been reported. The presence of hallucinations may indicate either agent toxicity or overdose of the antidote. Generally, the presence of associated nerve-agent-like effects will point to physostigmine overdose. 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. Many patients will be able to be managed by restraint, observation, and evacuation without the administration of physostigmine.

b. Indoles. No true antagonist to the indoles is 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 diazepam.

c. Cannabinols. Although stimulants such as d-amphetamine (15 mg) can antagonize the sedation and indifference induced by marijuana-like substances, their routine use is discouraged. Although amphetamine may slightly potentiate 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 (including airway management and circulatory support) 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). For example, if opioid use is suspected, naloxone may be administered in accordance with standard protocols. The judgment of the medical officer remains the only useful guide to action in these complex and unforeseeable circumstances.

PART TWO: CONVENTIONAL MILITARY CHEMICAL INJURIES

RIOT CONTROL AGENTS (IRRITANT AGENTS AND VOMITING AGENTS)

CHAPTER 7

Section I. IRRITANT AGENTS

7-1. General

Irritant agents (lacrimators) in very low concentrations act primarily on the eyes and mucous membranes, causing intense pain and lacrimation. Higher concentrations irritate the upper respiratory tract and the skin and sometimes cause nausea and vomiting. Although rare, certain irritant agents have been implicated in deaths, usually in confined spaces and due to either hypersensitivity reaction or acute exacerbation of restrictive lung disease. Lacrimators may be dispersed as fine particulate smoke (aerosols) or in solution 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 agents, such as CK and PS, also induce lacrimation.

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 uniform should be secured at the neck, wrists, and ankles.

b. Following exposure, clothing and individual equipment should be inspected for agent residue. If found, individuals should change or decontaminate clothing to protect themselves and other unmasked persons. Decontaminate CS-contaminated clothing by airing for a few minutes. Bleach, which produces irritating byproducts from these agents, should not be used for decontamination.

7-3. Properties

a. Agent CS. Agent CS is a white crystalline solid that melts at 194° F (90.0° 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.

b. Agent CR. Agent CR is a pale yellow crystalline solid that melts at 163° F (72.8° C) and is stable in organic solutions. It has limited solubility in water and is not hydrolyzed in aqueous solutions. It has a pepper-like odor. The agent is currently in solution only 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 lower. Agent CR differs from CS in being less toxic when inhaled, although its effects on the skin are more pronounced and longer lasting. It is also more persistent in the environment and on clothing.

c. Agents CN and CA. Agent CN is a white crystalline solid that boils at 478° F (247.8° C) and freezes at 129° F (53.9° C). Agent CN may also be used in liquid form in appropriate solvents. CN is about one-tenth as potent as CS. Agent CA is usually a liquid, with a boiling point of 468° F (242.2° C) and a freezing point of 77° F (25.0° C). 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 bluish-white clouds at points of release. Solid agents are 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. Irritation to the point of functional 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. Because CS and other agents can be disseminated as small-particle aerosols, foreign body eye injuries can result from inadvertent impaction into the cornea. 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. These agents may exacerbate pre-existing pulmonary disease.

(2) *Skin.* Warm, moist skin (especially on the face, neck, ears, and skin 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 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 seen in rare cases of exposure to CS.

b. Agent CR.. Agent CR is similar in effect to CS, but the minimal effective concentration is lower and the lethal dose (lethal concentration) (LCt) is higher. Thus, the safety ratio is greater than for CS. 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. Their effectiveness as lacrimators is 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. Particle impaction in the eyes is also a hazard when individuals are in close proximity to disseminating devices. Extremely high concentrations of these agents in enclosed spaces may cause tracheitis, bronchitis, pulmonary edema, or cerebral edema. Exposures of this magnitude are rare.

(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.

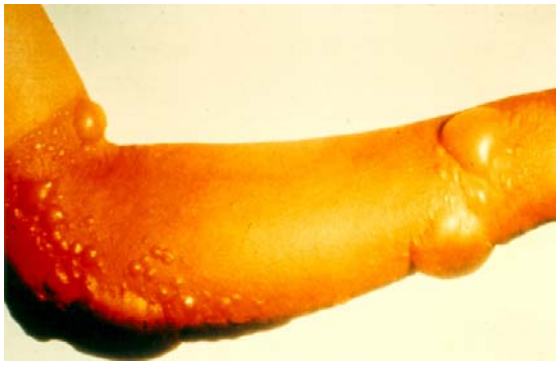


Figure 7-1. Skin burn caused by exposure to CS.

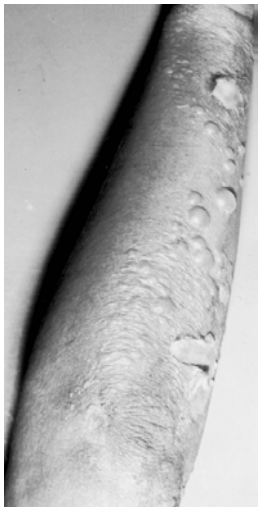


Figure 7-2. Desquamation of skin caused by CS burns.

7-5. Diagnosis

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

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

c. Agents CN and CA. Diagnosis 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. Headache and depression may also 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. If exposure has been heavy, significant erythema and, rarely, blisters may develop. The cutaneous reaction can be prevented by immediately flushing the skin with copious amounts of water. Do not use bleach.

CAUTION

Do not use bleach.

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 may be 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. Retained particles after irrigation should be treated as foreign bodies.

b. Skin. Ordinarily, early (up to one hour) erythema and stinging sensations 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, depending upon severity. Cases with blisters should be managed as a second degree burn. Secondary infections are treated with appropriate antibiotics. If significant pruritus occurs, an oral antihistamine should be used. Water, with or without soap, is the primary means of decontamination.

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 damage to the respiratory tract from pulmonary agents (Chapter 2).

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 also 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 and 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 secretions are greatly increased and quantities of ropy saliva flow from the mouth. Nausea and vomiting are prominent. Malaise and 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 then donned, 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 that 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 antiemetics IM, IV, orally, or rectally. Aspirin or acetaminophen 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. 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, 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. There are several newer obscurants, such as phthalic acid and graphite-based smokes, now used to defeat infrared (IR) and millimeter and microwave (mm-wave) technologies. 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 is similar to the bulk materials from which they are generated. The ignition of the HC mixture produces primarily zinc chloride and only traces of phosgene (CG) and carbon monoxide (CO), although several other pyrolysis products can also be detected and may vary in clinical importance according to the conditions of exposure. Burning phosphorus mixtures produce smokes composed of highly concentrated (60-80 percent) polyphosphorus acids.

b. Most smokes are not hazardous in concentrations that are useful for obscuring purposes. Any smoke can be hazardous to health if the concentration is sufficient or if the exposure is prolonged, and HC smoke is particularly dangerous in this regard. 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 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. In training, terephthalic-acid smoke should be substituted for HC smoke. Never use HC munitions indoors or in closed compartments. Should oil smoke be generated in closed spaces, personnel must immediately evacuate the area or wear self-contained air supply equipment.

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, grained aluminum powder, and zinc oxide. On burning, the mixture produces zinc chloride that 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. The smoke of HC has a sharp, acid odor, even at moderate concentrations. The smoke of HC can cause nose, throat, and chest irritation, and cough (typical central pulmonary effects) as well as slight nausea in some individuals. More serious are its effects on the peripheral compartment (the gas exchange region) of the respiratory tree, effects that can lead to pulmonary edema and death from exposures to sufficiently high concentrations for as little as one minute. In addition, patients recovering from pulmonary edema induced by HC smoke are at risk of developing late-onset pulmonary fibrosis (cryptogenic organizing pneumonia) (see paragraph 2-1).

b. Pathology. The irritant and corrosive action of zinc chloride may produce irritation and hyperemia of the larynx, trachea, and large bronchi along with functional narrowing of the smaller air passages. Irritation may be mild, and its absence does not exclude the possibility of severe or even fatal damage to the peripheral compartment of the respiratory tract. Chemical pneumonitis may result from moderate exposures. Death from exposure to HC smoke may occur quite rapidly from irritative laryngospasm, acutely from central pulmonary effects (acute tracheobronchitis, which may prove fatal within hours), within hours to days from pulmonary edema (peripheral pulmonary damage), or much later, in patients that after apparent recovery then develop cryptogenic organizing pneumonia, with growth of cuboidal epithelium from the bronchioles into the alveoli (sometimes completely lining or filling the alveoli) and development of fibrotic pulmonary changes with marked hypoxia. This late-onset process appears to be immunologically mediated.

c. Symptoms. The smoke of HC can cause a range of clinical effects. Central pulmonary damage resulting from disruption of smooth laminar bulk flow in central airways creates turbulence, which can be recognized clinically by airway noise: paroxysmal coughing, sneezing, hoarseness, inspiratory stridor, and wheezing. Nausea and retching may accompany these signs. With supportive therapy, these symptoms resolve rapidly, often within minutes to hours. Damage to peripheral airways and air spaces results in the accumulation of fluid, initially within alveolar septa; it is the thickening of these normally thin-walled septa that cause the dyspnea that is usually the first clinical indicator of incipient pulmonary edema. The dyspnea ordinarily occurs after a clinically asymptomatic latent period that is inversely correlated to inhaled dose and may last several hours. Objective signs and radiological and laboratory abnormalities may be absent at this stage, but the dyspnea by itself is an important clue that must not be overlooked. Finally, case reports of accidental exposure to moderate and high concentrations of HC smoke have shown that a certain percentage of victims will appear to recover from mild to more severe pulmonary edema only to develop fever, rapid pulse, malaise, shortness of breath, retrosternal pain, abdominal cramps, and cyanosis up to 48 hours after exposure. Chest radiographs associated with severe exposures have demonstrated a dense, diffuse, infiltrative process present in one or both lung field(s). Repeat radiographs 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 secondary bacterial infection, may lead to 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. It is important to follow medical recommendations even if all you are feeling is shortness of breath.

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 or acetaminophen will help relieve general discomfort. Oxygen therapy is required, and steroids should be administered prophylactically to reduce the risk of late-onset pulmonary fibrotic changes (see paragraph 2-9*e*).

f. Prognosis. 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 one to two weeks. In severe exposures, survivors may have reduced pulmonary function for some months after exposure. The severely exposed patient may develop marked progressive dyspnea, cyanosis, and fibrosis and may 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 acidic 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, upper (central) airways, and skin. Because of its extremely corrosive properties, it has become obsolete for US military use.

b. Pathology. Local inflammation of the eyes, respiratory tract (central pulmonary effects), 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. 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. Coughing (which may be explosive), soreness in the chest beneath the sternum, bronchoconstriction (especially in individuals with sensitized airways), 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 that 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 induced with the use of atropine sulfate.

(2) *Skin.* Wash irritated skin or skin burns with water (with or without soap); this may be followed by washing with a sodium bicarbonate solution. After washing, treat the burns as thermal burns of like severity.

(3) Respiratory tract. Administer warm, moist air. Use bronchodilators as clinically indicated.

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 that 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, but it is not commonly used.

b. Pathology. Liquid FM produces acid burns of the skin or eyes. It may also cause irritation of the upper (central) airways.

c. Symptoms. Exposure of the eyes to the spray will cause conjunctivitis with lacrimation 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. Titanium tetrachloride smoke may provoke bronchospasm in individuals with underlying reactive airway disease.

d. Self-Aid. Wear the mask in all concentrations of FM smoke that irritate the nose or the throat. Wash any liquid splash off the skin with water. If spray or liquid splash enters 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. Treatment is similar to that for FS (paragraph 8-5*e*).

f. Prognosis. The prognosis is good except in rare instances in which corneal erosions 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. The particles are converted by moist air into phosphoric acid (paragraph 9-4). White phosphorus is usually dispersed by explosive munitions. The WP smoke irritates the eyes and nose in moderate concentrations. In an artillery projectile, WP wedges 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 re-ignite if the felt is crushed and the unburned WP is exposed to the atmosphere.

b. Pathology. For WP burns, see paragraph 9-4. Inhaled smoke irritates the upper respiratory tract.

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 has been reported from exposure to WP smoke at usual field concentrations.

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 serious effects produced by exposure to these smokes. Anecdotally, discoloration of the urine has been noted.

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), white phosphorus (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 phosgene (CG), chlorine, carbon monoxide (CO), and hydrochloric acid. The field protective mask does NOT protect against some products of combustion 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 (1982.2° C) and scatter molten iron. Explosive charges are frequently added, which makes 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, treat as any other thermal burn.

9-3. Magnesium and Its Alloys

Magnesium burns at about 3600° F (1982.2° 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 MG 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 pointed object. It may be possible to remove some particles by rubbing with a wet cloth.

(4) Report for treatment as soon as the mission 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. Visible WP can then be removed. 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 and remove particles of WP that 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 filled with water, sand, or, preferably, oil 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, wet 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 promptly removed. 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. Separate the lids and instill a local anesthetic to aid in the removal of all embedded particles. Once all particles have been removed from the eyes, atropine ophthalmic ointment should be instilled. Transfer the patient to the care of an ophthalmologist as soon as possible.

NOTE

Cupric (copper) sulfate, used by US personnel in the past and still being used by some nations, may produce kidney and cerebral toxicity as well as intravascular hemolysis. It is no longer used to counteract white phosphorus.

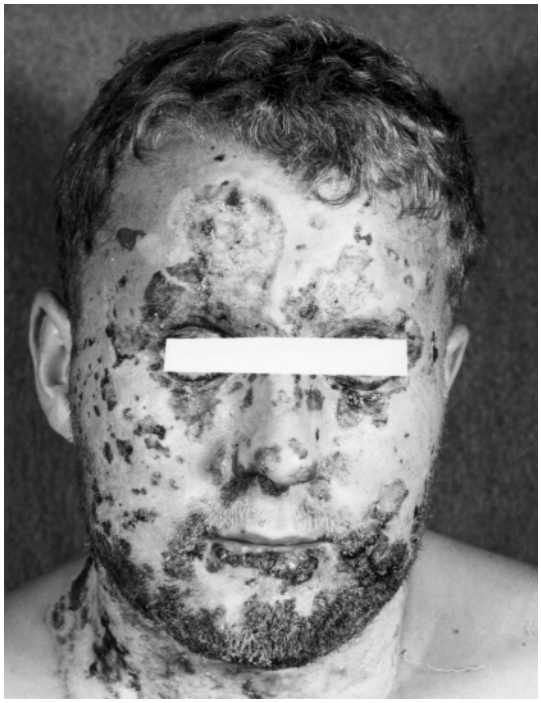


Figure 9-1. Casualty with white phosphorus burns.

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 (napalm). 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 these 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 irritative laryngospasm or laryngeal or glottic edema. Survivors may have thermal burns of the skin and upper respiratory tract and central 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 wet wool blanket is excellent. The protective mask may give partial protection against smoke but is NOT protective 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. Intubation, 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 container containing 100 or more gallons of thickened gasoline (such as napalm) 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 four to six 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 laryngospasm or 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) *First Aid.* Rapidly remove burning clothing and brush off burning fuel with a gloved hand or with several layers of other material.

(3) *Treatment.* In general, treatment is similar to that used after flame weapon attacks.

d. Replacement of Body Fluids. In severe burns, lost body fluid must be replaced quickly to prevent shock.

(1) *Intravenous Replacement*. The preferred method of replacing body fluids is the rapid administration of IV fluids. If liquid contamination is present, spot decontaminate the protective jacket at the site to be used for the IV. 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.

(2) *Oral Replacement.* An alternate method of body fluid replacement in conscious casualties is by oral replacement. In a contaminated atmosphere, fluids that are being replaced orally must be administered to the casualty without disrupting their MOPP. Oral fluid replacement may be accomplished by using the protective mask drinking tube and observing the following procedures:

(a) Do not remove the casualty's protective clothing or mask.

(b) If the casualty'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.

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

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

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

(f) Gradually give the water to the casualty a few sips every few minutes. If the casualty does not become nauseated, gradually increase the fluid intake. At the first sign of nausea, stop giving the water until the nausea subsides.

e. Arrange for the evacuation of the casualty to an uncontaminated area as rapidly as possible.

