***ATP 4-02.85**
**| MCRP 3-40A.1**
***NTRP 4-02.22**
***AFTTP(I) 3-2.69**

# MULTI-SERVICE TACTICS, TECHNIQUES, AND PROCEDURES FOR TREATMENT OF CHEMICAL WARFARE AGENT CASUALTIES AND CONVENTIONAL MILITARY CHEMICAL INJURIES

## AUGUST 2016

**DISTRIBUTION RESTRICTION:** Approved for public release; distribution is unlimited.

*This publication supersedes ATP 4-02.85/NTRP 4-02.22/AFTTP(I) 3-2.69, dated 19 August 2015.

# FOREWORD

This publication has been prepared under our direction for use by our respective commands and other commands as appropriate.

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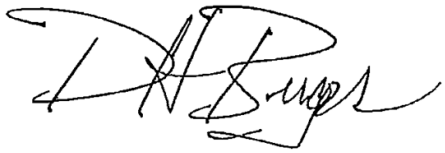

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Change 1

Army Techniques Publication No. 4-02.85
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Navy Technical Reference Publication 4-02.22
Air Force Tactics, Techniques, and Procedures (Instruction)
3-2.69

Headquarters
Department of the Army
Washington, DC, 16 April 2019

# Multi-Service Tactics, Techniques, and Procedures for Treatment of Chemical Warfare Agent Casualties and Conventional Military Chemical Injuries

1. This change updates pyridostigmine bromide (PB) use in accordance with Food and Drug Administration (FDA)-approved labeling and updates the Marine Corps number to ATP 4-02.85/MCRP 3-40A.1/NTRP 4-02.22/AFTTP(I) 3-2.69, *Multi-Service Tactics, Techniques, and Procedures for Treatment of Chemical Warfare Agent Casualties and Conventional Military Chemical Injuries*.

2. Change Army Techniques Publication (ATP) 4-02.85/MCRP 4-11.1A/NTRP 4-02.22/AFTTP(I) 3-2.69, dated 2 August 2016, as follows:

| Remove Old Pages | Insert New Pages |
|---|---|
| Front cover | Front cover |
| Foreword | Foreword |
| ii | ii |
| page 1-9 | page 1-9 |
| page 3-10 | page 3-10 |
| pages 3-18 through 3-20 | pages 3-18 through 3-20 |
| pages E-1 through E-3 | pages E-1 through E-3 |
| page F-3 | page F-3 |
| page H-2 | Page H-2 |
| Glossary-2 | Glossary-2 |
| Index-1 | Index-1 |

3. New or changed material is indicated by a bar mark ( ❙ ).

4. File this transmittal sheet in front of the publication.

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**16 April 2019**

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Maxwell Air Force Base, Alabama

2 August 2016

# Multi-Service Tactics, Techniques, and Procedures for Treatment of Chemical Warfare Agent Casualties and Conventional Military Chemical Injuries

## Contents





***This publication supersedes ATP 4-02.85//NTRP 4-02.22/AFTTP(I) 3-2.69, dated 19 August 2015.**

# Figures

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# Preface

## PURPOSE

This multi-Service publication provides tactics, techniques, and procedures and is designed for use as a reference for trained members of the Armed Forces Medical Services and other medically qualified personnel on the recognition and treatment of chemical warfare (CW) agent casualties and conventional military chemical injuries. Additionally, this publication provides information on first aid (self-aid and buddy aid) and enhanced first aid (combat lifesaver [United States (U.S.) Army and U.S. Marine Corps]) for these casualties.

## SCOPE

This publication classifies and describes CW agents and other hazardous chemicals associated with military operations, and describes how to diagnose and treat conventional military chemical injuries (for example, riot control agents, obscurants, incendiary agents, and other toxic industrial chemicals [TICs]). Further, this publication—

- Describes procedures for recognizing chemical agent casualties (Appendix A).
- Describes measures for handling contaminated clothing and equipment at medical treatment facilities (MTFs) (Appendix B).
- Describes medical management and treatment in chemical environment operations (Appendix C).
- Describes procedures for individual skin protection and decontamination (Appendix D).
- Describes procedures for administering nerve agent antidotes (Appendix E).
- Provides an immediate/emergency treatment ready reference for the treatment of CW agents and some TICs (Appendix F).
- Provides information regarding the Department of Defense (DOD) four-tier system for determining the levels of identification of CW agents (Appendix G).
- Provides information regarding protection and treatment of military working dogs (MWDs) (Appendix H).

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 this publication.

## APPLICABILITY

The principal audience for this publication is the members of the Armed Forces Medical Services and other medically qualified personnel.

Commanders, staffs, and subordinates ensure their decisions and actions comply with applicable U.S., international, and, in some cases, host-nation laws and regulations. Commanders at all levels ensure their Service members operate in accordance with the law of war and the rules of engagement. (See Field Manual [FM] 27-10.)

This publication uses joint terms where applicable. Selected joint and Army terms and definitions appear in both the glossary and the text. Terms for which this publication is the proponent publication (the authority) are italicized in the text and are marked with an asterisk (*) in the glossary. Terms and definitions for which this publication is the proponent publication are boldfaced in the text. For other definitions shown in the text, the term is italicized and the number of the proponent publication follows the definition.

This publication is in consonance with the following North Atlantic Treaty Organization (NATO) standardization agreements (STANAGs):

| TITLE | NATO STANAG |
|---|---|
| Medical Support Planning for Nuclear, Biological, and Chemical (NBC) Environments | 2478 |
| NATO Planning Guide for the Estimation of Chemical, Biological, Radiological, and Nuclear (CBRN) Casualties—Allied Medical Publication (AMedP)-8(C) | 2553 |
| Concept of Operations of Medical Support in Chemical, Biological, Radiological, and Nuclear Environments—AMedP-7(D) | 2873 |
| Principles of Medical Policy in the Management of a Mass Casualty Situation | 2879 |
| Training of Medical Personnel for CBRN Defence—AMedP-7.3 | 2954 |

This publication applies to the Active Army, Army National Guard/Army National Guard of the United States, and United States Army Reserve unless otherwise stated.

The proponent and the preparing agency of ATP 4-02.85/MCRP 4-11.1A/NTRP 4-02.22/AFTTP(I) 3-2.69 is the United States Army Medical Department Center and School, United States Army Health Readiness Center of Excellence (USAMEDDC&S, HRCoE). Send comments and recommendations on a DA Form 2028 (Recommended Changes to Publications and Blank Forms) to **Commander, USAMEDDC&S, HRCoE, ATTN: MCCS-FDL (ATP 4-02.85), 2377 Greeley Road, Building 4011, Suite D, JBSA Fort Sam Houston, Texas 78234-7731**; by e-mail to usarmy.jbsa.medcom-ameddcs.mbx.ameddcs-medical-doctrine@mail.mil; or submit an electronic DA Form 2028. All recommended changes should be keyed to the specific page, paragraph, and the line number. A rationale for each proposed change is required to aid in the evaluation and adjudication of each comment.

# IMPLEMENTATION PLAN

Participating Service command offices of primary responsibility will review this publication, validate the information and, where appropriate, reference and incorporate it in Service manuals, regulations, and curricula.

## UNITED STATES ARMY

The U.S. Army will incorporate this publication in U.S. Army training and doctrinal publications as directed by the Commander, U.S. Army Training and Doctrine Command.

## UNITED STATES MARINE CORPS*

The United States Marine Corps (USMC) will incorporate the procedures in this publication in USMC training and doctrinal publications as directed by the Deputy Commandant for Combat Development and Integration. Distribution is according to USMC publication distribution.

## UNITED STATES NAVY

The U.S. Navy will incorporate these procedures in U.S. Navy training and doctrinal publications as approved by the U.S. Navy Surgeon General and directed by the Commander, Navy Warfare Development Command. Distribution is according to Military Standard Requisitioning and Issue Procedures Desk Guide, Navy Supplement Publication (NAVSUP P-) 409.

## UNITED STATES AIR FORCE

The U.S. Air Force will validate and incorporate appropriate procedures according to applicable governing directives. Distribution is according to Air Force Instruction (AFI) 33-360.

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# USER INFORMATION

## THE U.S. ARMY MEDICAL DEPARTMENT CENTER AND SCHOOL

The USAMEDDC&S, HRCoE, is the proponent of this publication and developed this publication with the joint participation of the approving Service commands.

## SERVICE AND JOINT DOCTRINE

This publication reflects current Service and joint doctrine on prevention, protection, and medical management of CW agent casualties.

## RECOMMENDED CHANGES

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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# Introduction

Army Techniques Publication 4-02.85 remains generally consistent with the last version of this publication on key topics while adopting updated terminology and concepts as necessary. Key topics include recognition and treatment of CW agent casualties and conventional military chemical injuries. This publication also provides information on first aid (self-aid and buddy aid) and enhanced first aid (combat lifesaver [U.S. Army and U.S. Marine Corps]) for these casualties.

Summary of changes include—

- Adding the U.S. Marine Corps as a signatory to this publication. This publication supersedes ATP 4-02.85 dated 19 August 2015.
- Clarifying the difference between Antidote Treatment Nerve Agent, Autoinjector (ATNAA) and atropine injector.
- Clarifying the 2-pralidoxime chloride (2-PAM C1) usage as to when it is used as part of an atropine (combined) administration and used as a single drug administration.

Army Techniques Publication 4-02.85 consists of 10 chapters—

- Chapter 1 provides information on the threat, military employment, and classification of chemical warfare agents.
- Chapter 2 discusses protection, pathology, symptoms, diagnosis, and treatment of choking (lung-damaging) agents.
- Chapter 3 describes effects, prevention, symptoms, diagnosis, and treatment of nerve agents.
- Chapter 4 discusses protection, pathology, symptoms, diagnosis, and treatment of blood (cyanide) agents.
- Chapter 5 provides information on protection, properties, effects, symptoms, and treatment of blister (vesicant) agents.
- Chapter 6 describes diagnosis, protection, and treatment of incapacitating agents.
- Chapter 7 discusses protection, properties, effects, diagnosis, and treatment of riot control agents.
- Chapter 8 describes properties, pathology, symptoms, and treatment of different types of obscurants.
- Chapter 9 provides information on protection and treatment of different types of incendiary agents.
- Chapter 10 discusses properties, pathology, symptoms, diagnosis, and treatment of different types of toxic industrial chemicals.