CHAPTER 10

TOXIC INDUSTRIAL CHEMICALS

10-1. General

a. Toxic industrial materials (TIMs) are substances, including chemical, biological, or radiological materials, that in sufficient quantifies may cause harm to individuals, material resources, or the environment. A subset of TIMs, toxic industrial chemicals (TICs) may be encountered during any military operation. Effects from contact with these compounds in liquid form or through inhalation of their gases, vapors, or aerosols (including smoke) can be fatal, particularly in confined or poorly ventilated spaces, if correct personal protective equipment, including respiratory protection, is not immediately available. Many of these gases and vapors are released as thermal decomposition products (pyrolysis products) of chemical elements present in a wide variety of materials. Personnel are at increased risk when operating around manufacturing, storage, and major transportation (truck terminals and rail heads) facilities. Releases may be by accidental release or by enemy forces, terrorists, or belligerents.

b. The most widely encountered TICs are ammonia (NH_3), carbon monoxide (CO), chlorine gas, hydrogen sulfide, and oxides of nitrogen (NO_x).

c. Protection.

(1) The field protective mask and collective protection systems are of reduced value against many TICs. Self-contained breathing apparatus (SCBA) or supplied air respirators protect the respiratory tract against most TICs and provide an additional protection against low oxygen tensions in the ambient environment due to displacement of air by some TICs, especially in enclosed spaces. Depending on the TIC, specialized clothing may also be required, up to the level of fully encapsulating suits.

(2) The filter element/canister of the field protective mask provides only limited protection against smoke. Duration of the protection depends upon the type of smoke and its concentration. The filter element/canister does not generate oxygen but filters smoke and some 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.

NOTE

Always replace the filter element/canister after wearing the protective mask in a heavy concentration of oil fire smoke because the oil clogs the filter

10-2. Acids

a. Properties. Acids may be encountered as solids, liquids, gases, or aerosols. Liquids may also evaporate to form vapor. Vapors (commonly but incorrectly called “fumes”) and gases usually have a characteristic pungent odor. The most common acids are hydrochloric, nitric (HNO_3), and sulfuric (H_2SO_4).

b. Relevance to Military Operations. Acids are found in a variety of industrial settings in bulk quantity. They may be accidentally released as the result of combat fire, or intentionally release during enemy retrograde operations in order to retard force advancement of adversaries. Acids, like all TICs, pose the greatest threat in enclosed spaces or close quarters operations, such as urban combat.

c. Pathology. Acids are toxic to the skin, eyes, and mucous membranes. Severe burns are usually the result of direct contact with the acid. Inhalation of concentrated vapors may be fatal within minutes. To the extent to which acids are soluble in aqueous media and chemically reactive, they exert central pulmonary effects: release of hydrogen ions in moist tissue in the central airways leads to necrosis and denudation of respiratory epithelium. As dose (Ct product) increases, however, peripheral pulmonary effects (pulmonary edema) may also be seen.

d. Symptoms. Signs and symptoms may include severe burns with pain; destruction of the cornea and can result in blindness; turbulence-induced respiratory noise (coughing, sneezing, hoarseness, wheezing, stridor) in the upper (central) airways; shortness of breath (dyspnea), chest pain, and pulmonary edema; dizziness, shock, convulsions, and coma; and weak and rapid pulse with resultant circulatory collapse.

e. Diagnosis. Diagnosis will initially be empiric, based on signs and symptoms, which will primarily be related to the respiratory tract and vision. Individuals who experience symptoms listed in 10-2*d* should be presumed to have been exposed to a caustic vapor or gas, which would include acids in the differential diagnosis. Since the emergency treatment of these exposures is the same, exact agent diagnosis at that time is not required. Evidence of large, ruptured, or leaking containers in an industrial setting is the single environmental clue of potential acid exposure. The single agent it is important initially to rule out is nerve agent exposure, easily differentiated by the papillary changes, sweating, muscular fasciculations, and mental status changes.

f. Protection. Rescuers must determine the TIC concentration level and assume the appropriate respiratory and skin protective level before attempting to rescue or care for casualties in the contaminated area. Self-contained breathing apparatus and chemical resistant outer clothing (Occupational Safety and Health Administration [OSHA] Level A) afford the greatest protection and should be worn if the substance or concentrations are unknown, especially in a confined space.

g. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Remove casualty from contamination zone and decontaminate.
- Administer oxygen using a face mask.
- Start an IV or saline lock.
- Administer one or two glasses of water in cases of ingestion if casualty is conscious.
- Monitor and treat for shock, as necessary.
- Loosely cover burns with sterile gauze.
- Evacuate casualty.

(2) *Medical treatment facility care.*

- Maintain airway and be prepared for possible early intubation.
- Continue oxygen therapy with warm, humidified air.
- Use appropriate postural drainage and percussion to assist in removal of tissue debris from airways.
- Perform bronchoscopy as indicated to identify and remove pseudomembranes.
- Administer beta agonists to manage bronchospasm.
- Be alert for secondary pneumonitis, and treat with antibiotics once a causative organism has been identified.
- If estimated inhaled dose of acid is high, maintain patient at enforced bed rest (semiseated if tolerated by patient).
- Observe for and manage pulmonary edema.
- Manage circulatory collapse, if needed.
- Treat burns by applying a topical antimicrobial cream to cleansed burn wound. Use silver sulfadiazine and/or mafenide acetate burn creams.
- Treat eye injuries.

h. Prognosis. Long-term prognosis of individuals exposed to acid vapors is excellent. Prolonged exposure may, however, lead to pulmonary compromise, secondary infections, noncardiogenic pulmonary edema, and permanent functional pulmonary impairment due to scarring.

10-3. Ammonia

a. Properties. Ammonia is a pungent, suffocating, and colorless gaseous alkaline compound of nitrogen and hydrogen. The boiling point is -27° F (-32.8° C), but its vapor is heavier than air and may remain close to the ground for some time and inside structures for hours to days. Ammonia is readily soluble in water and forms a corrosive, alkaline liquid. It is used as a refrigerant, a fertilizer, as a cleaning and bleaching agent, and as a household cleaner. It is also used in a variety of manufacturing applications. Liquid NH_3 is a vesicant.

b. Relevance to Military Operations. Ammonia 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. Terrorists and belligerents may also release NH_3 from storage containers, transportation carriers, or large refrigeration systems.

c. Pathology. Exposure to high concentrations of NH_3 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. Damage to upper (central) airways may predominate at low to moderate doses and may be complicated by secondary bacterial bronchopneumonia; at higher concentrations, peripheral pulmonary damage (pulmonary edema) is also seen.

d. Symptoms. Low to moderate concentrations produce violent, burning pain in the eyes and nose, lacrimation, sneezing, pain in the chest, cough, and laryngeal spasm characteristic of central pulmonary damage. Often there is a temporary reflex cessation of respiration with spasm of the glottis. Edema of the glottis at a later period may interfere with breathing. Concentrations of 0.1 percent are intolerable to humans. Exposure to higher doses (Ct products) can lead to pulmonary edema.

e Diagnosis. Diagnosis will initially be empiric, based on signs and symptoms, which will primarily be related to the respiratory tract and vision. Individuals who experience symptoms listed in 10-2*d* should be presumed to have been exposed to a caustic vapor or gas, which would include acids in the differential diagnosis. Since the emergency treatment of these exposures is the same, exact agent diagnosis at that time is not required. Evidence of large, ruptured, or leaking containers in an industrial setting is the single environmental clue of potential acid exposure. The pungent odor of NH_3 is characteristic. The single agent it is important initially to rule out is nerve agent exposure, easily differentiated by the papillary changes, sweating, muscular fasciculations, and mental status changes.

f. Protection. Rescuers must determine the TIC concentration level and assume the appropriate respiratory and skin protective level before attempting to rescue or care for casualties in the contaminated area. Self-contained breathing apparatus and chemical resistant outer clothing (OSHA Level A) afford the greatest protection and should be worn if the substance or concentrations are unknown, especially in a confined space.

g. Treatment. Treatment consists of prompt removal from the contamination zone and administration of assisted ventilation. Later measures are directed toward the treatment of bronchitis, pneumonia, and pulmonary edema. In general, treatment is designed to address burns, airway compromise, and damage to the respiratory tract (see paragraph 10-2g).

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

10-4. Carbon Monoxide

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

b. Relevance to 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. Tissue hypoxia is caused chiefly by displacement of oxygen from binding sites on blood hemoglobin: carbon dioxide has an affinity for these sites that is 200 times that of oxygen, and it forms carboxyhemoglobin (COHb), a cherry-red compound that does not carry oxygen. The CNS is the most sensitive organ system to low oxygen availability. Postmortem examinations reveal little beyond the characteristic cherry-red color of the blood and hemorrhages in the brain.

d. Symptoms. Carbon monoxide is insidious in its actions, 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, weak pulse, and death.

e. Diagnosis. The diagnosis is made from the circumstances of exposure and the appearance of cherry-red skin and mucous membranes. Exposure to hydrogen cyanide (AC) may occasionally produce flushed skin, but from persistence of oxygenated blood in capillaries and veins rather than from

the presence of a colored compound. Co-oximetry in cases of CO poisoning will demonstrate increased COHb. Both cyanide and CO poisoning will produce lactic acidosis.

f. Protection. Adequate ventilation should be provided for all enclosed spaces where CO may be produced. Air safety in enclosed spaces for people to breathe may be tested by using 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. Relocate the victim to open air. If respirations are weak or absent, begin assisted ventilation at once. Administer oxygen using a face mask, preferably under pressure (up to 3 atmospheres) if available. Keep the patient warm and at rest. Sedation may cloud clinical assessment of mental status and should be avoided unless needed for severe agitation, which is uncommon in CO poisoning. After resuscitation, initial supportive measures (such as the need for parenteral fluids and pressor drugs) can best be decided by the medical officer. Hyperbaric oxygen has been shown to be efficacious, but its use in field operations is prohibitive. Methylene blue solution, morphine, and atropine are not indicated (TB MED 269).

h. Prognosis. The chance for recovery lessens as the period of the coma lengthens. Most mildly exposed individuals recover with early treatment. Tachycardia and dyspnea may continue for months. There may be chronic CNS disturbances.

10-5. Chlorine

a. Properties. Chlorine is a pungent, irritating clear to amber-colored liquid or green-yellow gas with a boiling point of -29° F (-33.9° C). It is a strong nonflammable oxidant that will readily evaporate in open air but that can remain in closed unventilated spaces for extended periods. Chlorine is moderately soluble in water to produce hypochlorous and hydrochloric acids; it reacts with NH_3 to form toxic chloramines.

b. Relevance to Military Operations. Weaponized for use during World War I, chlorine is an industrial chemical ubiquitous in modern society. It is therefore easily available for sabotage or terrorist use. Accidents involving chlorine, particularly in use in water purification, occasionally occur.

c. Pathology. Chlorine is an irritant and blistering agent. Because it is intermediate in both aqueous solubility and chemical reactivity, it exhibits both central pulmonary effects and also peripheral pulmonary effects (see paragraph 2-1) in approximately equal measure. Hydrochloric acid is formed when chlorine contacts moist tissue, and this acid is responsible for most of the irritation of and damage to the conducting (central) airways. Hypochlorous acid in the peripheral airways becomes a source of oxygen free radicals that damage endothelial cells in pulmonary capillaries and lead to transudation of fluid into alveolar septa and eventually into alveoli and airways (pulmonary edema).

d. Symptoms. Exposure to liquid chlorine can cause intense local pain with skin blistering and tissue necrosis; chlorine gas irritates the eyes, the skin, and mucous membranes and leads to the noise (coughing, sneezing, hoarseness, inspiratory stridor, and wheezing [bronchospasm]) indicative of damage to the central airways. A suffocating feeling may be experienced along with nausea and vomiting. Dyspnea after a latent period indicates peripheral damage to the respiratory tract and may progress to frank pulmonary edema with shock, circulatory collapse, and death.

e. Diagnosis. The odor of chlorine is characteristic. Unless intentionally weaponized, environmental clues will be similar as that for industrial acids. Diagnosis is made empirically, at least initially, based on individuals with symptoms as listed in 10-5*d*.

f. Protection. Rescuers must determine the concentration level in the contaminated area and assume the appropriate protective level before attempting to rescue or care for casualties. Closed-system breathing apparatuses (for example, SCBA) and fully encapsulated chemically protective suits should be worn when entering a contaminated confined space. The MOPP level 4 will usually be adequate in open-air contaminated areas.

g. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Mask casualty and remove from contamination zone as soon as possible.
- Decontaminate casualty with soap and water.
- Flush eyes with normal saline or water.
- Administer oxygen, as needed.
- Start an IV or saline lock.
- Monitor and treat for shock, if necessary.
- Evacuate casualty.

(2) *Medical treatment facility care.*

- Manage airway.
- Administer nebulized beta agonist as needed for bronchoconstriction and bronchospasm.
- Administer humidified oxygen, as needed.
- Enforce bed rest during observation.
- Observe for and manage pulmonary edema.
- Manage circulatory collapse, if required.
- Treat eye injuries.

h. Prognosis. Individuals with a mild or short-term exposure have excellent prognoses. Of over 21,000 cases reported to the American Association of Poison Controls Centers' National Data Collection System, 40 resulted in a major effect, 2,091 resulted in a moderate effect, 17,024 resulted in a minor effect, and 2,099 had no effect. Three fatalities occurred. Minor effects quickly resolve. Moderate effects may have a systemic nature and usually require some form of treatment. Major effects include signs or symptoms that are life-threatening or result in significant residual disability or disfigurement.

10-6. Ethylene Oxide

a. Properties. Ethylene oxide is a colorless gas at room temperature that becomes a liquid at temperatures below 54° F (12.2° C). It has an ether-like odor. The boiling point is 51° F (10.6° C), and its freezing point is -168° F (-11.11° C). The immediate danger to life is at 800 ppm and the lethal concentration for 50 percent of those exposed (LC_{50}) is 4350 ppm. The vapors are flammable and explosive.

b. Relevance to Military Operations. Ethylene oxide is used to sterilize surgical instruments, as an agricultural fungicide, to fumigate food items and textiles, and in organic synthesis.

c. Pathology. Ethylene oxide may injure the skin, mucous membranes, and eyes. The liquid may be absorbed via the skin or the eyes. Vapor and gas may injure the eyes and, through inhalation, the respiratory tract, in which both central and peripheral pulmonary damage may occur. Prolonged exposure to low concentrations has also been associated with peripheral polyneuropathy, teratogenicity, spontaneous abortions, and leukemia.

d. Symptoms. Symptoms may include red and inflamed eyes, skin (both chemical burns and frostbite from contact with refrigerated liquid may occur), and mucous membranes; a distinctive odd taste; coughing; and substernal pain. Shortness of breath (dyspnea) is a harbinger of developing pulmonary edema. Abdominal pain, nausea, and vomiting may also be seen, as may mental changes indicative of encephalopathy.

e. Diagnosis. Diagnosis will initially be empiric and based on clinical and environmental findings. Diagnosis that the individual has been exposed to some toxic substance (without differentiating) may be the best available at the time, and sufficient for initial emergency treatment.

f. Protection. Rescuers must determine the concentration level in the contaminated area and assume the appropriate protective level before attempting to rescue or care for casualties. Wear at least OSHA Level C (a respirator with face shield or goggles and chemical resistant outer clothing, boots, and gloves). Since ethylene oxide can reasonably be considered to be a carcinogen, higher levels of protection should be assumed when practical.

g. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Remove casualty from the contamination zone and decontaminate.
- Clear airway as indicated.
- Administer oxygen, as needed
- Start IV or saline lock.
- Administer beta agonist to manage bronchospasm.
- Administer one or two glasses of water in cases of ingestion if casualty is conscious.

(2) *Medical treatment facility care.*

- Continue IV and oxygen therapy, as needed.

- Administer 30 to 100 grams of activated charcoal as a suspension in 1 cup of water (12.5 to 25 grams for children), if ingestion has occurred.

- Administer a cathartic such as magnesium sulfate following the activated charcoal. Give 10 to 15 grams in a glass of water (5 to 10 grams for children).

- Irrigate the eyes, as needed.

h. Prognosis. Those with short-termed, acute exposure usually have a prompt resolution of symptoms after removal to an uncontaminated environment. Those with prolonged exposure may suffer irreversible central nervous system damage, including mental status changes, cognitive impairment, and cerebellar dysfunction. Deaths have occurred due to very high dose acute exposures, although displacement of oxygen with subsequent hypoxemia may be contributory.