## PROGRAM PARTICIPANTS

The following commands and agencies participated in the development of this publication:

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# Chapter 1
# Chemical Warfare Agent Casualties

## GENERAL

1-1. Chemical warfare agents remain a significant and continuing threat to the military forces. Delivery of CW agents may be accomplished through conventional or nonconventional means, causing extensive injury and contamination. 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 188 States Parties as of August 2012, will take many years to fully implement. Not all countries have signed the CWC. Countries such as Angola, Egypt, North Korea, Somalia, South Sudan, and Syria have neither signed, ratified, nor acceded to the CWC. However, in 2013, the Syrian government agreed to destroy their chemical weapon stockpile under the timetable of the Organisation for the Prohibition of Chemical Weapons, which is working with the United Nations to carry out the disarmament. In spite of the CWC and other diplomatic efforts, CW agents will be available to threat forces in regions where military forces may be deployed.

1-2. Chemical warfare agents are readily obtainable. The ease of obtaining these agents greatly increases the complexity and extent of the total threat. For example, nonmilitary organophosphate insecticide factories may also be used to produce nerve agents. 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 U.S. force projection. Chemical warfare agents and TICs (such as chlorine, phosgene, and cyanide) can also be encountered in a variety of situations off the battlefield.

## MILITARY EMPLOYMENT OF CHEMICAL WARFARE AGENTS

1-3. Chemical warfare agents can be tactically used anywhere within the range of current delivery systems. Chemical warfare agents can be used in conjunction with other agents 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; however, special design of such equipment and/or structures can prevent CW agent penetration.

1-4. The presence or threat of CW agent operations can create psychological and physiological problems, adversely affect morale, and reduce military or civilian efficiency. Chemical weapons may be employed with obscurants. Therefore, friendly forces must be prepared for chemical attacks when the enemy is employing obscurants.

1-5. All Service members must take every precaution against becoming chemical casualties. Medical personnel and Service members must apply the principles of first aid and chemical decontamination contained in this publication to protect themselves and increase their patients' chances for survival and recovery.

## ROUTES OF ENTRY

1-6. Chemical warfare agents may enter the body by several routes. When inhaled, gases, vapors, and aerosols may be absorbed via the respiratory tract. Absorption may occur through the mucosa of the upper and lower airway to include the nose, mouth, throat and/or the alveoli of the lungs. Vapor, liquid droplets, and solid particles can be absorbed by the surface of the skin, eyes, and mucous membranes. Chemical warfare agents that contaminate food and water can be absorbed through the gastrointestinal tract.

Wounds or abrasions are presumed to be more susceptible to absorption than the intact skin. Additional factors which affect absorption include occlusion of contaminated skin and warm and moist environments.

# CLASSIFICATION OF CHEMICAL WARFARE AGENTS

1-7. Chemical warfare agents are classified by either their physiological action or their military use. They are also classified as traditional, nontraditional, CW agent precursors and by-products, military compounds, and TICs.

1-8. Refer to table 1-1 for a list of chemical agents, obscurants, riot control agents, and TICs discussed in this publication with their acronym/chemical symbol.

**Table 1-1. Chemical agents, obscurants, riot control agents, toxic industrial chemicals, and their acronym/chemical symbol**

| *Acronym/chemical symbol* | *Chemical agents, obscurants, riot control agents, toxic industrial chemicals* |
|---|---|
| AC | hydrogen cyanide |
| BAL | British anti-Lewisite |
| BZ | 3-quinuclidinyl benzilate |
| CA | bromobenzyl cyanide |
| CG | phosgene |
| CK | cyanogen chloride |
| Cl | chlorine |
| CN | chloroacetophenone |
| CO | carbon monoxide |
| CR | dibenz(b,f)-1,4-oxazepine |
| CS | O-chlorobenzylidene malononitrile |
| CX | phosgene oxime |
| FS | sulfur trioxide-chlorosulfonic acid |
| GD | soman |
| H | impure sulfur mustard/Levinstein mustard |
| HC | zinc oxide and hexachloroethane |
| HD | distilled sulfur mustard |
| HN | nitrogen mustard |
| L | Lewisite |
| $NH_3$ | ammonia |
| $NO_x$ | oxides of nitrogen |
| OC | oleoresin capsicum |
| VX | O-ethyl methyl phosphonothiolate |
| WP | white phosporous |

## TRADITIONAL CHEMICAL WARFARE AGENTS AND THEIR PHYSIOLOGICAL ACTION

1-9. **A *choking agent* is a chemical warfare agent which produces irritation to the eyes and upper respiratory tract and damage to the lungs, primarily causing pulmonary edema.** A choking agent is also known as lung-damaging agent. Choking (lung-damaging) agents include phosgene (CG), diphosgene, chlorine, and chloropicrin. These agents produce injury to the lungs, attack the breathing passages and lungs, and irritate the eyes and the respiratory tract. They may also cause noncardiogenic pulmonary edema and predispose to secondary pneumonia.

1-10. *Nerve agent* is defined as a potentially lethal chemical agent that interferes with the transmission of nerve impulses (Joint Publication [JP] 3-11). Nerve agents (anticholinesterases), such as tabun, sarin, soman (GD), cyclosarin, and V-agents (for example, O-ethyl methyl phosphonothiolate [VX] and VR [Russian equivalent to VX]), inhibit the cholinesterase enzymes. The cholinesterase enzymes hydrolyze acetylcholine which is a chemical neurotransmitter. Inhibition of the cholinesterase enzymes creates an accumulation of acetylcholine at cholinergic synapses which results in an over stimulation 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.

1-11. *Blood agent* is defined as a chemical compound, including the cyanide group, that affects bodily functions by preventing the normal utilization of oxygen by body tissues (JP 3-11). 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. Toxicity with these agents leads to cessation of respiration followed by cardiovascular collapse.

1-12. *Blister agent* is defined as a chemical agent that injures the eyes and lungs, and burns or blisters the skin. Also called vesicant agent (JP 3-11). Blister (vesicant) agents include—the mustards (distilled sulfur mustard [HD] and nitrogen mustard [HN]), the arsenicals (Lewisite [L]), and the urticants (phosgene oxime [CX]). Blister (vesicant) agents produce pain and injury to the eyes, reddening and blistering of the skin (except for CX which produces solid lesions resembling welts or hives unlike mustard and L which produce fluid-filled blisters), and when inhaled, damage the mucous membranes and respiratory tract. These agents may produce major destruction of the epidermal layer of the skin.

1-13. *Incapacitating agent* is defined as a chemical agent which produces temporary disabling conditions that can be physical or mental and persist for hours or days after exposure to the agent has ceased (JP 3-11). Although a variety of different types of chemicals are classified as incapacitating agents, 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 of nerve agents and include erythema, decreased salivation, urinary retention, mydriasis (dilation of the pupils with decreased visual acuity), hyperthermia, and mental status changes.

## NONTRADITIONAL AGENTS

1-14. Nontraditional agents are chemicals and biochemicals reportedly researched or developed with potential application or intent as CW agents, but which do not fall in the category of traditional chemical agents or toxic industrial materials.

## CHEMICAL WARFARE AGENT PRECURSORS AND BYPRODUCTS

1-15. *Chemical agent* is defined as a chemical substance that is intended for use in military operations to kill, seriously injure, or incapacitate mainly through its physiological effects (JP 3-11).

1-16. *Chemical warfare* is defined as all aspects of military operations involving the employment of lethal and incapacitating munitions/agents and the warning and protective measures associated with such offensive operations (JP 3-11).

1-17. *Chemical weapon* is defined as together or separately, (a) a toxic chemical and its precursors, except when intended for a purpose not prohibited under the Chemical Weapons Convention; (b) a munition or device, specifically designed to cause death or other harm through toxic properties of those chemicals specified in (a), above, which would be released as a result of the employment of such munition or device; (c) any equipment specifically designed for use directly in connection with the employment of munitions or devices specified in (b), above (JP 3-11).

1-18. Chemical warfare agent precursors and by-products are those chemicals that are precursors to, or that in some cases can themselves be used as chemical weapons. These chemical precursors and by-products have a number of other commercial uses such as ingredients in resins, flame-retardants, additives, inks and dyes, insecticides, herbicides, lubricants, and some raw materials for pharmaceutical products.

### MILITARY COMPOUNDS

1-19. *Riot control agent* is defined as any chemical, not listed in a schedule of the Convention on the Prohibition of the Development, Production, Stockpiling and Use of Chemical Weapons and on Their Destruction that can produce rapidly in human sensory irritation or disabling physical effects that disappear within a short time following termination of exposure (JP 3-11). Riot control agents, herbicides, obscurants, incendiaries, and heavy metals (such as depleted uranium) are combat multipliers. Their use provides tactical advantages for offensive and defensive operations. For example, obscurants have long been employed as a means of concealing battlefield targets. Fire damage causes casualties and material damage and can impact psychologically.

### TOXIC INDUSTRIAL CHEMICALS

1-20. *Toxic industrial chemical* is defined as a chemical developed or manufactured for use in industrial operations or research by industry, government, or academia that poses a hazard (JP 3-11). Toxic industrial chemicals are found in abundance in all countries where Service members are deployed and are also found within the U.S. Toxic industrial chemicals are frequently found in areas with production facilities for petroleum, textiles, fertilizers, pesticides, plastics, as well as other products. Different chemicals amounting to billions of tons of material are produced, processed, or consumed by the global chemical industry. When burned, mixed, or exploded, many TICs produce additional highly toxic by-products.

### MILITARY USE

1-21. Chemical warfare agents produce serious injury or death. They include choking (lung-damaging) agents, nerve agents, blood (cyanide) agents, and blister (vesicant) agents.

1-22. Incapacitating agents produce temporary physical or mental effects or both. They include but are not limited to riot control agents, obscurants, and incendiary agents.