10-7. Hydrogen Fluoride

a. Properties. Hydrogen fluoride is a colorless gas with a strong irritating odor. It has a boiling point of 68° F (20.0° C) and a freezing point of -118° F (-83.3° C). It damages glass, ceramics, concrete, and alkali materials and will produce hydrogen gas when it comes in contact with metals. Exposure to 50 ppm for 30 to 60 minutes may be fatal.

b Relevance to Military Operations. Hydrogen fluoride is extensively used industrially and is widely available.

c. Pathology. Hydrogen fluoride affects the eyes, skin, nose, and throat. Direct contact may result in severe burns. Inhalation of concentrated vapor may be fatal by reason of both central and peripheral damage to the airways. Bone-density changes may be seen after prolonged exposure.

d. S*ymptoms.* The symptoms include red inflamed skin and eyes; severe skin burns, corneal injury and in some cases blindness; abdominal pain, nausea, and vomiting; upper-airway irritation and damage with concomitant noise (cough, sneezing, hoarseness, wheezing, stridor); shortness of breath (dyspnea) and pulmonary edema; weak and rapid pulse; dizziness; shock; convulsions; coma; circulatory collapse; and death. Pain is often not present in hydrogen fluoride burns that are severe enough to cause extensive tissue damage and systemic effects.

e. Diagnosis. Diagnosis is initially empiric and similar to that for acid or chlorine exposure.

f. Protection. OSHA Level A protects against exposure.

g. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Ensure casualty has been removed from hazard area and decontaminated.

- Administer oxygen, as needed.

- Administer one or two glasses of water in cases of ingestion if casualty is conscious.

- Monitor casualty for shock and treat if necessary.

- Loosely cover burns with sterile gauze.
- Evacuate casualty to supporting MTF.

(2) *Medical treatment facility care.*

- Treat burns.
- Manage airway.
- Administer bronchodilators for bronchospasm.
- Observe for and manage pulmonary edema.
- Manage circulatory collapse, if required.
- Treat eye injuries.

h. Prognosis. Prognosis is similar to that for individuals exposed to acids or chlorine and will be dose and time exposure dependent.

10-8. Hydrogen Sulfide

a. 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. It has a boiling point of -77° F (-60.6° C) and a freezing point is -122° F (85.6° C). It is incompatible with metals, acids, and strong oxidizing materials. Severe health effects occur at air concentrations of 70 ppm. Olfactory fatigue occurs at 100 ppm.

b. Relevance to Military Operations. Hydrogen sulfide is used industrially and may also be generated from bacterial action in the environment.

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 30 minutes to 1 hour. Higher concentrations (0.75 mg per liter or greater) are rapidly fatal as the result of inhibition of cytochrome oxidase in the mitochondria of cells. This mechanism is identical to that of AC (see paragraph 4-4*a*). All cells are affected, but nerve tissue is more sensitive than muscle, and the mechanism of death is central apnea from failure of the respiratory center in the medulla.

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 as in inhalation of AC. Any discolored copper coins in close proximity to exposure (for example, on the person of the casualty) should lead to a high suspicion of poisoning with hydrogen sulfide.

e. Diagnosis. Diagnosis is initially empiric and similar to that for acid or chlorine exposure.

f. Protection. Rescuers should wear OSHA Level C protection. Medical personnel caring for contaminated casualties should be at the same protective posture.

g. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Remove casualty from contamination zone and decontaminate with soap and water.
- Administer oxygen, as needed.
- Administer CANA or other forms of diazepam to control seizures.
- Start an IV or saline lock.
- Flush eyes with normal saline or water to relieve pain.

(2) Medical treatment facility care.

(a) Intravenously inject 300 mg of sodium nitrite over a period of three minutes. Hydrogen sulfide acts at the same site (at cytochrome oxidase within mitochondria) as does AC and can be removed from the enzyme by the same nitrite antidotal treatment that forms the first step in the treatment of cyanide poisoning. The sodium nitrite is given to produce methemoglobin, thus sequestering the sulfide on the methemoglobin. The use of sodium thiosulfate in cases of poisoning with hydrogen sulfide has not yet been demonstrated to be of benefit.

CAUTION

Administer sodium nitrite ONLY intravenously. Intramuscular administration will cause severe tissue necrosis.

(b) The decrease in blood pressure following sodium nitrite injections is usually not clinically significant 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.

- Maintain airway and ventilate, as necessary.
- Manage central airway effects as clinically indicated (such as with a bronchodilator to treat bronchospasm).
- Manage peripheral pulmonary damage (pulmonary edema), as indicated (see Chapter 2).
- Continue oxygen and IV therapy, as needed.
- Administer diazepam for seizures, as needed.
- Treat eye injuries.

h. Prognosis. Prognosis is similar to that for individuals exposed to acids or chlorine and will be dose and time exposure dependent. Those progressing to cardiovascular collapse or seizures have an especially grave immediate and long-term prognosis, and long-term disability among survivors is common. Asymptomatic patients who have no evidence of pulmonary edema or CNS or respiratory compromise and no signs of eye irritation may be discharged after four to six hours of observation. Prolonged exposure has been reported to cause low blood pressure, headache, nausea, loss of appetite, weight loss, ataxia, eye-membrane inflammation, and chronic cough. Neurologic symptoms, including psychological disorders, have been associated with chronic exposure.

10-9. Oxides of Nitrogen

a. Properties. Oxides of nitrogen include nitric oxide (NO), nitrous oxide (N_2O), and nitrogen dioxide (NO_2). The term "oxides of nitrogen" is also used for mixtures containing two or more of these compounds. Nitric oxide and N_2O are colorless gases; the other oxides are red-brown gases. Their boiling points are -241° F (-151.7° C) (NO), -127° F (-88.3° C) (N_2O), and 70° F (21.1° C) (NO_2). The term "nitrogen tetroxide" refers to an equilibrium mixture of the liquid forms (under pressure) of NO_2 and dinitrogen tetroxide (N_2O_4).

b. Relevance to Military Operations.

(1) Oxides of nitrogen are a component of photochemical smog and may also be seen in silos in agricultural settings, but these compounds are typically released in the military environment from burning munitions or munitions fire from weapons. The danger of poisoning is great if high explosives (such as smokeless powder or cordite) are burned or detonated in poorly ventilated areas. This may occur in gun pits, armored vehicles, ship magazines, and turrets as well as in mining and tunneling operations.

(2) Oxides of nitrogen as vapor or gas are emitted from fuming nitric acids (white and red) and are generated by the combustion of some plastics.

c. Pathology. Inhalation of NO causes the formation of methemoglobin and does not appear to lead to any tissue lesions. Inhalation of NO_2 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 NO_2 (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. The pathology of most military exposures relates in an acute setting to damage to pulmonary capillary endothelium and the eventual appearance of pulmonary edema, and in a longer-term setting to late-onset immunologically mediated cryptogenic organizing pneumonia with pulmonary fibrosis.

d. Symptoms. The symptoms following inhalation of vapor and gas from fuming nitric acids are due chiefly to NO_2. The symptoms presented depend upon the concentration of the gas. Exposures to low to moderate concentrations may not be irritating and may not be recognized by the victim. Exposures to higher concentrations cause central pulmonary effects (severe local irritation with choking and burning in the chest, violent coughing, yellow staining of the mucous membranes, and expectoration of yellow-colored sputum) in addition to the inevitable peripheral pulmonary damage and may also produce headache and vomiting. Even in cases in which central effects are seen, these effects usually resolve; a clinically asymptomatic latent period (shorter with higher doses and also shortened by exertion) then ensues, lasting 2 to 24 hours. Incipient pulmonary edema (peripheral damage) is then heralded by the often sudden onset of severe dyspnea, coughing, and production of copious quantities of sputum (often frothy). Nausea and vomiting are also common. Cyanosis, convulsions, and death may follow. 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. Some patients who develop pulmonary edema appear to recover completely, but dyspnea and cough, often with fever, chills, and cyanosis, may develop two to six weeks after the initial exposure. Crackles are present; chest radiography may demonstrate fluffy infiltrates consistent with pulmonary edema or cryptogenic organizing pneumonia. Respiratory failure and death may sometimes follow.

e. Diagnosis. In an acute setting, diagnosis is based on characteristic signs and symptoms, coupled with an index of suspicion based on environmental setting. The differential diagnosis includes inhalation of other TICs, including hydrogen sulfide, CO, and organophosphates. Any environmental toxin that may produce acute pulmonary symptoms may be included.

f. Protection. Positive-pressure, self-contained breathing apparatus is recommended in response situations that involve exposure to potentially unsafe levels of nitrogen oxides. Chemical-protective clothing is recommended when repeated or prolonged contact with liquids of NO_x or with high concentrations of NO_x vapors is anticipated because skin irritation or burns may occur.

g. Treatment. Treatment of casualties is the same as the treatment for victims exposed to HC smoke (see paragraph 8-4 and paragraph 2-9*e*). The use of steroids has not been proven to be beneficial in cases of noncardiogenic pulmonary edema induced by CG or most of the other peripheral pulmonary agents. Nevertheless, their use in cases of poisoning by NO_x or HC smoke should be encouraged since these agents appear to be able to induce late-onset pulmonary fibrosis by immunological means.

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

10-10. Inorganic Phosphorus Compounds

a. Properties. Inorganic phosphorus compounds exist as solids, liquids, vapors, gases, or aerosols. Many are highly flammable. They may react with water. The boiling and freezing points are dependent upon the formulation of the compound; phosphorous trichloride has a boiling point of 168° F (75.6° C) and a freezing point of -182° F (-118.9° C).

b. Relevance to Military Operations. They are used in chemical manufacturing and synthesis, for metal cleaning, and safety-match manufacturing.

c. Pathology. Inorganic phosphorus compounds are tissue irritants. Severe effects may be delayed up to 24 hours after exposure. Acute exposure can affect calcium metabolism and damage the liver and kidneys.

d. Symptoms. Exposure may be by ingestion, inhalation, or skin contact. Common effects include red and inflamed eyes, skin, and mucous membranes; intense tearing, salivation, blurred vision, and conjunctivitis; blindness; tissue damage and pain; abdominal cramps, nausea, vomiting, and diarrhea; hoarseness and both central and peripheral effects; and difficulty swallowing and speaking.

e. Diagnosis. Diagnosis in the acute setting is empiric. Patients resemble those exposed to other caustic liquids or vapors. There are no specific findings to narrow the differential diagnosis in these patients.

f. Protection. Rescuers must determine the concentration level in the contaminated area and assume the appropriate protective level before attempting to rescue or care for casualties. Wear OSHA Level A protection to enter a contaminated confined space/area. The MOPP level 4 will usually be adequate in open-air contaminated areas.

g. Medical Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Remove casualty from contamination zone and decontaminate.
- Administer oxygen using a non-rebreather mask.
- Administer one or two glasses of water in cases of ingestion if casualty is conscious.
- Monitor for shock and treat, if necessary.
- Evacuate casualty to supporting MTF.

(2) *Medical treatment facility care.*

- Manage airway and administer oxygen.
- Administer beta agonist if bronchospasm occurs.
- Monitor for and manage pulmonary edema.
- Manage shock.
- Irrigate eyes if irritation continues.
- Administer cathartic if ingestion is route of entry.

h. Prognosis. Although most survivors of acute exposure show no permanent disabilities, damage due to insufficient blood supply to the heart and brain has been reported. Subacute poisoning resulting from exposure for a few days may cause reactive airways dysfunction syndrome months later.

10-11. Organophosphorus Compounds

a. Properties. The organophosphorus compounds (often incorrectly called "organophosphate" compounds) are solids or liquids used as pesticides. Some formulations are highly flammable. Their physical properties vary with the specific manufacturing process. Although most are persistent, the length of persistence in the environment depends upon many factors, including the strength of the pesticide, temperature, and humidity; toxic quantities may last from days to months in soil and other absorbing materials.

b. Relevance to Military Operations. These compounds are widely used as pesticides in military, civilian, and public health settings. Common members of this class include diazinon, malathion, parathion, dichlorvos, and chlorpyrifos.

c. Pathology. The effects are qualitatively the same as for nerve agents. The toxic effects occur following ingestion, skin contact, or inhalation. Agriculture-grade compounds are the most toxic; the least toxic are ready-mix household formulations. Toxic effects will gradually increase, peaking within a few hours of exposure; paralysis occurs in some exposures. These compounds have greater lipid solubility than nerve agents; therefore, the clinical effects they produce may last longer.

d. Symptoms. Symptoms are the same as for nerve agent poisoning (see paragraph 3-4*a*).

e. Diagnosis. Diagnosis is similar to that for nerve agent intoxication. Exposure to vapors will produce miosis and pulmonary symptoms, which are dose dependent, followed by mental confusion, obtundation, seizures, flaccid paralysis, and death. Skin exposure will produce similar systemic symptoms, with fasciculations and sweating locally, but without significant visual impairment. Skin absorption will produce delayed and more gradual onset of symptoms than inhalation exposure.

f. Protection. Rescuers must determine the concentration level in the contaminated area and assume the appropriate protective level before attempting to rescue or care for casualties. Wear OSHA Levels A or B protection, depending upon concentration. The MOPP level 4 will usually be adequate in open-air contaminated areas.

g. First Aid.

(1) *Self-aid.* Put on protective mask and move out of the hazard area. Remove contaminated clothing and decontaminate skin with soap and water. Time is critical in removing the contamination from the skin; delayed decontamination will result in increase toxicity. Administer one MARK I or one ATNAA. Seek buddy aid or medical assistance if symptoms persist.

(2) *Buddy aid.* Mask casualty and move from the hazard area. Decontaminate skin with soap and water. Administer the MARK I autoinjectors (up to a total of three MARK I kits) or the ATNAA and CANA as for nerve agent, if required. Request medical assistance.

h. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Mask casualty, if needed.
- Remove casualty from contamination zone and decontaminate.
- Administer MARK I kits (up to a total of three MARK I kits), ATNAA, or additional atropine as needed to reduce secretions and to reduce airway resistance.
- Administer additional diazepam to manage convulsions.
- Administer oxygen, if available.
- Evacuate casualty.

(2) *Medical treatment facility care.*

- Administer additional atropine as needed to reduce secretions and to reduce airway resistance.

NOTE

The total amount of atropine required for victims exposed to organophosphorus pesticides will probably exceed the typical 20

mg or less required for nerve agent casualties and may reach a total of up to 1 to 2 grams over days.

- Consider additional oxime (2-PAM Cl) as clinically indicated.
- Administer additional CANA or other forms of diazepam to manage convulsions.
- Administer oxygen.
- Manage shock, as needed.

i. Prognosis. Complete recovery generally occurs within 10 days unless severe lack of oxygen has caused residual brain damage. Central nervous system effects such as confusion, fatigue, irritability, nervousness, and impairment of memory can occasionally last for several weeks. Six to 21 days after acute exposure to some organophosphate compounds, onset of nerve disorders of mixed sensory-motor type may occur; peripheral nerve recovery may never be complete.

10-12. Sulfur Dioxide

a. Properties. Sulfur dioxide is a colorless nonflammable gas with a strong suffocating odor. It has a boiling point of 14° F (-10.0° C) and freezes at -104° F (-75.6° C). It forms a corrosive acid when it reacts with water. Concentrations above 39 ppm can cause severe respiratory tract injury.

b. Relevance to Military Operations. Sulfur dioxide is a widely used and readily available industrial compound.

c. Pathology. Sulfur dioxide is injurious to the eyes and to the respiratory tract, where it acts primarily as a central pulmonary toxicant at low to moderate doses, but may also exhibit peripheral effects (pulmonary edema) at high doses.

d. Symptoms. Symptoms include eye irritation, headache, irritation to mucous membranes and to upper (central) airways (with concomitant coughing, sneezing, hoarseness, wheezing, stridor, or laryngospasm), dyspnea (shortness of breath, chest tightness) indicative of incipient pulmonary edema, shock, circulatory collapse, seizures, and coma.

e. Diagnosis. Diagnosis in the acute setting is usually empiric and includes other TICs or chemical agents that produce eye irritation and acute pulmonary symptoms.

f. Protection. Rescuers should wear OSHA Levels A or B to enter the contaminated area. Medical personnel caring for contaminated casualties should be at the same protective posture.

g. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Mask casualty, remove casualty from the contaminated area, or both.
- Manage airway.
- Decontaminate casualty.
- Administer oxygen using a non-rebreather mask.

- Irrigate casualty's eyes with copious amounts of water or, preferably, sterile isotonic saline.

- Start an IV or saline lock

- Administer one or two glasses of water in cases of ingestion if casualty is conscious.