### FIELD BEHAVIORS

1-23. The field behavior of CW agents is dependent on weather variables such as wind, temperature, air stability, humidity, and precipitation. The influence of each variable depends upon the situation and is locally influenced by topography, vegetation, and soil. Chemical warfare agents may appear in the field in different forms—vapors, aerosols, or liquids. To understand the impact of CW agents on the battlefield, the Service member must also understand how these agents are affected by weather and terrain. For more information on the field behavior of chemical agents, see JP 3-11.

## MEANS OF DELIVERY OF CHEMICAL WARFARE AGENTS

1-24. Chemical warfare agents can be dispersed by explosive shells, rockets, missiles, aircraft bombs, mines, spray devices, and through industrial accidents and sabotage. Water supplies have the potential for contamination by either water-soluble or miscible liquids or solids, although effective concentrations are difficult to maintain. Food supplies are also vulnerable to CW agent contamination. *Contamination* is defined as: 1. The deposit, absorption, or adsorption of radioactive material, or of biological or chemical agents on or by structures, areas, personnel, or objects. Also called fallout radiation. 2. Food and/or water made unfit for consumption by humans or animals because of the presence of environmental chemicals, radioactive elements, bacteria or organisms, the by-product of the growth of bacteria or organisms, the decomposing material or waste in the food or water (JP 3-11).

1-25. 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.

# DIAGNOSIS OF EXPOSURE TO CHEMICAL WARFARE AGENTS

1-26. Some agents have odors which may aid in their detection and identification (see tables 1-2a through 1-2c on pages 1-6 through 1-10), 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. 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 CW agent may become imperceptible. Standard detection devices are the most reliable means of identifying a CW 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.

1-27. Observations for signs and symptoms include the following:

- A brief history eliciting symptoms and their progression.
- Physical examination of the eyes (pupils, conjunctivae, and lids) and skin.
- 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 tables 1-2a through 1-2c on pages 1-6 through 1-10.

**Table 1-2a. Summary of selected chemical agents, obscurants, riot control agents, and toxic industrial chemicals effects (odor, mechanism, eyes, and nose and throat)**

| *Chemical* | *Agent* | *Odor* | *Mechanism* | *Eyes* | | | *Nose and throat* | |
|---|---|---|---|---|---|---|---|---|
| | | | | *Pupils* | *Conjunc-tival* | *Rest of eye* | | |
| Nerve | tabun<br>sarin<br>soman<br>cyclosarin<br><br>O-ethyl methyl phosphono-thiolate<br><br>VR (Russian version) | none, or faint sweetness, fruity or paint-like | anticholineste-rase agents | miosis | redness | pain (especially on focusing), dimness of vision, headache, lacrimation | increased salivation and rhinorrhea | |
| Blister (vesicant) | mustard<br><br><br><br><br>nitrogen mustard | garlic or horseradish, irritating stinky asphalt<br><br>none or fishy, irritating | vesicants, bone marrow depressant, alkylating agent, damages deoxyribonucleic acid | | redness, edema, irritation, gritty pain | edema of lids, pain, spasm of the eyelid, photophobia, lacrimation, corneal ulceration, and possibly scarring | swelling, irritation, ulceration, discharge, occasional edema of larynx, spasm of the larynx | |
| | Lewisite and other arsenical vesicants | fruity to geranium-like, very irritating | vesicants, arsenicals poisons | | prompt redness, edema, irritation | immediate burning sensation, iritis, corneal injury | prompt irritation | |
| | mustard/ Lewisite mixture | garlic-like | like Lewisite and mustard | like sulfur mustard, nitrogen mustard, and Lewisite | | | | |
| | phosgene oxime | unpleasant and irritating | powerful vesicant | | violently irritating, redness, edema | lacrimation, corneal injury with blindness | very irritating to mucous membranes | |
| Choking (lung-damaging) | phosgene | green corn, grass, or new-mown hay | choking (lung-damaging) agent | | irritation | lacrimation (after respiratory symptoms) | irritation | |
| | chlorine | pungent | irritant and blistering agent | | redness | eye irritation | damage to the epithelial lining with desquamation, swelling of the lungs | |
| Blood (cyanide) | hydrogen cyanide | faint, bitter almonds | interferes with oxygen utilization at cellular level | | | | | |
| | cyanogen chloride | very irritating | like hydrogen cyanide, lung irritant | | irritation | lacrimation | irritation | |
| Riot control | irritant agents: chloroace-tophenone<br><br>bromoben-zyl cyanide | irritating | local irritant | | redness, irritation | pain, blepharos-pasm, profuse lacrimation, photophobia | irritation, burning | tightness, burning |
| | irritant agents: O-chloroben-zylidene malononitrile<br><br>dibenz(b,f)-1,4-oxazepine | very irritating, pungent, pepper-like | local irritant | | intense irritation | pain, blepharos-pasm, profuse lacrimation, photophobia | irritation, burning, tightness | tightness, burning |
| Incapaci-tating | 3-quinuclidinyl benzilate | none | anticholinergic | mydriasis | | blurred vision | extreme dryness | extreme dryness |
| | D-lysergic acid diethylamide (LSD) | none | psychotomimetic | mydriasis | | | | |

**Table 1-2b. Summary of selected chemical agents, obscurants, riot control agents, and toxic industrial chemicals effects (skin, respiratory and gastrointestinal tracts, and cardiovascular and genitourinary systems)**

| *Chemical* | *Agent* | *Respiratory Tract* | *Skin* | *Gastrointestinal Tract* | *Cardiovascular system* | *Genitourinary system* |
|---|---|---|---|---|---|---|
| Nerve | tabun<br>sarin<br>soman<br>cyclosarin<br><br>O-ethyl methyl phosphono-thiolate | 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 | wide ranging heart rate from bradycardia to tachycardia, fluctuating blood pressure, and cardiac arrythmia | frequent micturition urinary incontinence |
| Blister (vesicant) | mustard<br><br>nitrogen mustard | slowly developing irritation, hoarseness, aphonia, cough, tightness, dyspnea, rales, pneumonia, fever, pulmonary edema in severe cases; hemorrhagic pulmonary edema in very severe cases | range is 1 to 48 hours (average is 4 to 8 hours) blister shows up after 4 hours after redness start | 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. Blister on first and second day. Pain worse and necrosis deeper than mustard. | diarrhea, nausea, vomiting, hepatic failure | shock after severe exposure, hemolytic anemia, hemoconcentration | renal failure |
| | mustard/ Lewisite mixture | like mustard, nitrogen mustard, and Lewisite | | | | |
| | phosgene oxime | rapid irritation, coughing; later, pulmonary edema | immediate severe irritation and intense pain. Within one 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) | | | |
| Choking (lung damaging) | 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 | shock after severe exposure, hypertension, and tachycardia | |

**Table 1-2b. Summary of selected chemical agents, obscurants, riot control agents, and toxic industrial chemicals effects (skin, respiratory and gastrointestinal tracts, and cardiovascular and genitourinary systems) (continued)**

| *Chemical* | *Agent* | *Respiratory Tract* | *Skin* | *Gastrointestinal Tract* | *Cardiovascular system* | *Genitourinary system* |
|---|---|---|---|---|---|---|
| Blood (cyanide) | hydrogen cyanide | rapid deep respiration followed rapidly by dyspnea, gasping, then cessation of respiration | initially pink (carbon monoxide) than usual; may change to cyanosis | nausea | profound hypertension | |
| | cyanogen chloride | irritation, cough, choking, dyspnea; pulmonary edema can be rapid | like hydrogen cyanide | | | |
| Riot control | irritant agents: chloroacetophenone<br><br>bromobenzyl cyanide | tightness and irritation if concentration is high | stinging (especially of face), occasional dermatitis, may blister | occasional vomiting | | |
| | irritant agents: O-chlorobenzylidene malononitrile<br><br>dibenz(b,f)-1,4-oxazepine | tightness in chest and difficulty breathing | stinging, occasional dermatitis, may blister | nausea and vomiting | | |
| Incapacitating | 3-quinuclidinyl benzilate | | dry, flushed | constipation | tachycardia, elevated blood pressure | urgency, urinary retention |
| | D-lysergic acid diethylamide (LSD) | | sweaty palms, cold extremities | | tachycardia | |

**Table 1-2c. Summary of selected chemical agents, obscurants, riot control agents, and toxic industrial chemicals effects (central nervous system, decontamination, and treatment)**

| *Chemical* | *Agent* | *Central nervous system* | *Other* | *Decontamination* | *Treatment* |
|---|---|---|---|---|---|
| Nerve | tabun<br>sarin<br>soman<br>cyclosarin<br><br>O-ethyl methyl phosphono-thiolate | apprehension, giddiness, insomnia, headache, drowsiness, difficulty concentrating, poor memory, confusion, slurred speech, ataxia, weakness, coma with areflexia, Cheyne-Stokes respiration, convulsions/seizure | fasciculations, easy fatigue, cramps, weakness (including respiratory muscles), flaccid paralysis | remove contaminated clothing. For skin, use soap and water or Reactive Skin Decontamination Lotion. For individual equipment use M295 kit. | pretreatment with pyridostigmine bromide for organophosphate nerve agents postexposure therapy:<br>(1) Cholinergic blockade- atropine<br>(2) Enzyme reactivation-oximes (2-PAM chloride)<br>(3) Anticonvulsant- diazepam<br>(4) Assisted ventilation<br>(5) Suction for respiratory secretions<br>(6) Check for airway, breathing, and circulation |
| Blister (vesicant) | mustard<br><br>nitrogen mustard | anxiety, depression | late depression of bone marrow. Malaise and prostration | for liquid contami-nation of eyes, initially irrigate with copious amounts of water; then at the field military treatment facility, with a sodium bicarbonate or saline eyewash. Remove contaminated clothing. For skin, use soap and water or Reactive Skin Decontamination Lotion. For individual equipment use M295 kit. | eyes: antibiotic steroid ointment/drops<br><br>skin: local dressings and antibiotics for infection<br><br>respiratory infection: intravenous antibiotic fluids |
| | Lewisite and other arsenical vesicants | anxiety, depression | systemic arsenic poisoning | like mustard and nitrogen mustard | like sulfur and nitrogen mustards. The antidote dimercaprol (British anti-Lewisite [BAL]), a heavy metal chelator, is not available through the United States military medical supply system, but may be available through coalition forces during international operations. |
| | mustard/ Lewisite mixture | like mustard, nitrogen mustard, and Lewisite | | | 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 |
| Choking (lung damaging) | phosgene | anxiety, depression | | | Consider prompt treatment with intravenous and inhalation corticosteroids. Rest, oxygen, antibiotics |
| Blood (cyanide) | hydrogen cyanide | may have initial excitation; then depression, giddiness, headache, irrational behavior, ataxia, convulsions or coma | | | (1) Drugs binding cyanide:<br>(a) Methemoglobin formers; nitrites<br>(b) Scavengers; hydroxocobalamin, and dicobalt edetate<br>(2) Provision of S-Groups, thiosulfate<br>(3) Assisted ventilation<br>(4) Oxygen |
| | cyanogen chloride | like hydrogen cyanide | | | like hydrogen cyanide and phosgene |