- Administer CANA or other forms of diazepam to control seizures.

(2) *Medical treatment facility care.*

- Maintain airway and administer warm, moist air.

- Continue supplemental oxygen and IV therapies, as needed.

- Manage central and peripheral pulmonary effects, as clinically indicated.

- Administer additional diazepam to manage seizures, as indicated.

- Administer 30 to 100 grams of activated charcoal suspension in one glass of water (12.5 to 25 grams for children) in cases of ingestion.

- Administer 10 to 15 grams of magnesium citrate in a glass of water (5 to 10 grams for children) in cases of ingestion.

h. Prognosis. High-level acute exposures have resulted in pulmonary fibrosis, chronic bronchitis, and chemical bronchopneumonia with bronchiolitis obliterans. Bronchospasm can be triggered in individuals who have underlying lung disease, especially those who have asthma and emphysema. Rarely, new onset airway hyperreactivity, known as reactive airways dysfunction syndrome, develops in patients without prior bronchospasm.

10-13. Hazards Caused by Fire

a. Properties. In fires, injury and/or death may be caused by blast, direct flame, anoxia, CO, heat, NO_x, cyanogens, other toxic fumes from burning chemicals and plastics, and smoke.

b. Relevance to Military Operations. Flame and smoke are frequent hazards in the military.

c. Pathology. Pathology specific to TIC exposure may be difficult to ascertain as a result of other injuries related to the fires. Further, depending on the materials consumed by flames, a variety of products of combustion, most of which have effects on primarily the eyes and the lungs, may be seen. With or without these secondary combustion materials, inhalation of smoke will cause an intense irritation of the central compartment of the lungs, with pathological evidence of extreme bronchorrhea and, if severe enough, peripheral compartment effects of noncardiogenic pulmonary edema.

d. Symptoms. In terms of respiratory damage, inhaled smoke particles act in the central airways to create burns by heat transfer to tissues. Inhaled vapors or gases may produce central effects, peripheral effects, or both. The presence of central-airway effects should always place the clinician on alert for possible early intubation, as should the presence of soot around the nose or mouth

e. Diagnosis. Diagnosis of exposure to smoke should be obvious. Depending on the history elicited, secondary effects, such as hypoxemia, carbon monoxide or cyanide inhalation, or exposure to other TICs should be considered during evaluation of the casualty.

f. Protection. A supplied air breathing device or SCBA must be used for respiratory protection.

g. Treatment.

(1) *Trauma specialist/hospital corpsman/Air Force medic (4N0 career field) care.*

- Remove casualty from contamination zone and decontaminate, if necessary.
- Administer oxygen using a non-rebreather mask, as required.
- Irrigate casualty's eyes with copious amounts of water, as needed.
- Start IV or saline lock.
- Administer CANA or other forms of diazepam to control seizures.
- Manage airway and prepare for early intubation.

(2) *Medical treatment facility care.*

- Manage airway aggressively
- Manage bronchospasm and pulmonary edema.
- Continue oxygen and IV therapies, as needed.
- Administer additional diazepam to manage seizures, if necessary.

h. Prognosis. Prognosis will be dependent on the duration and degree of exposure, concomitant hypoxemia, and specific products of combustion inhaled. Patients who survive acute exposure will likely survive the effects of smoke inhalation alone. Other injuries, however, such as extensive burns, or inhalation of these other products, may complicate treatment and worsen prognosis.

APPENDIX A

RECOGNITION OF A CHEMICAL CASUALTY

A-1. General

Medical units should rely on information not only from detectors and intelligence sources, but also from the casualties themselves. This principle applies particularly to agents (such as incapacitating agents) for which at present there is no satisfactory detector. Nerve agent signs and symptoms may range from mild (such as miosis, headache and tightness of the chest) to severe (such as convulsions and respiratory failure). The nature and timing of symptoms will vary with the state of the agent and the route of exposure. Although pulmonary agents are less likely to be employed, the possibility of their use must not be forgotten. The danger is that the latent, or clinically asymptomatic, period that 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 psychological 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 and the chain of command. 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

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

a. Conventional Casualties.

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

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

b. Direct Chemical Casualties.

(*1*) Chemical casualties with no other injury.

(*2*) 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) and pre-existing medical conditions. Whenever mixed casualties are encountered, the nature of the interactions, or synergism, of the co-existing diagnoses must be considered. For example, radiation casualties who are also exposed to sulfur mustard are at far greater risk than casualties exposed to just radiation or just sulfur mustard.

c. Indirect Chemical Casualties.

(1) Casualties suffering combat stress reactions (CSR) occur often in warfare, but may be more frequent where the CW threat exists. The service member will have the additional stress of claustrophobia or a sense of isolation from wearing the chemical protective ensemble, additional fatigue

when wearing the garments, and fear of chemical agents. The differential diagnosis between the CSR patients and chemical patients may sometimes be difficult. CSR patients could outnumber all others.

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

(3) 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). Both because of the possibility of heat injury and the psychological effects of wearing the protective ensemble, its use may degrade mission effectiveness.

A-3. Recognition of Chemical Casualties

a. 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 to ascertain the following:

- 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 effects and were those effects similar?
- Did the casualty notice any unusual smell?

b. 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 or dimness 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) The patient should also be questioned concerning a delay between the onset of symptoms and exposure or contamination.

- If so, how long was the delay?
- Did the effects of exposure persist after adjustment of the protective mask?
- Did the casualty use 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?

c. 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?
- What 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 CW agents inside MTFs, which may injure patients and medical personnel. Chemically contaminated clothing, blankets, and other equipment must be kept outside 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. 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 casualty decontamination area with a contaminated waste 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 should be at least 75 meters downwind from the MTF and living quarters. The dump should be clearly marked with standard chemical contamination markers (FM 4-02.7 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 the casualty, other patients, and medical personnel from contact with the liquid agent or from the vapor that accumulates in confined spaces.

c. The medical officer should notify designated 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. An informal inventory on the number of contaminated blankets sent to the contaminated waste dump is kept so that replacements requirements are known.

b. If the tactical situation permits, replacements are requisitioned through the normal medical supply channels. Emergency resupply may be requested from the nearest general supply support unit.

NOTE

In an emergency situation, if blanket replacement is not possible, blankets may be decontaminated and reused. Decontamination is accomplished by immersing in warm (100°F) soapy water (1 pounds of soap in 10 gallons of water) for one hour with light agitation or using a 5 percent sodium-carbonate solution for G agents.

B-4. The Chemical Protective Ensemble

a. All personnel handling or treating chemically contaminated casualties must be at MOPP 4 (paragraph 1-7*a*). Personnel must also be at MOPP 4 while decontaminating litters, ambulances, and other equipment.

b. The chemical protective overgarment is not removed until the danger of contamination has been eliminated. Contaminated chemical protective overgarmets/joint service lightweight integrated suite technology (JSLIST) may be worn safely in a contaminated environment for 24 hours. The uncontaminated suit may be worn for 45 days or as prescribed in FM 3-11.4/NTTP 3-11.27/MCWP 3-37.2/AFTTP(I) 3-2.46. FM 3-11.4/NTTP 3-11.27/ MCWP 3-37.2/,AFTTP(I) 3-2.46 gives further guidance on individual protection using the complete ensemble and FM 3-5/MCWP 3-37.3 contains the procedure to be followed in the MOPP gear exchange.

NOTE

Medical personnel who are required to wear the chemical protective ensemble will be severely restricted in their ability to treat casualties. Medical treatment may be limited to enhanced first aid in some situations. It is imperative that CPS or clean areas be located for the provision of medical care.

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 CW agent-tight containers to await later decontamination. If this is not possible, the items are discarded in the contaminated waste dump.

b. Shipboard Operations. For ships at sea, overboard dumping of hazardous waste is prohibited except under emergency conditions or if failure to discharge would endanger health and safety of shipboard personnel. If at all possible, contaminated suits should be double-bagged (with each bag a minimum of 3 mm thick) and stored in the weather for later transfer to a shore facility hazardous material (HAZMAT) team. For ships in port, double-bag contaminated suits for turn-in to shore-based disaster preparedness/HAZMAT teams for 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 the clothing is being worn. If a decontamination slurry is not available, blot liquid off with available absorbent material (such as rags). This should be done immediately if clothing is contaminated by splashes or large drops of CW 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 one hour. Do not stir or agitate. After one 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 (primarily worn by depot personnel) by spraying or applying a decontamination 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.

(*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 Skin Decontaminating Kit (SDK) (paragraph 1-10).

(*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 in water for one hour. 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 Decontamination Kit, Individual Equipment (DKIE). 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 four to six 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 one 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).* Use soap and water or available decontamination solution.

NOTE

If the above materials are not available, thoroughly soak the canvas litter with a 5 percent hypochlorite solution. Allow the litter to air dry.

(*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 hypochlorite solution to the entire surface of the litter and handles/poles. If the 5 percent hypochlorite solution is not available, remove gross contamination by scraping with a stick or other object, then use the M295 DKIE. Litters must be removed from the patient care area of the patient decontamination station for decontamination. Only a 0.5 percent concentration of hypochlorite solution is permitted in the patient care area.

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 CW 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 ICAM 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 ICAM 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 paragraph B-8*b*(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 suffering and loss of life and limb. 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.

a. At US Army Levels I and II (unit and division) including nondivisional units, the supported unit commander must provide eight nonmedical personnel to perform casualty decontamination. At Levels III and IV (corps and echelon above corps [EAC]) hospitals, a 20-man casualty decontamination augmentation team or 20 nonmedical personnel must be provided to perform casualty decontamination. The base cluster commander or units within the geographical area of the US Army hospital must provide these nonmedical personnel. Medical personnel supervise casualty decontamination operations to ensure that the casualty's condition is not compromised by the decontamination procedures. The final determination on the completeness of casualty decontamination rests with medical personnel. 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.

b. At USAF MTFs, casualty decontamination is performed by the USAF Wartime Medical Decontamination Team.

c. At US Navy MTF afloat, nonmedical personnel perform casualty decontamination procedures.

d. At MTFs supporting USMC units, casualty decontamination is performed by personnel as designated by the commander.

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

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

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

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

The initial management and treatment of casualties contaminated with a CW 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. Casualty decontamination sites are collocated with an MTF and should be positioned downwind (based on prevailing winds) from the adjacent MTF. This ensures medical supervision of casualty decontamination is available. Specifics on management of chemically contaminated casualties at the MTF are found in FM 4-02.7. Each Army MTF has identical medical equipment sets (MES) for chemical agent casualty decontamination and patient treatment. The numbers of each type of MES vary, depending on the level of care. 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 CW 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 CW agent poisoning.

c. For vesicant-contaminated casualties who have traumatic injuries or other illnesses, decontamination should be accomplished as soon as the situation permits. 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 CW 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 CW 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 life- or limb-threatening illnesses.

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

C-5. Casualty Decontamination Methods

a. Casualty decontamination serves two purposes: It prevents the casualty's system from

absorbing additional contaminants. It also protects medical personnel treating the casualty, other patients, and medical equipment and supplies, from contamination. Accumulated contamination in the MTF is a serious threat to medical personnel and patients. Accumulated contaminated material 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 casualty 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. Any casualty 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 downwind side of the MTF. The site is provided with overhead protection such as plastic sheeting, trailer covers, ponchos, or tarpaulins. Only those patients requiring immediate 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 CW agents during evacuation. There is only a limited supply of PPW; therefore, medical personnel must ensure they do not needlessly remove a patient's overgarment and clothing. Patients not requiring treatment at a forward MTF, but requiring evacuation to the next level MTF, must have their MOPP gear and equipment spot decontaminated and the integrity of their MOPP gear restored, such as by taping over tears or rips. Spot decontamination will remove gross contamination, reducing the hazard to the casualty and evacuation personnel.

d. Every person entering the decontamination area (including casualties) must be masked or have other respiratory tract protection in place. Most contaminants are removed by carefully removing all clothing. The casualty's protective mask is not removed. Remove the protective mask hood, overgarments, green or black vinyl overboots and boots, the uniform, and undergarments. For step-by-step procedures in performing casualty decontamination, refer to FM 4-02.7.

e. After patients have been decontaminated, exercise rigid control to prevent exposing their unprotected skin to a liquid CW agent. Skin exposure to a CW 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 may 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 those personnel proceed in groups of two or three to facilitate control. Ambulatory patients require constant observation and periodic assistance during the decontamination process. The trauma specialist/corpsman/Air Force medic 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 returned to duty or go to the evacuation point, as their medical conditions dictate. Ambulatory patients with injuries that do not require immediate attention but require treatment at a higher level MTF are evacuated in their MOPP ensemble. For example: A patient with a broken arm has a stabilizing splint on. This individual does not require treatment at the Level I or II MTF; however, his MOPP gear must be spot decontaminated to remove gross contamination before evacuation to the Level II or III 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 provided new MOPP ensemble or be placed in PPWs before they are moved to the evacuation point (paragraph C-5*e* 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 CW agent contaminated casualties. Personnel must be trained to protect themselves from CW 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 CW agent detection paper and the ICAM to monitor for and detect CW agents.
- Sorting and receiving contaminated casualties into a system designed for the treatment of both contaminated and noncontaminated casualties.
- Providing EMT.
- Performing casualty decontamination.
- Patient lifting and transfer techniques.
- Evacuating decontaminated casualties.
- Evacuating contaminated casualties.

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 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 4-02.7).

(1) Once treated, the patient is provided new MOPP ensemble or is placed in a PPW before movement to the evacuation pickup point. The PPW provides the same level of individual protection as does MOPP 4. 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 excessive heat build up. As stated earlier the use of the battery-operated blower will assist in reducing the heat load on the individual; however, care must be taken to prevent heat injury to the casualty. Place casualties in the PPW in a shaded area for maximum protection from heat injury. The protective mask must remain 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 US military aircraft or water craft, they should be decontaminated. Otherwise, the vapor from the CW 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. Applying the following measures can further minimize the hazards of the CW agent to other persons:

- 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 handled as contaminated. Decontaminate the litters before returning them to the inventory.

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 SKIN PROTECTION AND DECONTAMINATION PROCEDURES

Section I. SKIN PROTECTION

D-1. Use of Skin Exposure Reduction Paste Against Chemical Warfare Agents

a. The SERPACWA is a barrier cream for use by service members to protect against the toxic effects of CW agents (such as blister [vesicant] and nerve agents). The SERPACWA, when used in conjunction with MOPP gear, will prevent or significantly reduce the toxicity following cutaneous exposure to CW agents. The SERPACWA serves as an antipenetrant barrier to CW agent. The SERPACWA is not approved for use by Navy personnel.

b. The SERPACWA creates a physical barrier between the skin and the CW agent; only those areas of the skin having an intact layer of SERPACWA will be protected.

c. Individuals should use SERPACWA as an adjunct to MOPP, not as a substitute. Established doctrine for MOPP is followed if CW agent contamination is anticipated or suspected, even if the individual is wearing SERPACWA. Apply the SERPACWA before donning the MOPP.

d. All service members at risk in a potentially contaminated CW agent environment should use SERPACWA.

e. Each service member is issued six packets of SERPACWA (see Figures D-1 and D-2 for packet labels). This is sufficient material for six applications or for two days of use.

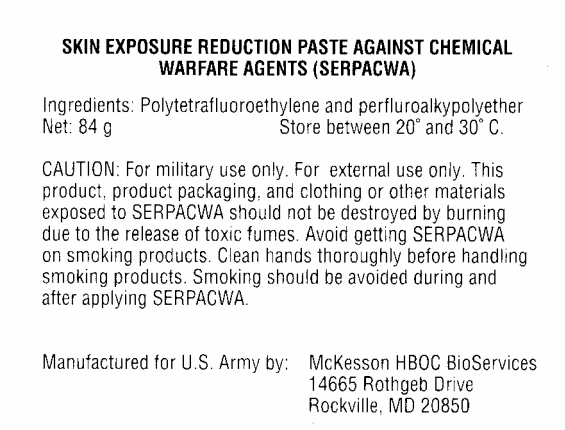
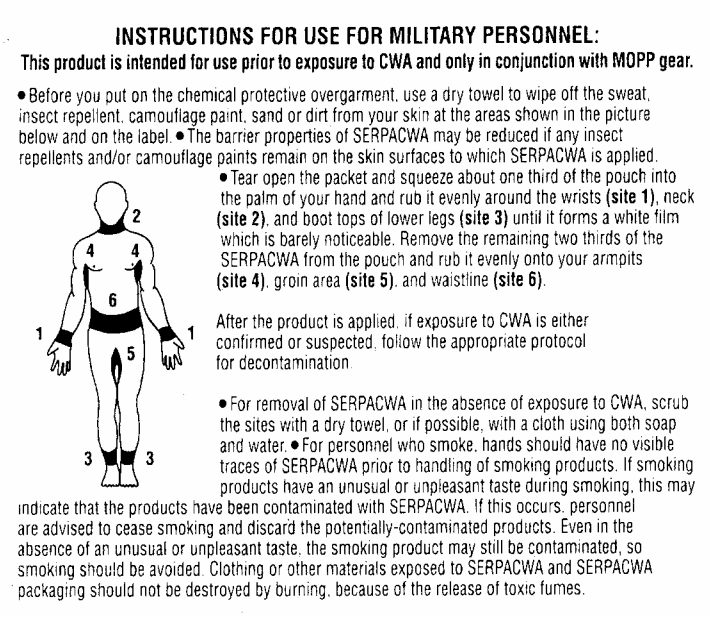


Figure D-1. Skin Exposure Reduction Paste Against Chemical Warfare Agent packet front label.



INSTRUCTIONS FOR USE FOR MILITARY PERSONNEL:

This product is intended for use prior to exposure to CWA and only in conjunction with MOPP gear.

• Before you put on the chemical protective overgarment, use a dry towel to wipe off the sweat, insect repellent, camouflage paint, sand or dirt from your skin at the areas shown in the picture below and on the label. • The barrier properties of SERPACWA may be reduced if any insect repellents and/or camouflage paints remain on the skin surfaces to which SERPACWA is applied.

• Tear open the packet and squeeze about one third of the pouch into the palm of your hand and rub it evenly around the wrists **(site 1)**, neck **(site 2)**, and boot tops of lower legs **(site 3)** until it forms a white film which is barely noticeable. Remove the remaining two thirds of the SERPACWA from the pouch and rub it evenly onto your armpits **(site 4)**, groin area **(site 5)**, and waistline **(site 6)**.

After the product is applied, if exposure to CWA is either confirmed or suspected, follow the appropriate protocol for decontamination.

• For removal of SERPACWA in the absence of exposure to CWA, scrub the sites with a dry towel, or if possible, with a cloth using both soap and water. • For personnel who smoke, hands should have no visible traces of SERPACWA prior to handling of smoking products. If smoking products have an unusual or unpleasant taste during smoking, this may indicate that the products have been contaminated with SERPACWA. If this occurs, personnel are advised to cease smoking and discard the potentially-contaminated products. Even in the absence of an unusual or unpleasant taste, the smoking product may still be contaminated, so smoking should be avoided. Clothing or other materials exposed to SERPACWA and SERPACWA packaging should not be destroyed by burning, because of the release of toxic fumes.

Figure D-2. Skin Exposure Reduction Paste Against Chemical Warfare Agent packet back label.

f. The commander will decide whether to begin, continue, or discontinue SERPACWA use based on the threat. The intelligence officer, the chemical officer, and the surgeon act as advisors to the commander in making the decision if a CW agent threat exists (for example, the enemy having vesicants or nerve agents in the combat zone and the probability of their use).

D-2. Application of Skin Exposure Reduction Paste Against Chemical Warfare Agent

The effectiveness of SERPACWA is dependent on the thickness and integrity of the SERPACWA layer and the length of time between application and agent exposure (wear time).

a. Skin Surface Coverage. When applying SERPACWA to the skin, first priority should be given to covering those areas adjacent to the closure or the battle dress overgarment (BDO); the neck, wrists, and lower legs (areas around the top of the boots). Additional SERPACWA may be applied to the armpits, groin area, creases and crack of the buttocks, and around the waist. Do not apply SERPACWA to open wounds or remove bandages to apply SERPACWA to these areas.

b. Thickness of Skin Exposure Reduction Paste Against Chemical Warfare Agent. Under normal conditions, SERPACWA is effective when spread over the skin as a thin layer (0.1 mm thickness or 0.01 ml/square cm). One packet of SERPACWA contains 1.35 fluid ounces (about 2.7 weight ounces or 84 g) for one application. A third of the packet should cover the skin areas of neck, wrists, and lower legs (at boot tops). This amount of SERPACWA will produce a smooth coating on the skin which is a barely visible cream color and detectable by touch. The rest of the packet of SERPACWA may be applied to the armpits, groin area, creases and crack of buttocks, and around the waist. Refer to Figure D-2 for application areas.

c. Wear Time. The SERPACWA, which is not water soluble, cannot be washed off by water or removed by sweat without brushing and scrubbing, but it may physically wear off with time. Abrasion of SERPACWA by clothing or other contacts, such as sand or dirt, will reduce the wear time. The SERPACWA needs to be reapplied when the coating is generally embedded with particulate matter (dirt

or sand), or the sites are decontaminated, or after eight hours on the skin. Normally, SERPACWA on the skin is effective for four hours in preventing CW agents from penetrating and contacting the skin.

D-3. Use of Skin Exposure Reduction Paste Against Chemical Warfare Agent with Other Nuclear, Biological, or Chemical Protective Material

a. Military M40 Protective Mask. Use of SERPACWA and the military protective mask together does not require any change in doctrine on the use of the protective mask. The SERPACWA does not interfere with the sealing capability of the protective mask. No loss of vision (such as eye irritation or fogging on the mask lens) due to SERPACWA use is expected. The SERPACWA is odorless.

b. Battle Dress Overgarment/JSLIST. Use of SERPACWA should not reduce the effectiveness of the BDO/JSLIST. Since it has no water content, it will not wet the BDO/JSLIST.

c. Chemical Agent Detection Systems. The SERPACWA on the skin will not register a false alarm with the automatic detectors (such as ICAM) and CW agent detector systems, such as M8 paper for G-nerve agents or vesicants (SERPACWA must not be on the surface of M8 paper because it prevents the CW agent from contacting the M8 paper).

d. M291 Skin Decontaminating Kits. The M291 SDKs are more effective when SERPACWA is applied on the skin because it is easier to physically remove CW agents from the SERPACWA layer than from the skin. Service members should perform skin decontamination immediately after chemical contamination, as the effectiveness of SERPACWA decreases with time.

e. Insect Repellent, DEET. Use of DEET on the skin, before or after SERPACWA application, will decrease the effectiveness of the SERPACWA. Avoid applying DEET as much as possible on skin areas where SERPACWA is to be applied. (The SERPACWA can still provide significant protection by physically removing DEET from the skin using a dry wipe [towel, gauze, or clothing], not a wet wipe, before applying the SERPACWA.)

D-4. Steps for Applying Skin Exposure Reduction Paste Against Chemical Warfare Agent

a. When directed by your commander/leader, apply SERPACWA as follows:

(1) Remove the SERPACWA from your uniform pocket or rucksack.

(2) Wipe off sweat and remove all loose dirt or sand from your neck, hands, wrists, and lower leg (at the boot tops). If applicable, remove insect repellent with dry (must not be wet) towel or gauze or any other available clean item. Dry your armpits, waistline, creases and crack of buttocks, and groin area as much as possible.

(3) Tear open a SERPACWA packet. Place about a third of the SERPACWA from the container into your hand.

(4) Rub and work the SERPACWA into the neck (all surfaces from the back of the hairline to the jaw line, then under the chin), to the lower legs (at the boot tops); using one hand for each side; then to the wrists and back of the hands.

(5) Apply and work the remainder of the package contents to the groin area, all creases and the crack of the buttock, and the waist (about 2-inch wide band around the waist and the armpits).

(6) Rub and work excess SERPACWA, if any, evenly over areas where it has been applied (in order: wrist, neck, and legs at boot top) and ensure an even distribution.

b. If the CW agent threat continues, reapplication of SERPACWA will be needed at the following times:

(1) After decontamination of CW agent from the SERPACWA protected skin areas;

(2) After washing and brushing the SERPACWA protected areas;

(3) When the SERPACWA barrier becomes disturbed by embedded particulate matter such as sand or dirt, or by rubbing with towel or clothing;

(4) After eight hours of continuous wear if mission permits; or

(5) At the direction of your commander/leader.

D-5. Removal of Skin Exposure Reduction Paste Against Chemical Warfare Agent

The SERPACWA can be removed by brushing and scrubbing the skin areas with soap and water.

NOTE

The protective overgarment will not be removed to apply additional layers of SERPACWA when in a contaminated environment.

Section II. INDIVIDUAL SKIN AND PERSONAL EQUIPMENT DECONTAMINATION

D-6. Detailed Procedures for Decontaminating the Eyes

Any suspected CW 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 CW 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-7 below.

D-7. Detailed Procedures for Decontaminating the Skin (Hands, Face, Neck, Ears, and Other Exposed Areas) Using the M291 Skin Decontaminating Kit

The M291 Skin Decontaminating Kit (Figure D-3) is provided to service members for skin decontamination only. This kit may also be used if necessary to partially decontaminate selected individual equipment, such as load-bearing equipment, protective gloves, mask, hood, and weapon, but does not contain sufficient resin to guarantee decontamination. The M295 DKIE should be used for equipment decontamination if available.

WARNING

The M291 SDK 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 hypochlorite solution, if available, to wash CW agents out of cuts or wounds.

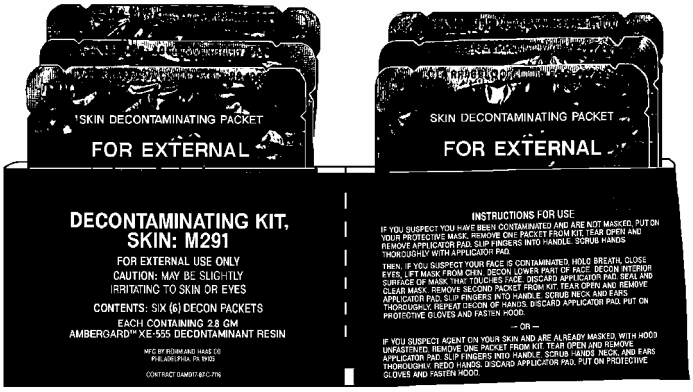


Figure D-3. The M291 skin decontaminating kit.

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 fingers 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 step *s* 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 JSLIST suit, the hood is attached to the jacket.

WARNING

Injury or death may result if you breathe CW 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 the 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 the 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 the mouth and to

the 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 the chin and to the 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. Used M-291 SDK materials can become an off-gassing risk. Bury the used pads and

packets, if circumstance permit.

D-8. Procedures for Decontaminating Individual Equipment Using the XM295 Kit

a. The XM295 Decontaminating Packet, Individual Equipment (Figure D-4) is designed for use in decontamination of individual equipment. The contents of this kit are identical to those contained in the M291 SDK, except that the packets are much larger.

b. Use a stick or other object to remove any thickened spots of CW agent from the equipment.

c. Open the packet, remove the pad, and place your fingers through the slot in the pad.

d. Rub all surface areas of the equipment with the pad.

WARNING

The XM295 is not approved for use on the skin by the FDA. Only use the XM295 kit on equipment. Keep the decontaminating material out of the eyes; it may be slightly irritating to the skin and eyes.

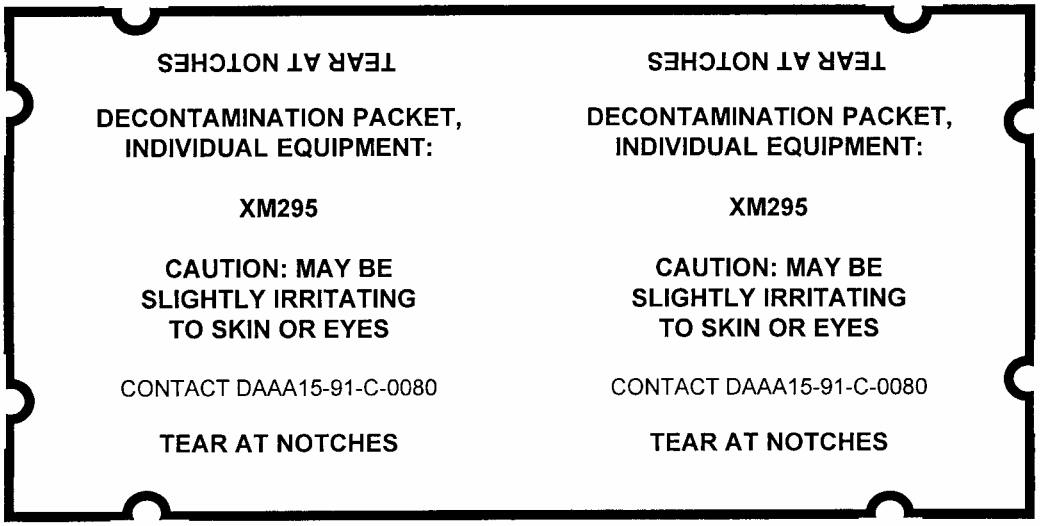


Figure D-4. The XM295 Decontaminating packet, individual equipment.

APPENDIX E

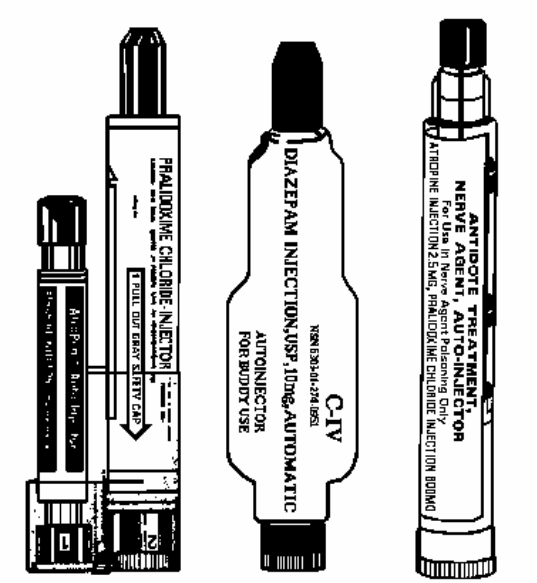
PROCEDURES FOR ADMINISTERING THE NERVE AGENT ANTIDOTES

E-1. Injection Site

The injection site for administering the ATNAA or MARK I and CANA (Figure 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 (Figure E-2). Injections should 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 (Figure E-3). Injecting in the buttocks of thinly-built individuals avoids injury to the thigh bone.

NOTE

The ATNAA will replace the MARK I when the supplies of MARK I are exhausted.



MARK I CANA ATNAA

Figure E-1. Nerve agent antidotes.

E-2. Self-Aid

a. Self-Administer MARK I.

(1) If you experience any or all of the nerve agent mild symptoms (paragraph 3-5*b*(1)), you must immediately put on your protective mask and self-administer one MARK I (Figure E-1).

Follow the procedure given Table E-1. The MARK I is carried in your protective mask carrier, pocket of the MOPP overgarment, or other location as specified in your unit tactical standing operating procedure (TSOP). (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 immediately used to provide you with antidote, when needed. (The MARK I can still be used after complete thawing.)