**Table 1-2c. Summary of selected chemical agents, obscurants, riot control agents, and toxic industrial chemicals effects (central nervous system, decontamination, and treatment) (continued)**

| *Chemical* | *Agent* | *Central nervous system* | *Other* | *Decontamination* | *Treatment* |
|---|---|---|---|---|---|
| Riot control | irritant agents: chloroaceto-phenone<br><br>bromobenzyl cyanide | headache | | wash eyes with copious amounts of water | spontaneous improvement. Analgesic eye and nose drops, if necessary |
| | irritant agents: O-chlorobenzyli-dene malononitrile<br><br>dibenz(b,f)-1,4-oxazepine | headache | | wash eyes with copious amounts of water | symptoms disappear rapidly in fresh air |
| Incapacita-ting | 3-quinuclidinyl benzilate | headache, giddiness, drowsiness, disorientation, hallucinations and occasional maniacal behavior. Ataxia or lack of coordination | | for contamination of skin, wash with soap and water | restraint, cool environment. Physostigmine. Treatment may be required over several days |
| | D-lysergic acid diethylamide (LSD) | mental excitation, poor concentration, tremor, indecisiveness, inability to act in a sustained or purposeful manner, hallucinations | pyrexia | | reassurance, restraint, prompt evacuation, diazepam |

## PROTECTIVE MEASURES AND HANDLING OF CHEMICAL WARFARE AGENT CASUALTIES

1-28. Mission-oriented protective posture (MOPP) 4 (consisting of wearing the protective overgarment, mask and hood, gloves, and overboots) will be assumed immediately—

- When the local alarm or command is given.
- When there is a positive reading (chemical agent monitor or detector paper/tape).
- When entering an area known to be or suspected of being contaminated with CBRN hazards.
- 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.

1-29. In addition to above conditions, the mask should be put on immediately upon detection of a CW agent and worn until the *all clear* signal is given by authorized personnel.

1-30. It is the responsibility of all individuals to decontaminate themselves or to decontaminate other personnel in their unit. Contaminated casualties may arrive at an MTF, presenting a hazard to unprotected personnel. Handlers must wear their individual protective equipment or appropriate individual protective equipment while handling these casualties. A patient decontamination area should be 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). For more information on patient decontamination, refer to ATP 4-02.7/MCRP 4-11.1F/NTTP 4-02.7/AFTTP 3-42.3.

1-31. Handling chemically contaminated patients presents a great challenge to the Military Health System. The vapor hazard associated with contaminated patients may require medical personnel to remain at

MOPP 4 for long periods; therefore, medical personnel must locate clean areas to set up their MTF. The MTF should operate in a contaminated environment only until medical personnel have the time and means to move to a clean area. When an MTF is expected to operate in a contaminated area, collective protective shelters (CPSs) must be used (see Appendix C and multi-Service tactics, techniques, and procedures for health service support [HSS] in a CBRN environment for more information).

1-32. Military commanders, leaders, and medical personnel should be on the alert for the possibility of anxiety reactions (combat and operational stress reactions among personnel following CW agent attacks). All possible steps must be taken to prevent or control anxiety situations.

1-33. 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.

1-34. Personnel in protective clothing are particularly susceptible to heat injury. Refer to Technical Bulletin (Medical) (TB MED) 507/Air Force Pamphlet 48-152 (I) for more information. Ambient temperature is considered when determining the degree of physical activity feasible in protective clothing. Wet bulb globe temperature 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 the ambient humidity. At MOPP 4, add 10° Fahrenheit (F), (5.6° Celsius [C]) to the wet bulb globe temperature index for light work. Refer to table 1-3 on page 1-12 for work and rest cycles and also water consumption chart.

**Table 1-3. Work/rest cycles and water consumption**

| *Heat category* | *WBGT index degrees Fahrenheit* | *Easy work* | | *Moderate work* | | *Hard work* | |
|---|---|---|---|---|---|---|---|
| | | *Work/rest minutes* | *Water intake quart/hour* | *Work/rest minutes* | *Water intake quart/hour* | *Work/rest minutes* | *Water intake quart/hour* |
| 1 (WHITE) | 78 to 81.9 | NL | ½ | NL | ¾ | 40/20 | ¾ |
| 2 (GREEN) | 82 to 84.9 | NL | ½ | 50/10 | ¾ | 30/30 | 1 |
| 3 (YELLOW) | 85 to 87.9 | NL | ¾ | 40/20 | ¾ | 30/30 | 1 |
| 4 (RED) | 88 to 89.9 | NL | ¾ | 30/30 | ¾ | 20/40 | 1 |
| 5 (BLACK) | > 90 | 50/10 | 1 | 20/40 | 1 | 10/50 | 1 |

- The work/rest times and fluid replacement volumes will sustain performance and hydration for at least 4 hours of work in the specified heat category. Fluid needs can vary based on individual differences (± ¼ quart/hour) and exposure to full sun or full shade (± ¼ quart/hour).
- Hourly fluid intake should not exceed 1 quart and daily fluid intake should not exceed 12 quarts.
- Rest means minimal physical activity while sitting or standing, accomplished in the shade if possible.

**Legend:**
NL no limit to work time per hour
WBGT wet bulb globe temperature

**CAUTION**

The toxicological agent protective apron adds an additional 10 degrees to the wet bulb globe temperature index.

Wearing protective overgarments adds 10°F (5.6°C) to the web bulb globe temperature index and if conducting moderate or hard work and wearing protective overgarments, add 20°F (10.2°C) to wet bulb globe temperature index.

Wearing body armor increases this by another 5°F (2.8°C).

A worker may produce as much as 2 to 3 gallons of sweat in the course of a day's work. Because many heat disorders/injuries involve excessive dehydration of the body, it is essential that water intake during the workday be about equal to the amount of sweat produced.

## CHEMICAL WARFARE AGENT CONTAMINATION DETECTION AND IDENTIFICATION

1-35. Identification of CW agents will greatly assist in the diagnosis and treatment of chemical injuries. Chemical warfare agent detector paper or tape (for example, M8, M9) can be used to detect/identify some liquid chemical agents. The following are means of detecting and identifying CW agent contamination:

- The chemical agent detector paper can be used to detect and identify liquid V- and G-type nerve agents and H-type blister (vesicant) agents. It does not detect CW agent vapors. Some solvents and standard decontaminating solutions cause false-positive reactions on the chemical agent detector paper. However, any color change may indicate a possible hazard.

- The chemical agent detector tape, which can be worn on the uniform, detects the presence of liquid nerve agents (V and G) and blister (vesicant) 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 can cause false-positive reactions on the chemical agent detector tape.
- Automatic CW agent alarm systems and chemical agent monitors detect agent aerosol and vapor contamination consistent with their design specifications and operational limitations. For more information on detailed listing of detectors/monitors, refer to the Department of the Army Pamphlet (DA PAM) 385-61.
- Detector kits (for example, M256A1) detect vapor concentrations of nerve, blister, and blood (cyanide) agents.

## Medical Management

1-36. Medical management consists of those procedures for optimizing medical care to ensure the maximum return to duty on the battlefield. This includes triage, basic medical treatment, decontamination, emergency medical treatment, advanced trauma management, evacuation, and continuing protection of CW agent casualties.

## Immediate Decontamination

1-37. When an individual becomes contaminated with a CW agent, immediate decontamination must be carried out at the time of exposure. For those individuals who cannot decontaminate themselves, the nearest able person should assist them as the situation permits. Immediate decontamination consists of decontamination of the eyes, skin decontamination by either agent removal and/or by agent neutralization (agent removal is preferred), and equipment decontamination (as soon as situation permits; preferably within 15 minutes of exposure). Refer to Appendix D for immediate decontamination procedures.

## Casualty Decontamination

1-38. Contaminated casualties entering the medical treatment system are decontaminated through a decentralized process. Units decontaminate the casualty before evacuation, that is, if patient status, situation, and time permit. Immediate and operational decontamination of the casualty should be accomplished (mission, enemy, terrain and weather, troops and support available, time available, and civil considerations-dependent). Patient decontamination stations are established at all roles of care to decontaminate individuals as required prior to entry into MTF. Medical supervision is required to prevent further injury to the casualty and to provide emergency medical treatment 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. Nonmedical augmentees are usually required to perform patient decontamination while supervised by medical 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.

## First Aid

1-39. First aid is comprised of self-aid, buddy aid, or enhanced first aid provided by those nonmedical personnel trained as combat lifesavers (U.S. Army/U.S. Marine Corps).

### *Self-Aid*

1-40. Self-aid consists of measures that Service members can apply in helping themselves. These include individual decontamination, administration of antidotes (only for nerve agent exposure), and assumption of the appropriate individual protective equipment.

### *Buddy Aid or Combat Lifesaver Aid*

1-41. Buddy aid or combat lifesaver aid consists of emergency actions to restore or maintain vital body functions in a casualty who cannot administer 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. The nearest individual may need to follow these steps:

- Put on mask first, then mask the casualty (if not already masked).
- Administer antidotes (only for nerve agent exposure).
- Decontaminate the casualty.
- Put remaining protective clothing on the casualty to preclude further absorption of contamination through any exposed skin.
- Evacuate the casualty as soon as possible.