Table. E-1. Self Aid for Nerve Agent Poisoning

MARK I*	ATNAA*
STEP 1. OBTAIN ONE MARK I**	**STEP 1.** OBTAIN ONE ATNAA**
STEP 2. CHECK INJECTION SITE	**STEP 2.** CHECK INJECTION SITE
STEP 3. HOLD MARK I AT EYE LEVEL WITH NONDOMINANT HAND WITH THE LARGE INJECTOR ON TOP (FIGURE E-4A)	**STEP 3.** HOLD ATNAA WITH DOMINANT HAND (FIGURE E-9A).
STEP 4. GRASP SMALL INJECTOR (ATROPINE) (FIGURE E-4B) AND REMOVE FROM CLIP (FIGURE E-4C).	**STEP 4.** GRASP SAFETY CAP WITH NONDOMINANT HAND AND REMOVE FROM INJECTOR (FIGURE E-9B). DROP THE SAFETY CAP TO THE GROUND.
STEP 5. CLEAR HARD OBJECTS FROM INJECTION SITE.	**STEP 5.** CLEAR HARD OBJECTS FROM INJECTION SITE.
STEP 6. INJECT ATROPINE AT INJECTION SITE APPLYING EVEN PRESSURE TO THE INJECTOR (FIGURE E-5 OR E-6). HOLD IN PLACE FOR 10 SECONDS.	**STEP 6.** INJECT ATNAA AT INJECTION SITE APPLYING EVEN PRESSURE TO THE INJECTOR (FIGURE E-5 OR E-6). HOLD IN PLACE FOR 10 SECONDS.
STEP 7. HOLD USED INJECTOR WITH NONDOMINANT HAND (FIGURE E-7A)	**STEP 7.** BEND NEEDLE OF USED INJECTOR BY PRESSING ON A HARD SURFACE TO FORM A HOOK.
STEP 8. GRASP THE LARGE (2-PAM Cl) INJECTOR (FIGURE E-7B) AND PULL IT FROM CLIP (FIGURE E-7C. DROP CLIP TO GROUND	**STEP 8.** ATTACH USED INJECTOR TO BLOUSE POCKET FLAP OF BDO/JSLIST (FIGURE E-10).
STEP 9. INJECT 2-PAM Cl AT INJECTION SITE APPLYING EVEN PRESSURE TO THE INJECTOR (FIGURE E-5 OR E-6). HOLD IN PLACE FOR 10 SECONDS.	**STEP 9.** MASSAGE INJECTION SITE, MISSION PERMITTING.
STEP 10. BEND THE NEEDLES OF ALL USED INJECTORS BY PRESSING ON A HARD SURFACE TO FORM A HOOK.	
STEP 11. ATTACH ALL USED INJECTORS TO BLOUSE POCKET FLAP OF BDO/JSLIST (FIGURE E-8).	
STEP 12. MASSAGE INJECTION SITE, MISSION PERMITTING.	
LEGEND: * Use steps listed for type of antidote device issued. ** Only administer one MARK I or ATNAA as self-aid. Do not self-administer CANA.	

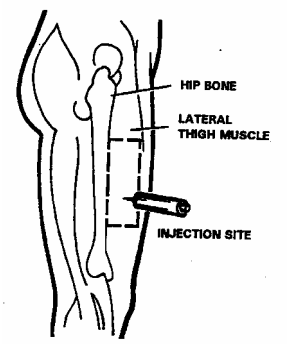


Figure E-2. Thigh injection site.

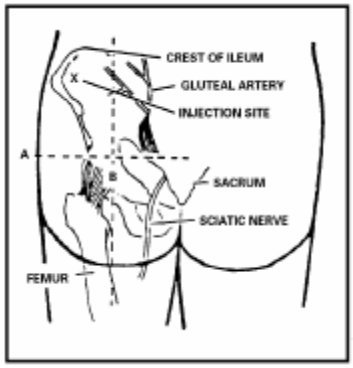


Figure E-3. Buttocks injection site.

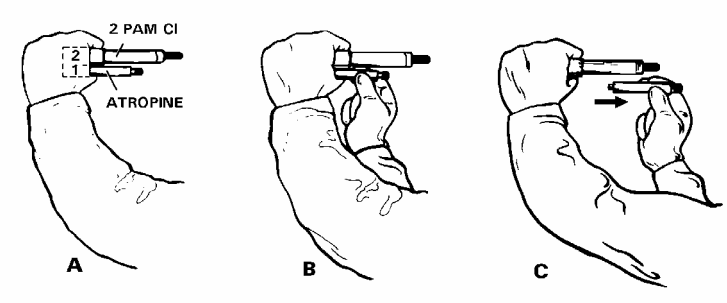


Figure E-4. Removing atropine autoinjector from clip.

Figure E-5. Self-aid thigh injection.

Figure E-6. Self-aid buttocks injection.

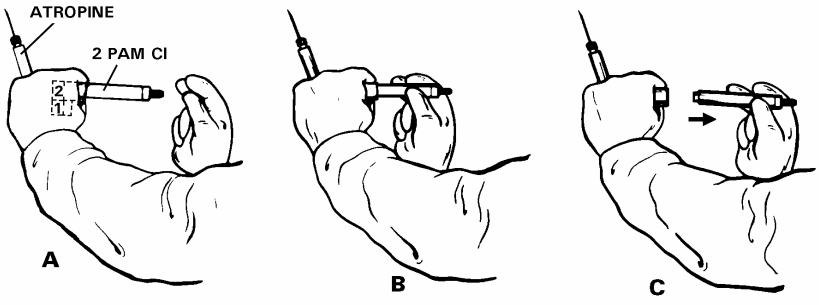


Figure E-7. Removing 2-PAM Cl autoinjector from clip.

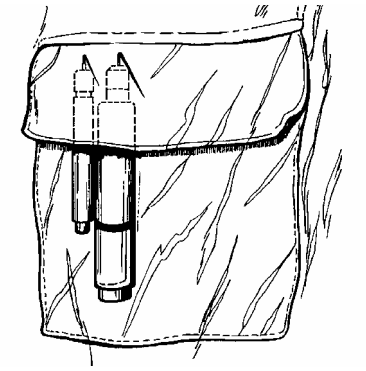


Figure E-8. One set of used autoinjectors attached to pocket flap.

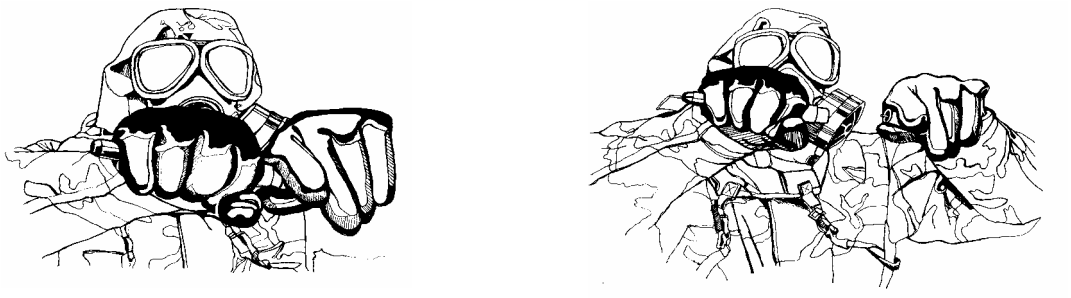
(2) After self-administering the first set of injections, wait 5 to 10 minutes. After administering one set of injections, you should decontaminate your skin (Appendix D), if necessary, and put on any remaining protective clothing.

(a) 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 MARK I. 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 MARK I injection may create a nerve agent antidote overdose, which could cause incapacitation.)

(b*)* 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.

b. Self-Administer ATNAA.

(1) If you experience any or all of the nerve agent mild symptoms (paragraph 3-5*b*(1)), you must immediately put on your protective mask and self-administer one ATNAA (Figure E-1). Follow the procedure given Table E-1 above. The ATNAA is carried in your protective mask carrier, pocket of the MOPP overgarment, or other location as specified in your unit TSOP. (In cold weather, the ATNAA should be stored in an inside pocket of your clothing to protect the antidote from freezing.) A frozen ATNAA cannot be immediately used to provide you with antidote, when needed. (The ATNAA can still be used after complete thawing.)



A B

Figure E-9. Preparing ATNAA or CANA for injection.

(2) After administering the first injection, wait 10 to 15 minutes. After administering one ATNAA, you should decontaminate your skin (Appendix D), if necessary, and put on any remaining protective clothing.

(a) 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 ATNAA. If you are able to walk without assistance (ambulate), know who you are, and where you are, you will not need the second ATNAA. (If not needed, giving yourself a second ATNAA may create a nerve agent antidote overdose, which could cause incapacitation, especially if you are hot.)

(b) If you continue to have symptoms of nerve agent poisoning, seek someone else (a buddy) to check your symptoms and administer your remaining antidotes, if required.

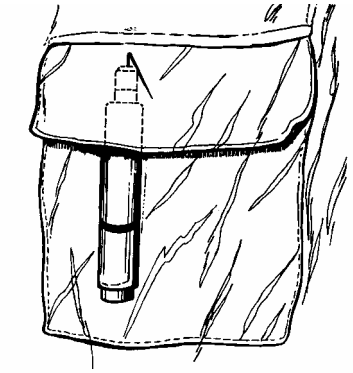


Figure E-10. Used ATNAA attached to clothing.

E-3. Buddy Aid/Combat Lifesaver Aid

Service members may seek or require further assistance after self-aid (self-administering one MARK I or ATNAA). 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 (paragraph 3-5b(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 ATNAA or one set of MARK I autoinjectors has already been used. No more than three sets (total) of the antidote are to be administered. Buddy aid also includes administering the CANA with the third MARK I or ATNAA to prevent convulsions. Follow the procedures indicated in Table E-2.

WARNING

Squat, do not kneel, when masking the casualty or administering the nerve agent antidote to the casualty. Kneeling may force the chemical agent into or through your protective clothing.

CAUTION

Do not use your own MARK I, ATNAA, or CANA on a casualty. If you use your own, you may not have any antidote for self-aid.

WARNING

Do not inject into areas close to the hip, knee, or thighbone.

Table E-2. Buddy Aid/Combat Lifesaver Aid for Nerve Agent Casualty

MARK I*	ATNAA*	CANA**
STEP 1. MASK THE CASUALTY AND POSITION HIM ON HIS SIDE (SWIMMER'S POSITION).	**STEP 1.** MASK THE CASUALTY AND POSITION HIM ON HIS SIDE (SWIMMER'S POSITION).	**STEP 1.** OBTAIN BUDDYS' CANA.
STEP 2. POSITION YOURSELF NEAR THE CASUALTY'S THIGH.	**STEP 2.** POSITION YOURSELF NEAR THE CASUALTY'S THIGH.	**STEP 2.** CHECK INJECTION SITE.
STEP 3. OBTAIN BUDDYS' THREE OR REMAINING MARK Is.	**STEP 3.** OBTAIN BUDDYS' THREE OR REMAINING ATNAAs.	**STEP 3.** HOLD CANA IN A CLOSED FIST WITH DOMINANT HAND (FIGURE E-9A).
STEP 4. CHECK INJECTION SITE.	**STEP 4.** CHECK INJECTION SITE.	**STEP 4.** GRASP SAFETY CAP WITH NONDOMINANT HAND AND REMOVE FROM INJECTOR (FIGURE E-9B) DROP SAFETY CAP TO THE GROUND.
STEP 5. HOLD MARK I WITH NONDOMINANT HAND (FIGURE E-4A).	**STEP 5.** HOLD ATNAA IN A CLOSED FIST WITH DOMINANT HAND (FIGURE E-9A).	**STEP 5.** CLEAR HARD OBJECTS FROM INJECTION SITE.
STEP 6. GRASP SMALL INJECTOR (ATROPINE) AND REMOVE FROM CLIP (FIGURE E-4B.)	**STEP 6.** GRASP SAFETY CAP WITH NONDOMINANT HAND AND REMOVE FROM INJECTOR (FIGURE E-9B). DROP THE SAFETY CAP TO THE GROUND.	**STEP 6.** INJECT CANA AT INJECTION SITE BY APPLYING EVEN PRESSURE TO THE INJECTOR, NOT A JABBING MOTION (FIGURE E-11 OR E-12). HOLD IN PLACE FOR 10 SECONDS.
STEP 7. CLEAR HARD OBJECTS FROM INJECTION SITE.	**STEP 7.** CLEAR HARD OBJECTS FROM INJECTION SITE.	**STEP 7.** BEND NEEDLE OF INJECTOR BY PRESSING ON A HARD SURFACE TO FORM A HOOK.
STEP 8. INJECT ATROPINE AT INJECTION SITE BY APPLYING EVEN PRESSURE TO THE INJECTOR, NOT A JABBING MOTION (FIGURE E-5 OR E-6). HOLD IN PLACE FOR 10 SECONDS.	**STEP 8.** INJECT ATNAA AT INJECTION SITE BY APPLYING EVEN PRESSURE TO THE INJECTOR, NOT A JABBING MOTION (FIGURE E-11 OR E-12). HOLD IN PLACE FOR 10 SECONDS.	**STEP 8.** ATTACH USED INJECTOR TO BLOUSE POCKET FLAP OF BDO/JSLIST (FIGURE E-13 AND E-14).

Table E-2. Buddy Aid/Combat Lifesaver Aid for Nerve Agent Casualty, Continued

MARK I*	ATNAA*	CANA**
STEP 9. HOLD USED INJECTOR BETWEEN LITTLE FINGER AND RING FINGER OF NONDOMINANT HAND (FIGURE E-4A).	**STEP 9.** BEND NEEDLE OF INJECTOR BY PRESSING ON A HARD SURFACE TO FORM A HOOK.	**STEP 9.** MASSAGE INJECTION SITE, MISSION PERMITTING.
STEP 10. PULL LARGE INJECTOR (2-PAM Cl) FROM CLIP (FIGURE E-4C.). DROP CLIP TO GROUND.	**STEP 10.** ATTACH ALL USED INJECTORS TO BLOUSE POCKET FLAP OF BDO/JSLIST (FIGURE E-14).	
STEP 11. INJECT 2-PAM Cl AT INJECTION SITE BY APPLYING EVEN PRESSURE TO THE INJECTOR, NOT A JABBING MOTION (FIGURE E-11 OR E-12). HOLD IN PLACE FOR 10 SECONDS.	**STEP 11**. MASSAGE INJECTION SITE, MISSION PERMITTING.	
STEP 12. REPEAT STEPS ABOVE FOR REMAINING MARK Is.		
STEP 13. BEND THE NEEDLES OF ALL USED INJECTORS BY PRESSING ON A HARD SURFACE TO FORM A HOOK.		
STEP 14. ATTACH USED ALL INJECTORS TO BLOUSE POCKET FLAP OF BDO/JSLIST (FIGURE E-13).		
STEP 15. MASSAGE INJECTION SITE, MISSION PERMITTING.		
LEGEND: * Use steps listed for the type of antidote device issued. ** CANA is used in buddy aid/combat lifesaver aid only. Do not use in self-aid.		

Figure E-11. Injecting the casualty's thigh.

Figure E-12. Injecting the casualty's buttocks.

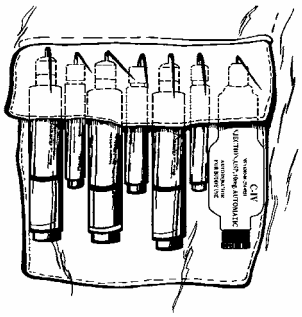


Figure E-13. Three sets of used MARK I autoinjectors and one CANA attached to pocket flap.

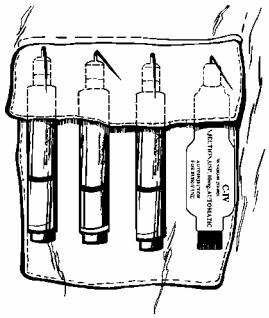


Figure E-14. Three used ATNAA autoinjectors and one CANA autoinjector attached to clothing.

GLOSSARY

Section I. ABBREVIATIONS AND BREVITY CODES

4N0 Medical Service Technician (Air Force)

AC hydrogen cyanide (also called hydrocyanic acid)
ACADA Automatic Chemical Agent Detector and Alarm
AFI Air Force Instruction
AFM Air Force Manual
AFMAN Air Force Manual
AFP Air Force Pamphlet
AFTTP(I) Air Force Technical Training Publication (Interim)
AR Army Regulation
ASD(HA) Assistant Secretary of Defense (Health Affairs)
ATNAA Antidote Treatment, Nerve Agent, Autoinjector
ATP adenosine triphosphate
ATTN attention

BAL British anti-lewisite (dimercaprol)
BDO battle dress overgarment
BZ 3-quinuclidinylbenzilate (incapacitating agent)

C Celsius
CA bromobenzylcyanide (riot control agent)
CAM chemical agent monitor
CANA convulsant antidote for nerve agent (diazepam)
CASEVAC casualty evacuation
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
COHb carboxyhemoglobin
COMM commercial
COSR combat/operational stress reactions
CPAP continuous positive airway pressure
CPS collective protection shelter
CR dibenz-(b,f)-1,4-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)
DOD Department of Defense
DEET 75 percent N N-diethyl-M-tolumide

DKIE Decontamination Kit, Individual Equipment (M295)
DM diphenylaminochloroarsine (Adamsite) (vomiting agent)
DNA deoxyribonucleic acid
DP diphosgene (choking agent)
DSN Defense Switched Network

EAC echelon above corps
ED ethyldichloroarsine
EMT emergency medical treatment
EO Executive Order

F Fahrenheit
FDA Food and Drug Administration
FHPPP Force Health Protection Prescription Products
FM titanium tetrachloride
FM field manual (when used with a number)
FS sulfur-trioxide chlorosulfonic acid solution (smoke mixture)

GA tabun (a G-agent)
G-agent a nerve agent
GB sarin (a G-agent)
G CSF granulocyte colony stimulating factor
GD soman (a G-agent)
GF a nerve agent
gm gram(s)

H European countries' term for HD (sulfur mustard)
HAZMAT hazardous material
H_2SO_4 sulfuric acid
HC a mixture of grained aluminum, zinc oxide, and hexachloroethane (a smoke producer)
HD sulfur mustard (a blister agent)
HN nitrogen mustard (a blister agent)
HN1 2,2'-Dichlorotriethylamine (type of mustard agent)
HN3 2,2',2"-Trichlorotriethylamine (type of mustard agent)
HNO_3 nitric acid
HQ headquarters

IARC International Agency for Research on Cancer
ICAM improved chemical agent monitor
IDN initial distribution number
IM intramuscular
IND investigational new drug
IPE individual protective equipment
IPPB intermittent positive pressure breathing
IR infrared
IV intravenous

JSLIST joint-service lightweight integrated-suit technology

L Lewisite (a blister agent); chlorovinyldichloroarsine
LC_{50} lethal concentration for 50 percent of exposed persons
LCt lethal concentration
LD_{50} lethal dose 50 percent

LSD d-lysergic acid diethylamide

MARK I (NAAK) MARK I (Nerve Agent Antidote Kit)
MCCDC Marine Corps Combat Development Command
MCPDS Marine Corps Publication Distribution System
MCRP Marine Corps Reference Publication
MD methyl dichloroarsine
MES medical equipment sets
MG magnesium
mg milligram(s)
ml milliliter(s)
mm millimeter(s)
mm-wave millimeter microwave
MILSTRIP military standard requisitioning and issue procedure
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 Cl
NAVSOP Naval Standard Operating Procedure
NATO North Atlantic Treaty Organization
NAVFAC Naval Facilities Engineering Command
NBC nuclear, biological, and chemical
NH_3 ammonia
NO_X oxides of nitrogen
NO nitric oxide
N_2O nitrous oxide
NO_2 nitrogen dioxide
N_2O_4 dinitrogen tetroxide
NTRP Navy Technical Reference Publication
NWDC Navy Warfare Development Command

OPR Office of Primary Responsibility
OSHA Occupational Safety and Health Administration

Pam pamphlet
PB pyridostigmine bromide
PD phenyldichloroarsine (blister agent)
PFIB perfluoroisobutylene
PEEP positive end-expiratory pressure
ppm parts per million
PPW patient protective wrap
PS chloropicrin (irritant agent)

RP red phosphorus
® registered trademark
RTD return to duty

SCBA self-contained breathing apparatus
SDK skin decontaminating kit
SERPACWA skin exposure reduction paste against chemical warfare agents
SGF2 fog oil (smoke producing product)
SNAPP soman nerve agent pretreatment pyridostigmine

SOP standing operating procedure
STANAG standardization agreement

TB MED technical bulletin, medical
TH thermite (incendiary)
TIC toxic industrial chemical
TIM toxic industrial material
TM technical manual
TRADOC training and doctrine command
TSOP tactical standing operating procedure
2-PAM Cl pralidoxime chloride

US United States
USAF United States Air Force
USMC United States Marine Corps
USN United States Navy

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

ACADA Automatic Chemical Agent Detector and Alarm

acetic acid A clear, colorless organic acid with a distinctive pungent odor.

acetylcholine A chemical compound formed from an acetic acid and choline that 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 acid 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.

adenosine triphosphate (ATP) A nucleotide involved in energy metabolism. It is produced by hydrolysis of ATP and converted back to ATP by oxidative phosphorylation and substrate-level phosphlorylation.

aerosols A suspension or dispersion of small particles (solids or liquids) in a gaseous medium.

alveolar septa Septum of the alveolus.

alveoli Microscopic air sac in the lungs where oxygen and carbon dioxide diffusion takes place through the alveolar walls.

ammonia A colorless alkaline gas (NH_3) having a penetrating odor.

amphetamine A central nervous system stimulant. May be used as an incapacitating agent. Most common form is a tablet.

amyl nitrite A flammable clear liquid with an ethereal odor, volatile at low temperatures and administered by inhalation. It is a vasodilator and can be used as a diagnostic aid and in the treatment of cyanide poisoning to promote formation of methemoglobin, which combines with the cyanide ion to form nontoxic cyanmethemoglobin.

anaerobic metabolism Metabolism that occurs in the absence of atmospheric oxygen.

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.

analgesia The relief of pain without loss of consciousness.

analgesic An agent that alleviate pain without causing loss of consciousness.

anhidrosis The absence or severe deficiency of sweating.

anorexia Loss of appetite.

anoxia Lack of oxygen.

antecubital fossae A small cavity in the arm in front of the elbow.

antibiotic A natural 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.

antiemetics An agent that prevents or alleviates nausea and vomiting.

antimalarial An agent that is therapeutically effective against malaria.

antimicrobial An agent that kills microorganisms or suppresses their multiplication or growth.

aphonia Inability to phonate or produce speech sounds.

apnea Cessation of breathing.

apneic Without breathing or respirations.

areflexia Failure to empty the bladder completely on urination.

arrhythmias Any variation from the normal rhythm of the heartbeat.

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.

asthma Difficult breathing associated with bronchial obstruction precipitated by respiratory inhalants, toxins, or allergies. Inhaled chemical agents may cause bronchial spasms ormucous 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.

autonomic nervous system The portion of the nervous system concerned with regulation of the activity of cardiac muscle, smooth muscle, and glands.

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 antidotalproperties 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), arsenicals (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.

bradycardia Heart rate less than 50.

British anti-lewisite Commercial name for a chemical compound (dimercaprol) which is used as an (BAL) antidote for heavy metal poisoning—specifically, arsenic (a component of L).

bromides Any binary compound of bromine in which the bromine carries a negative charge; specifically a salt of hydrobromic acid.

bromine A reddish-brown liquid element that gives off suffocating vapors.

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.

bronchi Any of the larger air passages of the lungs.

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.

bronchioles One of the finer subdivision of the branched bronchial tree, differing from the bronchi in having no cartilage plate and having cuboidal epithelial cells.

bronchioloitis obliterans Inflammation of the bronchioles that may follow inhalation of irritating gases or foreign bodies.

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.

bronchodilators An agent that causes expansion of the channels in the air passages of the lungs.

bronchopneumonia Inflammation of the terminal bronchioles and alveoli, causing edema and consolidation of alveoli.

bronchorrhea Excessive discharge of mucus from the bronchi.

bronchoscopy Examination of the bronchi through a bronchoscope.

bulbar Pertaining to a bulb or involving the medulla oblongata.

bullae A large elevation on the skin containing serous fluid.

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 marijuana. (See cannabinols.)

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.

carbonyl groups A major group of compounds studied in organic chemistry in which an oxygen atom is double-bonded to a carbon atom that has two additional bonds to attach to the rest of the molecule.

carboxyhemoglobin (COHb) Hemoglobin in which the sites usually bound to oxygen are bound to carbon monoxide.

carcinogen Any cancer-causing substance.

cardiospasm Failure of the esophagus to relax when swallowing.

cathartic An agent that causes emptying of the bowels.

celiac ganglion Nerve cell bodies in the abdomen.

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.

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.

chloramines An irritating and odorous chemical compound containing chlorine.

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.

chlorovinyldichloroarsine Lewisite.

chlorpyrifos A common organophosphate insecticide that is a cholinesterase inhibitor.

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 body Tissue that include the group of muscles that act on the eye lens.

ciliary spasm Spasm of the muscles of the eyelids which is usually painful and may interfere with functioning of the eyelid.

coagulation necrosis A type of necrosis in which the affected tissue is seen as a dry mass due to the coagulation of protein.

codeine An analgesic obtained from opium, acceptable for the relief of moderate pain and used to suppress coughing.

collagen Protein substance of connective tissue.

conjunctiva 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.

continuous positive airway pressure (CPAP) Respiratory therapy in which airway pressure is maintained above atmospheric pressure by pressurization of the ventilatory circuit.

convalescence The state of recovery following disease, surgery, or an injury.

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.

cordite A smokeless explosive powder consisting of nitrocellulose, nitroglycerin, and petrolatum that has been dissolved in acetone, dried, and extruded in cords.

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.

crackle A discontinuous sound consisting of a series of short noises heard during inhalation. See rale.

cryptogenic organizing pneumonia A condition resembling the inflammation of the bronchioles with an ingrowth of connective tissue combined with organizing pneumonia. Terminal bronchioles and alveoli become occluded with masses of inflammatory cells and fibrotic tissue.

cuboidal epithelium Cuboidal refers to cells that are shaped like a cube. Cells lining the surfaces of organs and the body are known as epithelium.

cupric (copper) sulfate An emetic used orally as an antidote to phosphorus poisoning.

curare A wide variety of highly toxic extracts from numerous botanical sources.

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 agent. 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.

cycloplegic An agent that causes paralysis of the ciliary muscle.

cytochrome oxidase The terminal enzyme of the electron transport chain.

d-amphetamine (dextroamphetamine sulfate) A medication that is a CNS stimulant. Frequently used in drug abuse, a common isomer of amphetamine sulfate.

dermatitis An inflammation or infection of the skin.

desquamation Exfoliation.

diazepam An anticonvulsant drug used to decrease convulsive activity and reduce the brain damage caused by prolonged seizure activity. Used in the treatment of nerve agent poisoning.

diazinon An insecticide that is a cholinesterase inhibitor.

dibenz-(b,f)-1,4-oxazepine (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).

dichlorvos An organophosphorus insecticide.

diphenylaminearsine chloride (Adamsite, DM) A vomiting agent.

diphenylchloroarsine (DA) A vomiting agent.

diphenylcyanoarsine (DC) A vomiting agent.

diphosgene (DP) A gas that is intensely irritating to the lungs, producing pulmonary edema.

diphtheria An acute infectious disease caused by toxigenic strains of *Corynebacterium diphtheriae*, usually confined to the upper respiratory tract and characterized by the formation of a tough false membrane attached to the underlying tissue.

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.

dyspnea Labored breathing resulting from an increased need for oxygen or inadequate air exchange in the lungs.

dysrhythmias Abnormal cardiac rhythm.

edema Excess fluid buildup in the tissues causing swelling.

emetic An agent that causes vomiting.

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.

encephalopathy Any degenerative disease of the brain.

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.

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.

eructation Belching.

erythema Red area of the skin, caused by heat or cold injury, trauma, or inflammation. May be l ocalized 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.

fentanyl derivatives Derivatives of a narcotic analgesic that can be inhaled and act on the central nervous system.

fibrosis Scar tissue, replacement by fibrous tissue.

flaccid paralysis Loss of muscle tone and capability to function. Nerve agents cause this condition.

fog oil A smoke made from a special petroleum oil.

G-agent A nerve agent such as GA, GB, GD and GF.

ganglia Group of nerve cell bodies located outside the central nervous system.

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.

glycolate A salt, anion, or ester of glycolic acid.

granulocyte colony stimulating factor (G-CSF) A glycoprotein that stimulates the growth and maturation of white blood cells.

hallucinogen A drug which produces visual, auditory, and olfactory imaginary sensations. Such drugs are cannabinols, LSD, peyote, and alcohol.

halogenated oximes An oxime that contains a halogen.

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.

hemorrhage Bleeding.

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.

hexachloroethane A colorless crystalline compound used as a camphor substitute and in pyrotechnics, explosives, and veterinary medicine.

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 corrosive solution of hydrogen chloride gas in water.

hydrogen chloride A colorless, corrosive gas.

hydrogen cyanide (AC) 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 fluoride A corrosive and poisonous inorganic acid.

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.

hyperpigmentation Abnormally increased pigmentation.

hypertension High blood pressure, usually greater than 140 systolic and 90 diastolic.

hyperthyroidism A condition caused by excessive production of iodinated thyroid hormones.

hyperthermia The elevation of the core body temperature to above 99° F (37.2° C).

hyperventilation Excessive breathing (too rapid and/or too deep) with a resultant decrease in carbon dioxide tension and respiratory alkalosis.

hypopigmentation Abnormally diminished pigmentation resulting from decreased melanin production.

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.

indoles A heterocyclic compound (more than one atom in a ring) obtained from coal tar and also produced by the decomposition of tryptophan in the intestine.

inspiratory stridor A harsh, high-pitched breath sound heard on inhalation.

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.

investigational new drug (IND)

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.

keratitis Inflammation of the cornea.

labile Unstable.

lacrimal glands Tear glands.

lacrimation Secretion and discharge of tears.

lacrimator A substance which induces the secretion of tears.

lactic acidosis The accumulation of lactic acid.

laryngitis Swelling, redness, and inflammation of the larynx.

laryngospasm Spasmodic closure 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.

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.

lipophilic Having an affinity for fat.

liquefaction necrosis Death of tissue, with softening to the point that tissue becomes at least partially liquefied.

lumen The cavity or channel within a tube or tubular organ.

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.

M8 Detector Paper A chemical agent detector paper used to detect and identify liquid V- and G-type Chemical Agent nerve agents and H-type blister agents. It does not detect chemical agent vapors.

M256 Chemical Agent Detector Kit A kit that detects and identifies vapor concentrations of nerve, blister, and blood agents.

M291 Skin Decontaminating Kit A kit to perform emergency decontamination of the skin and mask. The kit contains six decontamination packets.

M295 Decontamination Kit, Individual Equipment (DKIE) A kit (similar to the M291 Skin Decontaminating Kit) used to decontaminate Individual 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.

maceration Destruction of soft tissue, usually associated with prolonged immersion in water or wetness and may, in some cases, have been associated with trauma.

mafenide acetate The monoacetate salt of mafenide used as a topical anti-infective for adjunctive therapy of patients with second- and third-degree burns.

magnesium An element which, in metal form, burns readily at high temperatures, splatters readily upon burning, and may cause severe burns.

magnesium citrate A saline laxative.

magnesium sulfate A salt used as an anticonvulsant and an electrolyte replenisher.

malaise A vague feeling of bodily discomfort and fatigue.

malathion (Dimethoxyphosphinothioyl)thio- butanedioic acid, a commercial organophosphorus insecticide. Also known as carbophos, maldison and mercaptothion and sold commercially as Celthion, Cythion, Dielathion, El 4049, Emmaton, Exathios, Fyfanon and Hilthion, Karbofos and Maltox.

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.

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.

mucosa A membrane covering or lining a body part or organ.

muscarinic A specific type of poisoning affecting the postganglionic parasympathetic neuralmuscular junction, resulting from excess acetylcholine due to inhibition of acetylcholinesterase. 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.

mutagenic Causing change or inducting genetic mutation.

mydriasis Large or dilated pupils.

naloxone An alkaloids (group of nitrogenous substances found in plants) antagonist of morphine and of the opiate peptides.

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 autoinjector, 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.

neuromuscular Pertaining to muscles and nerves.

neurotransmitter A group of substances that are released on excitation from the axon terminal of a presynaptic neuron of the central or peripheral nervous system and travel across the synaptic cleft to either excite or inhibit the target cell.

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.

nitrites Salts of nitrous acid.

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 an 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.

nitrous oxide (N_2O) A colorless and odorless gas used as an anesthetic and analgesic.

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.

O-chlorobenzylidene malononitrile A tear gas used primarily as a riot control agent. Potent eye, throat, and skin irritant, but incapacitation is short-lived.

off-label indications The use of licensed mediations for purposes that are not approved by the FDA.

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.

organophosphate A compound with a specific phosphate group which inhibits acetylcholinesterase. Used in CW and as an insecticide.

organophosphorous A compound containing phosphorus bound to an organic molecule. They are highly toxic cholinesterase inhibitors.

oropharyngeal airway A short airway inserted into the oropharynx to prevent the tongue from obstructing the airway.

orthopnea Shortness of breath that is relieved by assuming an upright position.

OSHA Level A Encapsulating chemical resistant protective clothing with self-contained breathing apparatus.

OSHA Level B Nonencapsulating chemical resistant clothing, boots, and gloves with ACBA type devices.