## Medical Treatment

1-42. Medical treatment consists of those procedures undertaken to return injured or ill Service members to duty, to save life and limb, and to stabilize the patient for evacuation to the next role of medical care. Specific CW agent treatment procedures are described in the ensuing chapters.

## Medical Evacuation

1-43. Casualties requiring evacuation should be decontaminated, if possible, before evacuation. For more information on levels of decontamination see ATP 3-11.32/Marine Corps Warfighting Publication (MCWP) 3-37.2/Navy Tactics, Techniques, and Procedures (NTTP) 3-11.37. In many instances, the casualty must be evacuated to the first role of care before decontamination is completed. Ground ambulances are the preferred means to evacuate the casualties in contaminated forward areas, when feasible. This does not mean that medical evacuation by air ambulance should not be used. When used, the number 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.

1-44. During mass casualty situations, commanders may be required to employ nonmedical vehicles/aircraft for casualty evacuation. En route care is not available for casualty evacuation. If medical personnel augmentation is available, limited en route care may be available. Refer to STANAG 2879 for more information on principles of medical policy in the management of a mass casualty situation.

1-45. For detailed information on medical evacuation see JP 4-02, ATP 4-25.13, and multi-Service tactics, techniques, and procedures for HSS in a CBRN environment.

## Individual Prescriptions

1-46. All Force Health Protection (FHP) Prescription Products 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); the Surgeon Generals of the Army, Navy, or Air Force; The Medical Officer, U.S. Marine Corps; or the command surgeon of a combatant command. Although the inclusive list of FHP Prescription Products may vary between areas of responsibility based on differing threats, examples of such products include atropine and the 2-PAM C1 autoinjectors; convulsant antidote for nerve agent (CANA) (diazepam); certain antimicrobials, including antimalarials; and pyridostigmine bromide (PB). The provision or issuance of FHP Prescription Products shall be documented in medical records of all personnel or other individuals receiving these products. For more information, refer to Assistant Secretary of Defense (Health Affairs) Policy Memorandum 03-007, dated 24 April 2003.

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***Note***. The CANA (diazepam) will be replaced by the Advanced Anticonvulsant System (midazolam).

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## Investigational New Drugs and Emergency Use Authorization

1-47. Department of Defense Instruction (DODI) 6200.02 instructs that, when there is the need for a FHP program for a medical countermeasure against a particular threat and no satisfactory Food and Drug Administration (FDA)-approved medical product is available, the Heads of DOD components may request approval by the Assistant Secretary of Defense (Health Affairs) to use an unapproved product under an

emergency use authorization (EUA) or, if EUA is not feasible, under an investigational new drug (IND) application. Such requests must—

- Be justified based on available evidence of the safety and efficacy of the medical product and the nature and degree of the threat to personnel.
- Document a high threat for which the use of a drug under EUA or IND application is needed, consideration of the risks and benefits of use of the drug involved, and compliance with the requirement of DODI 6200.02.
- Be coordinated with the Chairman of the Joint Chiefs of Staff (and if from the Commander of a combatant command, be submitted through the Chairman of the Joint Chiefs of Staff), the Secretary of the Army as Lead Component, and the General Counsel of the DOD.

1-48. When using medical products under the FHP program pursuant to an EUA, the Heads of DOD components shall comply with the U.S. Code, Title 21, *Food and Drugs*, Chapter 9, *Federal Food, Drug, and Cosmetic Act*, Subchapter V, Part E, Section 360bbb-3, *Authorization for Medical Products for Use in Emergencies*, and applicable FDA requirements.

1-49. The Secretary of the Army, as Lead Component for development of medical protocols and regulatory submissions to the FDA shall—

- In concert with the Head of the DOD components involved and the Assistant Secretary of Defense (Health Affairs), develop a specific medical protocol, including appropriate record keeping and reporting of adverse events, and required FDA regulatory submissions for use of the medical product under EUA or IND application.
- Ensure that the Army Medical Research and Materiel Command Human Subjects Research Review Board, under the Surgeon General of the Army, carries out the responsibilities as the designated Institutional Review Board according to DODI 6200.02.
- In cases when the medical product has a similar potential use by the Centers for Disease Control and Prevention to protect the public's health from bioterrorism or other threats, consult with Centers for Disease Control and Prevention officials on the potential for collaborative action in pursuing an EUA or IND application.
- Prepare annually, in coordination with the Secretaries of the Military Departments and the Chairman of the Joint Chiefs of Staff, a plan for using medical products under EUAs or IND protocols under FHP programs against health threats when there is no satisfactory approved medical product available. This plan shall establish responsibilities and action timelines to make the best possible medical products available.

1-50. For more information on notice of use of an IND or a drug unapproved for its applied use, refer to U.S. Code, Title 10, *Armed Forces*, Subtitle A, Part II, Chapter 55, Section 1107, *Notice of Use of an Investigational New Drug or a Drug Unapproved for its Applied Use*. For more information on use of FDA-regulated investigational products in humans including Schedule I controlled substances, refer to Army Regulation (AR) 40-7.

## Health Surveillance

1-51. All personnel who have been deployed are subject to postdeployment health assessments according to DOD and component Service guidance. In the event that personnel have been exposed to CW agents, including TICs, during deployed operations, they will be afforded additional postdeployment aftercare treatment and evaluation as indicated. For more information on postdeployment health assessment process, see DODI 6490.03. For more information on comprehensive health surveillance, refer to DOD Directive (DODD) 6490.02E.

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# Chapter 2
# Choking (Lung-Damaging) Agents

## GENERAL

2-1. Chemical warfare agents that primarily cause pulmonary edema by attacking lung tissue have traditionally been classified as choking (lung-damaging) agents or pulmonary edematogenic agents. They include CG, diphosgene, chlorine, and chloropicrin. The best known of these agents is CG. There are also numerous TICs or products of combustion that pose a primary threat similar to choking (lung-damaging) agents. Obscurants are covered in Chapter 8 and Chapter 10 (including chlorine and oxides of nitrogen [NOx]).

2-2. 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 central airways. The terms choking (lung-damaging) agents and respiratory irritants are sometimes ambiguous and are not as 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. Most pulmonary agents in high doses will affect both the central and peripheral compartments.

## CENTRAL PULMONARY AGENTS

2-3. 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.

2-4. 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.

2-5. 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. Impure sulfur mustard (H) or Levinstein mustard, even though officially classified as a vesicant, can be regarded as the prototypical central pulmonary CW agent.

### PATHOPHYSIOLOGY

2-6. After dissolving in aqueous solutions, central pulmonary agents typically act as acids and damage or 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.

### CLINICAL PRESENTATION

2-7. Identification of a particular CW agent is important mainly as a means of predicting, identifying, and managing central versus peripheral pulmonary damage. Central pulmonary agents produce irritation (a symptom) of the airways and sounds indicative of airway dysfunction (a sign). The clinical hallmark of central damage to the central compartment is characteristic airway sounds. Casualties may cough, sneeze, become hoarse, exhibit inspiratory stridor, or develop coarse rhonchi or wheezing. In severe cases, irritation

may lead to obstruction of the airway from reactive laryngospasm. For most central pulmonary agents, airway irritation and sounds occur relatively soon after exposure, although these effects may be delayed with slowly dissolving but extremely reactive agents such as HD.

### MANAGEMENT

2-8. 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 oxygen, 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.

## PERIPHERAL PULMONARY AGENTS

2-9. 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 (the portion of the respiratory tract distal to the terminal bronchioles).

2-10. 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.

2-11. The World War I agents CG and diphosgene are relatively insoluble, relatively chemically unreactive, and exhibit peripheral effects at low to moderate concentrations. Perfluoroisobutylene (a high-temperature combustion product of polytetrafluoroethylene); isocyanates; NOx; and hexachloroethane, grained aluminum, and zinc oxide (HC) obscurants 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 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.

### PATHOPHYSIOLOGY

2-12. 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 obscurants 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).

### CLINICAL PRESENTATION

2-13. Identification of a particular CW agent is important mainly as a means of predicting, identifying, and managing central versus 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 (airway sounds), 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 NOx or HC obscurants are at risk of late-onset pulmonary fibrosis heralded by cough, fever, chills, dyspnea, cyanosis, and radiological evidence of cryptogenic organizing pneumonia.

### MANAGEMENT

2-14. 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 NOx or HC obscurant should be considered since these agents appear capable of inducing late-onset pulmonary fibrosis by immunological means.

### PROTECTION

2-15. The protective mask or a collective protection system gives protection against military choking (lung-damaging) agents. High concentrations of certain lung-damaging industrial chemicals (such as $NH_3$, chlorine, and carbon monoxide [CO]) may defeat the filters of the field protective mask.

## PROPERTIES OF PHOSGENE

2-16. 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°F (8.3°C) and its vapor density is 3.4 times that of air. It is extremely volatile making it a nonpersistent CW agent; CG's vapor density (heavier than air) may cause it to 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 and carbon dioxide.

### PATHOLOGY

2-17. 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 chloropicrin 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 NOx and HC obscurants, 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).

## SYMPTOMS

2-18. During and immediately after exposure, there may either be no symptoms at all or, at moderate to high doses, coughing, choking, and 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.

## DIAGNOSIS

2-19. 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.

## PROGNOSIS

2-20. 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.

## SELF-AID

2-21. When a CG attack is suspected, the protective mask should be put on immediately. Other indications of a CG attack are—

- Odor like newly mown hay, garlic, or bitter almonds (do not rely upon odor as an indication of a chemical attack).
- Irritation of the eyes.

2-22. 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 Service members should be kept at rest, evaluated, and promptly evacuated if the operational situation permits.

2-23. If potentially affected Service members develop 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). Those Service members who are at high likelihood of exposure should be kept at rest, observed, and promptly evacuated even if they are not yet clinically symptomatic.

## TREATMENT

2-24. A casualty with potentially significant unprotected exposure to a choking (lung-damaging) agent should be kept at rest until the danger of pulmonary edema is past, if the operational situation permits.