OSHA Level C Nonencapsulating chemical resistant clothing, boots, and gloves with specialized respiratory protection. Respirator either removes particulate matter or gases and vapors from the atmosphere.

oxidative phosphorylation The phosphorylation of ATP.

oxime A compound used to treat nerve agent poisoning. Oximes attach to the nerve agent that is inhibiting the cholinesterase and break the agent-enzyme bond to restore the normal activity of the enzyme. Oximes are less useful after aging occurs, but with the exception of GD (soman) intoxicated individuals, casualties will be treated before significant aging occurs.

ozone A major air pollutant that is irritating and toxic to the respiratory system. It is a bluish explosive gas or liquid formed when oxygen is exposed to the silent discharge of electricity.

pallor A pale appearance to the skin.

palpebral Pertaining to an eyelid.

pancytopenia Deficiency of all cellular elements of the blood.

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.

parasympathetic Pertaining to the division of the autonomic nervous system made up of the ocular, bulbar, and sacral divisions.

parasympathomimetic The effects obtained from stimulation of the parasympathetic portion of the autonomics nervous system, causing cholinergic effects.

parathion A toxic cholinesterase inhibitor used as an insecticide.

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.

parotid gland A gland located near the ear.

paroxysmal coughing A sudden recurrence or intensification of coughing.

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.

peripheral Pertaining to or situated at or near the periphery.

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.

pharyngitis Inflammation of the pharynx.

pharynx The air passageway from the posterior nose to the trachea.

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 (CX) 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.

phthalic acid Any of the isomers (usually theorthoisomer) of the dicarboxylic acid-substituted benzene ring.

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.

pleural effusion A collection of fluid in one side of the chest cavity.

pneumonia Inflammation of the lungs, usually caused by an infective agent. May be secondary to injury to the lungs.

pneumonitis Inflammation of 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 expiratory cycle, thus maintaining a higher pressure than the pulmonary circulation which reduces the pooling or shunting of blood in the lungs.

postural drainage Drainage technique in which the patient's body is positioned so that the trachea is leaning below the chest.

pralidoxime chloride (2-PAM CL) is an oxime used in the treatment chloride of organophosphate insecticides and nerve agent poisoning to block the inhibition of acetycholinesterase.

prednisolone A steroid (glucocorticoid) used in the treatment of choking agents over a course of several days.

presynaptic Situated before or proximal to a synape or occurring before the synapse is crossed.

prostration Extreme exhaustion or powerlessness.

pruritus Itching.

pseudomembrane A false membrane.

psychotomimetic A drug or substance that causes psychological or behavioral changes that resemble those of psychosis.

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.

pulmonary edematogenic Abnormal, diffuse, extravascular accumulation of fluid in the pulmonary tissues and air spaces.

pulmonary fibrosis Formation of fibrous tissues in the lungs.

pyrexia Fever.

pyridostigmine bromide (PB) A cholinesterase inhibitor, which acts by inhibiting destruction of acetylcholine and facilitating transmission of impulses across the neuromuscular junction.

rales A discontinuos sound consisting of a series of short nonmusical noises, heard primarily during inhalation (crackles).

rhinitis Inflammation of the nasal mucosa.

rhinorrhea Thin watery discharge from the nose.

rhonchi A continuous sound consisting of a dry, low-pitched snorelike noise due to a partial obstruction such as by secretions.

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 (GB) A nerve agent of the organophosphorus group which inhibits acetylcholinesterase.

secondary pneumonia Pneumonia seen as a complication of some other disorder.

sepsis The presence of pathogenic microorganisms or their toxins in the blood or other tissues.

septa A dividing wall or partition.

sequelae Any lesion or affection following or caused by an attack of disease.

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 sulfacetamide A medication used either as an ointment or solution in the eye. It is a mild antibacterial agent.

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 (GD) A nerve agent member of the organophosphorus group; inhibits acetylcholinesterase. Used as a chemical warfare agent.

Soman Nerve Agent Pyridostigmine Pretreatment (SNAPP) 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.

stenosis An abnormal narrowing of a duct or canal.

steroid See corticosteroid.

stridor A harsh, high-pitched breath sound.

stupor A lowered level of consciousness manifested by the response to only vigorous stimulation.

sublingual gland Gland beneath the tongue.

submaxillary gland Gland beneath the maxilla (upper jaw).

substernal Under the sternum.

sulfadiazine topical burn cream A sulfonamide drug used in the treatment of infections.

sulfur mustard A severe irritant and vesicant of the eyes, skin, and lungs.

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 sulfur, oxygen, and hydrogen used in industry. It is caustic and corrosive.

suppurative bronchitis Pustular inflammation of the bronchi.

sympathetic A portion of the autonomic nervous system.

synechia Adhesion of parts, especially adhesion of the iris to the lens and cornea.

synesthesia A secondary sensation accompanying an actual perception.

systemic poison A poison that affects the whole body.

tabun (GA) A nerve agent member of the organophosphorus group which inhibits acetylcholinesterase. Is used as a chemical warfare agent. Is the least toxic of the nerve agents but can cause death rapidly.

tachycardia Excessive rapidity in the action of the heart; usually applies to a heart rate above 100 beats per minute in an adult.

tenesmus Straining, especially ineffectual and painful straining at stool or in urination.

thermite Incendiaries that are a mixture of powdered iron oxide, powdered aluminum, and other materials.

thiosulfate The $S_2O_3^{2-}$ anion or a salt containing this ion. It is produced in the metabolism of cysteine and is excreted in the urine.

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.

tobramycin An aminoglycoside antibiotic.

toxidrome A specific sydromelike group of symptoms associated with exposure to a given poison.

trachea The cartilaginous and membranous tube descending from the larynx and branching into the main bronchi.

tracheitis Inflammation of the trachea.

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.

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).

vacuoles A small space or cavity formed in the protoplasm of a cell.

V-agent (VX) A nerve agent of the organophosphorus group that inhibits acetylcholinesterase.

valerate A salt or ester of vlaeric acid.

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.

vesicle A small sac containing liquid.

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.

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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Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Lung-Damaging (Choking) Agents	
Agents	Phosgene (CG), diphosgene (DP), chlorine, and chloropicrin (PS).
Signs & Symptoms	Eye and airway irritation. Dyspnea, coughing, chest tightness, and respiratory distress. Pathophysiology: Central agents: laryngospasm, loss of airway Peripheral agents: non-cardiogenic pulmonary edema.
Detection	Odor – newly mown hay or freshly cut grass or corn. Sensors – MINICAMS, Monitox Plus, Draeger tubes, Individual Chemical Agent Detector (ICAD), M18A2, M90, and M93A1 Fox. Other – M256A1, M8 paper, and M9 paper are not designed to identify phosgene.
Protection	Military chemical protective mask.
Decontamination	Vapor – Removal of victim to uncontaminated/fresh air. Liquid – Copious water or soap/water solutions.
First Aid/Buddy Aid	Termination of exposure. Enforced rest, warmth, and observation.
Medical Management	Termination of exposure. Basic life support: airway control, oxygenation, and ventilation, and circulatory support, as needed. Enforced rest, warmth, and observation. Supplemental oxygen with/without positive airway pressure. More aggressive supportive therapy (pulmonary and airway management), if required.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Cyanogens (Blood Agents)	
Agents	Hydrogen cyanide (AC) and cyanogen chloride (CK)
Signs & Symptoms	Low threshold between initial symptoms and severe physiological distress. After exposure to high Ct, seizures, respiratory and cardiac arrest.
Detection	Odor – Peach kernels or bitter almonds (absent in 50%). Sensors – M256A1 detector ticket: AC vapor or gas in the air M272 kit detects cyanide in water. ICAD, M18A2, and M90 detectors detect AC. Other – CAM/ICAM, M8A1, and M8 and M9 paper do not detect cyanide.
Protection	Military chemical protective mask (vapor); MOPP IV (liquid).
Decontamination	Usually unnecessary. Remove wet, contaminated clothing and decontaminate underlying skin with water or soap/water solutions.
First Aid/Buddy Aid	Termination of exposure. Fresh, uncontaminated air.
Medical Management	Termination of exposure. Basic life support: airway control, oxygenation, and ventilation, and circulatory support, as needed. Antidotes: amyl nitrite inhalation ampules if available, followed by intravenous (IV) sodium nitrite and sodium thiosulfate. Supportive: administer oxygen, correct metabolic acidosis.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Vesicants	
Agents	H (sulfur mustard), HD (distilled mustard), HN (nitrogen mustard), arsenical vesicants (Lewisite, phenydichorarsine [PD], ethyldichloroarsine [ED], methyldichloroarsine [MD]), CX (phosgene oxime).
Signs & Symptoms	Initial: Asymptomatic (except Lewisite) Subacute: Skin, eye, and respiratory tract irritation; erythema and blisters on the skin and all exposed mucous membranes; conjunctivitis, corneal opacity, and reactive blepharospasm; pulmonary tissue and respiratory tract inflammation; secondary bacterial pneumonia Late: Bone marrow suppression, generalized sepsis
Detection	Odor – Usually none, although some have a faint odor. Sensors – M256A1, M272, MINICAMS, ICAD, M18A2, M21, M90, M93A1 Fox, Bubbler, CAM/ICAM, DAAMS, M8 paper, or M9 paper. Other – M8A1 will NOT detect.
Protection	MOPP 4. OSHA Level A or B, depending on concentration.
Decontamination	Skin Decontamination Kit, copious water or soap/water solutions.
First Aid/Buddy Aid	Termination of exposure/immediate decontamination. Protect blisters and open wounds.
Medical Management	Termination of exposure/immediate decontamination. Basic life support: airway control, oxygenation, and ventilation, and circulatory support, as needed. Supportive care: correct fluid losses, protective bandages for bullae, open lesions.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Nerve Agents	
Agents	GA (tabun), GB (sarin), GD (soman), GF (cyclosarin), VX
Signs & Symptoms	Mild: unexplained runny nose, unexplained sudden headache, sudden drooling, difficulty in seeing (dimness of vision and miosis), tightness in the chest or difficulty breathing, wheezing and coughing, localized sweating and muscular twitching in the area of contaminated skin, stomach cramps, nausea with or without vomiting, and tachycardia followed by bradycardia. Severe: strange or confused behavior, increased wheezing and increased dyspnea, severely pinpointed pupils, red eyes with tearing, vomiting, severe muscular twitching and general weakness, involuntary urination and defecation, convulsions, unconsciousness, respiratory failure.
Detection	M256A1, CAM/ICAM, M8 paper, M9 paper, M8A1, and M8 alarm systems.
Protection	MOPP 4 Skin Exposure Reduction Paste Against Chemical Warfare Agent (SERPACWA) OSHA A or B depending on concentration
Pretreatment	Soman Nerve Agent Pyridostigmine Bromide Pretreatment (SNAPP).
Decontamination	Skin Decontamination Kit, copious water or soap/water solutions.
First Aid/Buddy Aid	Termination of exposure/immediate decontamination. Antidotes: Atropine and 2 PAM Cl by autoinjector. (Self aid – one MARK I kit or 1 ATNAA; buddy aid or combat life support – up to three sets of MARK I kit or 3 ATNAA).
Medical Management	Termination of exposure/immediate decontamination. Antidotes: Atropine and 2-PAM Cl. Diazepam (severe exposure or convulsions). Basic life support: airway control, oxygenation, and ventilation, and circulatory support, as needed. Ventilation and suction of airways for respiratory distress. Medical Aerosolized Nerve Agent Antidote (MANNA) (Atropine inhaler) Occular symptoms – atropine sulfate ophthalmic ointment.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Incapacitating Agents	
Agents	BZ. Others include Agent 15, anticholinergics, indoles, and cannabinols.
Signs & Symptoms	Mydriasis; dry mouth; dry skin; altered mental status; confusion; disorientation; disturbances in perception and interpretation (illusions and/or hallucinations); denial of illness; short attention span; impaired memory.
Detection	None.
Protection	Mark 41 Chemical Mask Air purifying respiratory.
Decontamination	Gentle, but thorough flushing of skin and hair with soap and water is required. Bleach is not necessary. M291, SDK can be used if washing is not possible. Remove clothing.
First Aid/Buddy Aid	Termination of exposure/immediate decontamination.
Medical Management	Termination of exposure/immediate decontamination. Antidote: physostigmine. Supportive: monitoring of vital signs, especially core temperature. Ice should not be used for skin cooling. CANA/diazepam may be used to control seizures.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Riot Control Agents (Irritants)	
Agents	CS (O-chlorobenzylidene malononitrile), chloroacetophenone in chloroform (CNC), bromobenzylcyanide (CA), diben-(b, f)-1,4-oxazepine (CR), CN (chloroacetophenone).
Signs & Symptoms	Burning and pain on exposed mucous membranes and skin, eye pain and tearing, burning in the nostrils, respiratory discomfort, and tingling of the exposed skin.
Detection	M256A1, CAM/ICAM, M8 paper, M9 paper, M8A1 and M8 alarm systems.
Protection	Military chemical protective mask; field clothing. Individuals handling CS should wear rubber gloves, protective mask with hood, rubber boots, and rubber apron.
Decontamination	Eyes: thoroughly flush with water, saline, or similar substance. Skin: flush with copious amounts of soap and water. Bleach, which produces irritating byproducts from these agents, should not be used for decontamination. Decontaminate CS-contaminated clothing by airing for a few minutes.
First Aid/Buddy Aid	Termination of exposure/immediate decontamination. Usually none is necessary; effects are self-limiting.
Medical Management	Termination of exposure/immediate decontamination. Usually none is necessary; effects are self-limiting.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Vomiting Agents	
Agents	DA (diphenylchloroarsine), Adamsite [DM] (diphenylaminochloroarsine) and DC (diphenylcyanoarsine).
Signs & Symptoms	Fullness in the nose and sinuses, severe headache, burning in the throat, and chest tightness; eye irritation and lacrimation; intense coughing, sneezing and rhinorrhea. Nausea and vomiting are prominent.
Detection	None available to field units.
Protection	The protective mask provides adequate protection. No protective clothing is required.
Decontamination	Eyes: thoroughly flush with water, saline, or similar substance. Skin: flush with copious amounts of soap and water. Bleach, which produces irritating byproducts from these agents, should not be used for decontamination. Decontaminate CS-contaminated clothing by airing for a few minutes.
First Aid/Buddy Aid	Wear the protective mask until in uncontaminated, fresh air.
Medical Management	Antiemetics for continued symptoms. Aspirin or acetaminophen for headaches and general discomfort.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Toxic Industrial Chemicals	
Agents	Wide range of chemicals. Most commonly encountered—ammonia, carbon monoxide (CO), chlorine vapor, hydrogen sulfide, and oxides of nitrogen.
Signs & Symptoms	Signs and symptoms primarily due to chemical burns of eyes, airways, and skin. In enclosed spaces, secondary effects due to displacement of oxygen. CO binds to hemoglobin and causes symptoms similar to cyanogens.
Detection	Many TICs can only be detected by commercial, industrial chemical detectors, such as Draeger tubes, organic vapor analyzers (Photoionization Detectors (PIDs) and Flame Ionization Detectors (FIDs)), and gas chromatographic analyzers. Typical chemical agent detectors fielded by the military services will not detect or identify many hazardous TICs.
Protection	Self-contained breathing apparatus (SCBA). Military chemical protective masks in general provide no protection against TICs. OHSA Level A, B or C suits may be required depending on concentrations.
Decontamination	In general: Copious amounts of water or soap/water solutions.
First Aid/Buddy Aid	Termination of exposure/immediate decontamination.
Medical Management	In general: Removal from exposure area/decontamination of liquid agents most important aspect of treatment. Monitor and treat for shock. Supportive/symptom-based treatment. Agents with pulmonary effects may require supplemental oxygen, suctioning, and airway control.

Chemical Warfare Agents and Toxic Industrial Chemicals
Immediate/Emergency Treatment Ready Reference

Smokes	
Agents	HC mixtures (containing zinc oxide), fog oil (SGF2), diesel fuel, sulfur trioxide-chlorosufonic acid, titanium tetrachloride, RP (red phosphorus), WP (white phosphorus).
Signs & Symptoms	Eye irritation, burning, lacrimation. Dyspnea, coughing, stridor.
Detection	N/A
Protection	Military chemical protective mask, field clothing.
Decontamination	Eyes: Saline or water. Skin: Copious amounts of water or soap/water solution.
First Aid/Buddy Aid	Termination of exposure/immediate decontamination.
Medical Management	Supportive care with oxygen administration if needed. Bronchial constriction to HC smoke can be treated with epinephrine hydrochloride, as required.