### Rest and Warmth

2-25. 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.

### Sedation

2-26. Sedation should be used sparingly. Codeine in doses of 30 to 60 milligrams (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.

### Oxygen

2-27. Hypoxemia may be controlled by oxygen supplementation. Early administration of positive airway pressure (intermittent positive pressure breathing, continuous positive airway pressure mask, positive end-expiratory pressure mask, or, if necessary, intubation with or without a ventilator), may delay and/or minimize the pulmonary edema and reduce the degree of hypoxemia.

### Antibiotics

2-28. Antimicrobial therapy should be reserved for cases complicated by suspected bacterial bronchitis/pneumonitis modified by culture results if available. Prophylactic therapy is not indicated.

### Steroids

2-29. After exposure to a sufficiently high dose of CG or similar agent, pulmonary edema will follow. Steroids have been demonstrated to be useful for treatment of NOx and HC obscurants. 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 regimens 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.

2-30. Using dexamethasone-sodium phosphate—

- 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.
- 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 systemic 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.

2-31. Using beclomethasone dipropionate or betamethasone valerate (the differences occur due to the various absorption characteristics of these steroids and limited systemic therapy is necessary, even for precautionary treatment), the procedure is as follows:

- Treatment should commence as soon as possible with the inhalation of the steroid from an inhaler. Inhalational therapy is considered necessary for at least five days. Systemic therapy will be required as a 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 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.
- Pulmonary fibrosis is typical of damage caused only by NOx and HC obscurants. 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 infections. 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.

## Convalescent Care

2-32. 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

## GENERAL

3-1. 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.

3-2. 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, sarin, GD, and cyclosarin. V-agents include VX and VR.

3-3. Nerve agents can be dispersed by artillery shell, mortar shell, rocket, land mine, missile, aircraft spray, aircraft bomb or bomblet, or through passive evaporation as noted in the Tokyo subway attack.

3-4. Several related but somewhat less toxic compounds have proven to be useful in medicine and agriculture. For example, carbamates are among the most popular pesticides for home use. Carbaryl is perhaps the best known and most applied carbamate pesticide, used primarily for lawns and gardens.

## PHYSICAL AND CHEMICAL PROPERTIES

3-5. Nerve agents are colorless to light brown liquids. Some are volatile, while others are relatively nonvolatile 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. *Persistent agent* is defined as a chemical agent that, when released, remains able to cause casualties for more than 24 hours to several days or weeks (JP 3-11). *Nonpersistent agent* is defined as a chemical agent that when released dissipates and/or loses its ability to cause casualties after 10 to 15 minutes (JP 3-11). Thickening substances may be added to nonpersistent agents, reducing volatility and allowing these mixtures to remain in the environment for extended periods of time.

## ABSORPTION OF AND PROTECTION AGAINST NERVE AGENTS

3-6. 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. The respiratory tract (inhalation) is the most rapid and effective route of absorption. 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 (fasciculation). The local ocular effects from liquid exposure to the eye are similar to miosis and often, conjunctival hyperemia. Effects of liquid on mucous membranes include twitching or contracting of the underlying muscle and glandular secretions. Refer to table 3-1 on page 3-2 for route of exposure comparison.

**Table 3-1. Route of exposure comparison**

| | *Vapor (Unmasked)* | *Liquid* |
|---|---|---|
| ***Contact signs*** | Miosis | Localized (sweating, fasciculations) |
| ***Latent period*** | None | Delayed (up to 18 hours) |
| ***Earliest systemic effect*** | Respiratory | Nausea |
| ***Progression of illness*** | Limited | Sustained |

3-7. The protective mask and hood protect the face and neck, eyes, mouth, and respiratory tract against nerve agent spray, vapor, and aerosol. To prevent inhaling an incapacitating or lethal dose, one should close his/her eyes and stop breathing immediately and don the mask within nine seconds at the first warning of a nerve agent presence. Nerve agent vapor is absorbed through the skin very slowly, so proper masking may provide some protection against the effects of low vapor concentrations.

3-8. Liquid nerve agents rapidly penetrate ordinary clothing. Although absorption through the skin usually requires at 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 soap and water or reactive skin decontamination lotion (RSDL). Refer to Appendix D for more information. 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.

3-9. The MOPP 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.

# EFFECTS OF NERVE AGENTS

3-10. Nerve agents inhibit cholinesterase enzymes throughout the body.

## MECHANISM OF ACTION

3-11. Since the normal function of the cholinesterase 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 (see figure 3-1 on page 3-4).

3-12. Accumulation of acetylcholine at these sites results in characteristic signs and symptoms 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. Finally, accumulation of excessive acetylcholine in the brain and spinal cord results in characteristic CNS symptoms. Refer to table 3-2 for more information.

3-13. 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. Refer to table 3-2 for more information on signs and symptoms of nerve agent poisoning.

**Table 3-2. Signs and symptoms of nerve agent poisoning**

| *Site of action* | *Signs and symptoms* |
|---|---|
| | ***Following local exposure*** |
| ***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 bonchoconstriction 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 urination. |
| ***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. |
| ***Central nervous system*** | |
| Immediate (acute) effects | Generalized weakness, depression of respiratory and circulatory centers with dyspnea, cyanosis, and hypotension, convulsions, loss of consciousness, and coma. |
| Delayed (chronic) effects | Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, bursts of slow waves of elevated voltage in electroencephalography, especially on hyperventilation, drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, and ataxia. |

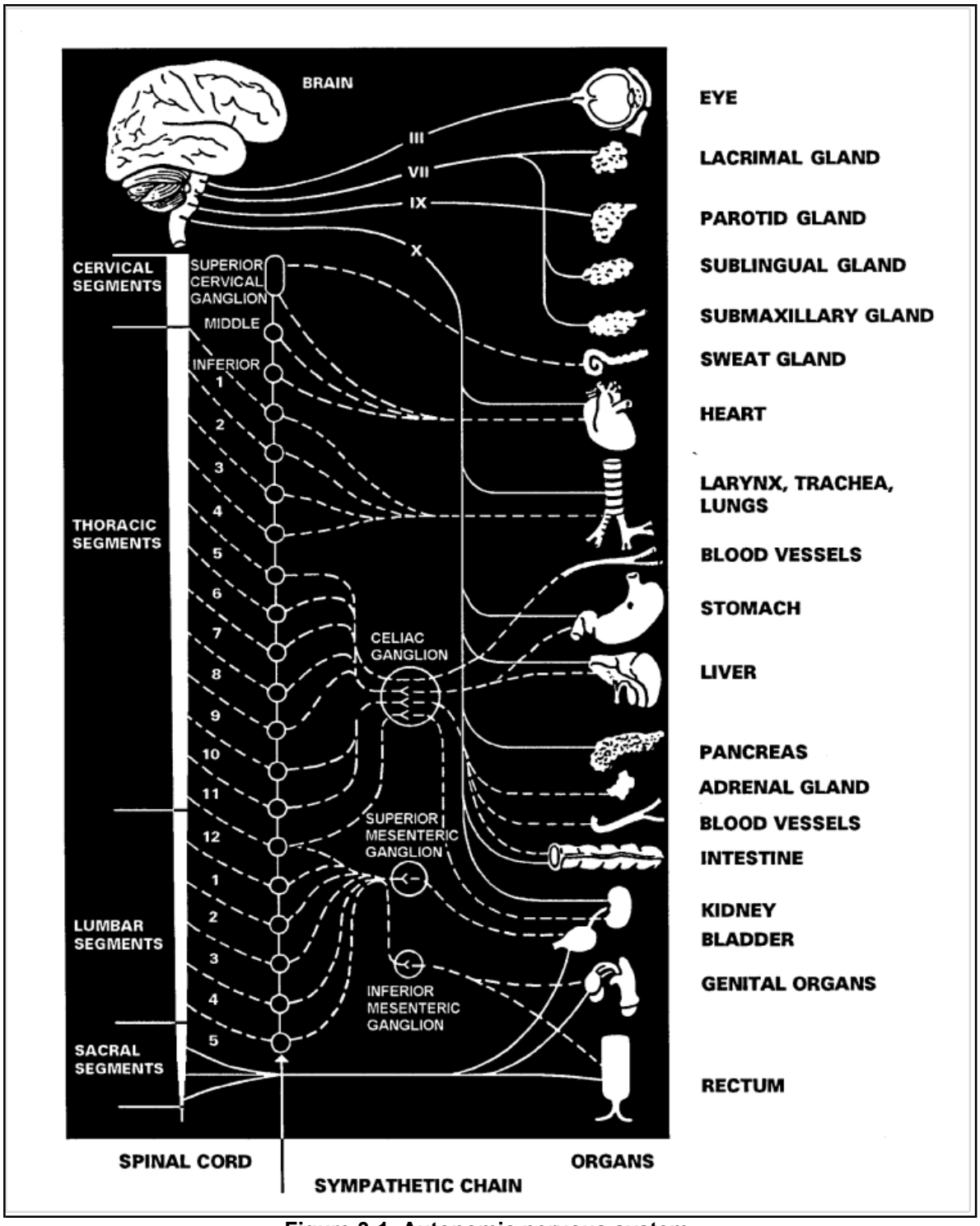


**Figure 3-1. Autonomic nervous system**

## PATHOLOGY

3-14. 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.

## EFFECTS OF VAPOR

3-15. 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.

### Local Ocular Effects

3-16. Local ocular 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, it 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.

### Local Respiratory Effects

3-17. The 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.

### Systemic Effects

3-18. 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 3-3 on page 3-6. 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.

### Muscarinic Effects

3-19. 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, sticky, and persistent. 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 3-3. 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* | *Duration of effects after 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 | Systematic | Respiratory or eyes | Muscarinic, nicotinic, and central nervous system effects | Less than one minute after moderate or severe exposure; about 30 minutes after mild exposure | Several hours to a day | Acute effects: 2 to 3 days<br><br>Central nervous system effects: days to weeks |
| Liquid | Local | Eyes | Same as vapor effects | Instantly | Similar to effects of vapor | – |
| Liquid | Local | Ingestion | Gastrointestinal | 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 | Systematic | Bronchial tree | Bronchoconstriction, cyanosis | Several minutes | – | 1 to 5 days |
| Liquid | Systematic | Eyes | Same as vapor | Several minutes | – | 2 to 4 days |
| Liquid | Systematic | Skin | Generalized sweating | 15 minutes to 2 hours | – | 2 to 5 days |
| Liquid | Systematic | Ingestion | Gastrointestinal | 15 minutes to 2 hours | – | 3 to 5 days |

**Note:** After lethal or near lethal exposures to nerve agents, the time to onset of symptoms and to maximal severity 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.

### Nicotinic Effects

3-20. Increased fatigability 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.

### Central Nervous System Effects

3-21. 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 confusion 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.

## EFFECTS OF LIQUID NERVE AGENT

3-22. This section will discuss effects of liquid nerve agent.

### Local Ocular Effects

3-23. 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, the blister agent, Lewisite). Bloody tears have been reported.

### Local Skin Effects

3-24. 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.

### Local Gastrointestinal Effects

3-25. Following the ingestion of substances containing a nerve agent (which is essentially tasteless), the initial symptoms include abdominal cramps, vomiting, and diarrhea.

### Systemic Effects

3-26. 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.

### TIME COURSE OF EFFECTS OF NERVE AGENTS

3-27. 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 to moderate effects from a tiny drop of VX to the skin may take up to 18 hours to appear.

### CUMULATIVE EFFECTS OF REPEATED EXPOSURE

3-28. 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.

### MECHANISM OF DEATH

3-29. Death is due to respiratory depression caused by four mechanisms—

- Bronchoconstriction.
- Increased respiratory secretions obstructing airways.
- Paralysis of respiratory muscles, especially the diaphragm.
- Central apnea or failure of the respiratory center in the brain.

3-30. When overwhelming doses of the agent are absorbed quickly, death occurs rapidly without an orderly progression of symptoms.

## CLINICAL PRESENTATION AND DIAGNOSIS OF NERVE AGENT POISONING

3-31. Nerve agent poisoning may be identified from the characteristic signs and symptoms. 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.

3-32. 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).

### MILD POISONING (SELF-AID)

3-33. Casualties with mild exposure 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.

### SEVERE SYMPTOMS (BUDDY AID)

3-34. 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).
- Apnea.
- Severely pinpoint pupils.
- Red eyes with tearing.
- Vomiting.
- Severe muscular twitching and general weakness.
- Involuntary urination and defecation.
- Convulsions.
- Unconsciousness.
- Respiratory failure.
- Tachycardia or bradycardia.

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***Note.*** Casualties with severe symptoms will not be able to treat themselves and must receive prompt buddy aid, combat lifesaver aid, and prompt follow-on medical treatment 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.

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3-35. The progress of symptoms from mild to severe indicates either inadequate treatment or continuing exposure to the agent.

## PREVENTION AND TREATMENT OF NERVE AGENT POISONING

3-36. The essential prevention and treatment elements of nerve agent poisoning are—

- Donning the protective mask and hood at the first indication of a nerve agent attack.
- Administering antidotes (ATNAA) as soon as any signs or symptoms are noted.
- Administering the CANA to severely poisoned casualties or those obviously seizing.
- Removing or neutralizing any liquid contamination immediately.
- Removing airway secretions if they are obstructing the airway. Airway suction may be needed.
- Removing mask and establishing an open airway (for example, endotracheal tube or cricothyroidotomy) 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.
- Administering supplemental oxygen as available.

## PREVENTION OF POISONING

3-37. 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. To prevent inhaling an incapacitating or lethal dose, immediately close eyes and stop breathing 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 time will occur before full signs and symptoms are present. 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.

3-38. 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 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 supplies are rapidly used up in the treatment of nerve agent poisoning and repeated doses may be necessary.

3-39. Pyridostigmine bromide (PB) (also known as Soman Nerve Agent Pretreatment Pyridostigmine or SNAPP) affords protection effects against organophosphate nerve agents when given as a pretreatment. As a stand-alone drug, PB does not protect against exposure to nerve agents. The efficacy of PB is dependent upon the rapid use of atropine and pralidoxime after exposure to nerve agents. Once the symptoms of nerve agent poisoning begin, do not be initiated PB or continue its use as this may significantly worsen symptoms and may lead to death. Refer to Assistant Secretary of Defense (Health Affairs) Policy Memorandum 03-011 for more information.

3-40. Nerve agents (liquid or vapor) can contaminate food and water. For details on the management and decontamination of food and water, see multi-Service tactics, techniques, and procedures for HSS in a CBRN environment.

## EFFECTS OF NERVE AGENT ANTIDOTES

3-41. Atropine sulfate remains a principal drug in the treatment of nerve agent poisoning. It blocks the effects of acetylcholine at muscarinic receptors and produces relief from 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, 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 pralidoxime (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 administration of 2-PAM Cl.

3-42. The 2-PAM Cl 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. The 2-PAM Cl 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 oxime 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. However, almost all of the complexes of GD and cholinesterase have aged within 10 minutes of binding. This renders 2-PAM Cl ineffective against GD exposure unless administration occurs relatively early.

3-43. Diazepam, the active ingredient in CANA, is approved by the FDA for injection as an adjunct to severe recurrent convulsive seizures. These seizure types are often associated with nerve agent poisoning. Other benzodiazepines (for example, midazolam) have shown to be effective in reducing seizures caused by nerve agents but are not yet approved by FDA for this purpose. Other anticonvulsants that are not in the benzodiazepines family, such as phenobarbital and phenytoin are not effective against nerve agent induced seizures.

# ANTIDOTE TREATMENT NERVE AGENT, AUTOINJECTOR

3-44. The ATNAA replaced the nerve agent antidote kit (MARK I) autoinjector. For more information about the administration of the ATNAA see Appendix E.

## DESCRIPTION

3-45. The ATNAA is a multichambered 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.

3-46. 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.

3-47. 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.

3-48. The chemically hardened 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.

***Note.*** The ATNAA is packaged in a chemically hardened pouch. The ATNAA needs to remain stored in the chemically hardened pouch and not removed until it is needed for use.

### Issue to Service Members

3-49. Each Service member will be issued and will carry three ATNAAs 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).

3-50. The use of the ATNAA by or upon persons to whom it has not been prescribed (such as DOD civilians or contractors that are deployed or casualties of terror or combat actions) is enabled by a DOD policy (DODI 3020.41 and DODD 1404.10) that empowers health care providers and other first responders and Service members to use these medications in an emergency, as an element of prehospital or on-site emergency medical actions. Also, see AR 40-400 for more information.

***Note***. Separately packaged atropine autoinjectors will still be available for use by medical personnel.

3-51. Additional ATNAA is given by a buddy since casualties requiring more will be unable to administer additional injections to themselves. The additional administration of ATNAA 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 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 ATNAA unless signs of nerve agent intoxication reappear.

### Protection From Freezing

3-52. 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 (still packaged in a chemically hardened pouch) 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 the ability to administer the antidote when needed, which could lead to increased injury from exposure to a nerve agent.

**WARNING**

**Service members should avoid extreme temperatures if possible.**

## ATROPINE

3-53. The atropine injection, rate of absorption, symptoms, and atropine administration are discussed below.

### Rate of Absorption

3-54. A 2 mg intramuscular (IM) injection will reach peak effectiveness in 3 to 10 minutes. 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. This can be provided through additional IM injections or the slow IV administration of atropine.

### Symptoms Produced by Antidotes in the Absence of Nerve Agent Poisoning

3-55. 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 symptoms to 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. A third 2 mg dose of atropine (again without nerve agent challenge) administered within an hour will incapacitate most people. 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. 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. 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.

3-56. 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.

***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.

3-57. 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. 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. 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.

3-58. 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. Hence, it is important that Service members not give themselves more than one ATNAA. Casualties who are able to walk (ambulate) and know who and where they are may not need any more ATNAAs. If the symptoms do recur, additional ATNAAs, 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 the Service member needs additional injections of ATNAA. If an individual's breathing appears normal, bronchial secretions have diminished, and the skin is dry, the individual does not need any more ATNAA at that time.

### Atropine Administration

3-59. Patients with severe symptoms due to systemic absorption of a nerve agent require increase levels of atropine to control the effects of nerve agents. 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) that may be required in patients poisoned by ingestion of organophosphorous (organosphosphate) pesticides. More than three injections of ATNAA will be administered only by the combat lifesaver or medical personnel. After the administration of three injections of ATNAA autoinjectors, repeat doses may be given (as needed) of atropine alone (every 3 to 10 minutes).

## 2-PRALIDOXIME CHLORIDE

3-60. The 2-PAM Cl rate of absorption and symptoms are discussed below.

### Rate of Absorption

3-61. Depending on the degree of intoxication, a 600 mg injection (of 2-Pam Cl as a part of the ATNAA) will be effective in 6 to 8 minutes and may maintain effectiveness for 1 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. When challenged by a nerve agent, additional IM doses of 2-PAM Cl may be needed and should normally be separated by approximately 60 to 90 minutes.

### Symptoms Produced by Antidotes

3-62. Blurred vision, nausea, vomiting, vertigo, and, most significantly, elevations of heart rate and blood pressure may occur after overdosage with 2-PAM Cl.

# CONVULSANT ANTIDOTE FOR NERVE AGENT, AUTOINJECTOR

3-63. The CANA is an anticonvulsant that is used by the Armed Forces to prevent or treat seizures from nerve agent poisoning. Convulsant antidote for nerve agent (diazepam) will be replaced by the Advanced Anticonvulsant System (midazolam).

## DESCRIPTION

3-64. 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.

3-65. 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.

3-66. The pouch has easy-to-tear notches on all sides. The lettering on the pouch is black.

### Issue to Service Members

3-67. Each Service member will be issued and will carry one CANA for use in the prevention and treatment of seizures from nerve agent poisoning.

3-68. The use of the CANA by or upon persons to whom it has not been prescribed (such as DOD contractors and civilian deployed or casualties of terror or combat actions) is also enabled by DODI 3020.41 and DODD 1404.10. Also, see AR 40-400 for more information.

***Note***. The CANA is not for use as self-aid.

### Protection From Freezing

3-69. Follow the atropine and the 2-PAM Cl solutions instructions above when protecting CANA from freezing.

## DIAZEPAM

3-70. The diazepam rate of absorption, symptoms, and administration are discussed below.

### Rate of Absorption

3-71. 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.

### Symptoms Produced by Antidotes

3-72. 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 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.

## ELEMENTS OF SELF-AID AND BUDDY AID

3-73. Don the protective mask immediately at the first signs of a chemical attack. The protective overgarment should have already been put on prior to the use of CW agents on the battlefield. In the event of a CBRN incident, immediately close eyes and stop breathing, put on mask, clear and seal the mask, and resume breathing. Wear the mask and protective clothing continually until the *all clear* signal is given.

- Immediately mask any casualty who does not have a mask on if the atmosphere is still contaminated.
- The appearance of severe nerve agent poisoning symptoms calls for the immediate IM injection of the nerve agent antidote (ATNAA) and CANA.
- Promptly remove any liquid nerve agent on the skin or on the clothing. Remove agent in wounds and eyes by irrigation.

3-74. If a liquid nerve agent gets on the skin, decontamination should ideally be accomplished within 1 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.

3-75. 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 nerve agent antidote is not needed.

3-76. 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. 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. Combat medics/corpsmen/Air Force medics also carry extra CANA for administration to nerve agent patients. Combat medics/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 an MTF as soon as the combat situation permits.

3-77. 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 eye ointment.

3-78. 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.

3-79. Severe nerve agent exposure may rapidly cause unconsciousness, muscular paralysis, and the cessation of breathing. When this occurs, antidote alone will not save life. Immediately after a buddy administers three sets of ATNAA autoinjectors 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.

## SELF-AID PRINCIPLES

3-80. This section will discuss self-aid administration principles.

### Administration of Antidote Treatment Nerve Agent Autoinjector

3-81. If a Service member experiences most or all of the mild symptoms of nerve agent poisoning, this individual should immediately close his/her eyes and stop breathing and put on the assigned protective mask. Then the Service member should self-administer one ATNAA injection into the lateral (outer portion) thigh muscle or buttocks. Self-aid procedure for administering the autoinjectors is found in Appendix E.

3-82. The Service member should wait 10 to 15 minutes after the first injection since it takes that long for the antidote to take effect. If the Service member is able to walk and know where he/she is, the Service member may not need a second ATNAA injection.

**WARNING**

**Injecting a second ATNAA injection when not exposed to a nerve agent may result in adverse effects.**

3-83. A buddy must administer the second and third sets of injections, if needed.

### Administration of Convulsant Antidote for Nerve Agent

3-84. The CANA is not for use as self-aid. If the Service member knows who and where he/she is, then CANA is not needed. If symptoms do not subside after the Service member self-administered one ATNAA, then buddy aid is required.

## BUDDY AID AND COMBAT LIFESAVER AID PRINCIPLES

3-85. This section will discuss buddy aid and combat lifesaver administration principles.

### Administration of Antidote Treatment Nerve Agent Autoinjector

3-86. If a Service member is suffering from severe signs of nerve agent poisoning, render the following aid:

- Mask the casualty, if necessary. Do not fasten the hood.
- Administer, in rapid succession, three ATNAAs. Follow administration procedures outlined in Appendix E.

3-87. If a Service member has already self-administered one ATNAA, then administer two more in rapid succession.

***Note***. Use the casualty's own antidote autoinjectors when providing first aid. Service members should not use their own issued injectors on a casualty. If they do, they may not have any antidote available when needed for self-aid.

### Administration of Convulsant Antidote for Nerve Agent

3-88. Special considerations when administering CANA:

- Administer the CANA with the third ATNAA to prevent convulsions.
- Do not administer more than one CANA. Follow administration procedures outlined in Appendix E.

**WARNING**

**Service members should not use their own CANA on the casualty. If they do, they may not have any CANA available when needed for their own treatment.**

## COMBAT MEDICS/CORPSMEN/AIR FORCE MEDICS CARE PRINCIPLES

3-89. This section will discuss combat medics/corpsmen/Air Force medics administration principles.

### Administration of Antidote Treatment Nerve Agent Autoinjector

3-90. If a patient has received three ATNAAs but is not yet medically stable, then administer additional atropine at approximately 5- to 10-minute intervals until breathing becomes easier and secretions are reduced 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.

### Administration of Convulsant Antidote for Nerve Agent

3-91. The combat medic/corpsman/Air Force medic should administer additional CANA to patients suffering seizures/convulsions. If a patient has flaccid paralysis, assume seizures are present. Administer CANA (30 to 40 mgs) in succession until seizures or convulsion stops and then reassess patient's symptoms. If seizures/convulsions persist or return, administer additional CANA. Throughout this process, the patient's airway should be maintained and monitored.

## ROLE 2/3 TREATMENT PRINCIPLES

3-92. Upon arrival at the MTF, a patient may still have signs/symptoms of nerve agent poisoning. The patient may have received self-aid, buddy aid, combat lifesaver care, or treatment by the combat medic/corpsman/Air Force medic, or other medical personnel in the field before and during evacuation.

Additional injections or IV administration of the nerve agent antidotes must be administered at the MTF. 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.

## Atropine

3-93. Atropine in large quantities will be required in the treatment of moderate and severe nerve agent poisoned patients. A patient may require as much as 50 mg of atropine per 24 hours of care.

3-94. 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:

- 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.
- 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. For severe symptoms, the interval for administering atropine should be 3 to 5 minutes either IM or IV; with IV route preferred.
- For patients who are in severe respiratory distress or are convulsing, all three ATNAA autoinjectors should have been given (convulsions are treated with diazepam). 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, administer additional atropine IM (autoinjector every 3 to 5 minutes) or IV as often as needed. In severe nerve agent poisoning, patients may require up to 20 to 30 mgs over an hour period.

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***Note***. Doses of 2 mg of atropine (without 2-PAM Cl) can be injected every 10 to 30 minutes as long as needed.

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## The 2-Pralidoxime Chloride

3-95. 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. The ATNAA is a specially designed unit for automatic self- or buddy-administration by military personnel. When activated, the ATNAA sequentially administers atropine and pralidoxime chloride through a single needle.

3-96. 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. The 2-PAM C1 single injector device (currently 60 IM injectors) can be found in an MTF. At the MTF, 2-PAM Cl titration can be continued if needed.

3-97. At the MTF, 2-PAM Cl can also be given IV. The oxime must be given slowly over 30 minutes. Therapeutic dosage will depend on the nerve agent, the time since poisoning, and individual physiology. Therapeutic dose is estimated to be 1000 to 2000 mg. A serious side effect of 2-PAM Cl is hypertension. Hypertension can be transiently reversed by 5 mg phentolamine given intravenously. There are other oximes beside 2-PAM Cl that may be used by countries conducting joint operations with U.S. forces. The oximes differ in their required doses, their toxicity, and their effectiveness. The 2-PAM Cl is the only FDA-approved oxime for nerve agent poisoning.

## Diazepam

3-98. Seizures/convulsions should be anticipated in all severe cases and treated with CANA and repeated as necessary.

### Management of Increased Airway Resistance

3-99. 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.

### Management of Bronchial Secretions and Salivation

3-100. 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.

### Assisted Ventilation

3-101. 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, an intubation or a cricothyroidotomy is the most practical means of providing an airway for assisted ventilation, using a hand-powered ventilator equipped with a CBRN filter. Only medical personnel trained to perform these procedures should attempt them. It is important to anticipate increased airway resistance and to administer atropine, preferably before intubation or cricothyroidotomy, to minimize this problem. Intubation or cricothyroidotomy should not be deferred if required merely because atropine is not available. When a casualty reaches an MTF (the MTF must be in a clean environment or the patient must be inside a CPS) 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***. Some treatments outlined here are based on U.S. Army doctrine on the use of the ATNAA 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.

### Treatment of Ocular Symptoms

3-102. 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.

## PYRIDOSTIGMINE BROMIDE

3-103. Pyridostigmine bromide should be administered for any credible threat of nerve agent exposure, including soman. Pyridostigmine bromide, in conjunction with individual protective equipment and immediate atropine and 2-PAM C1 therapy, may enhance survivability of nerve agent-poisoned casualties. Command, unit, and individual responsibilities for the PB pretreatment regimen are discussed in this section.

**WARNING**

**Pyridostigmine bromide (PB) is for pre-exposure prophylaxis only. Do not give to any patient who has been exposed to or presents with symptoms of organophosphate nerve agent poisoning.**

3-104. The FDA has approved 30 mg PB tablets as a pretreatment against organophosphate nerve agent poisoning.

3-105. Any potential benefits that may be derived from use of this pretreatment regimen are those casualties who have been treated with the ATNAA at the time of nerve agent exposure, and who have taken their pretreatment medication.

3-106. Minimal detrimental effects are expected at the recommended dosages.

## EFFECTS OF PYRIDOSTIGMINE BROMIDE

3-107. Pyridostigmine bromide protects the acetylcholinesterase enzyme in the body from the action of the organophosphate nerve agent. 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.

3-108. 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 within one to three hours after taking the first tablet. Maximal benefit develops with time and is reached when a tablet is taken consistently every 8 hours.

## EMERGENCY MEDICAL TREATMENT FOR PYRIDOSTIGMINE BROMIDE ADVERSE SIDE EFFECTS, ALLERGIC REACTIONS, AND OVERDOSE

3-109. Ordinarily, discontinuing PB 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.

3-110. 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 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 10 to 15 minutes, thereby permitting the previous injection of atropine to exert its anticholinergic effect prior to the next injection.

3-111. Severe cases may require assisted ventilation because of weakness but would be unusual when the pretreatment medication was administered every eight hours as directed.

3-112. When stabilized, the patient should be evacuated for further observation and treatment.

## RESPONSIBILITIES

3-113. The corps/brigade/wing/fleet commander should—

- Decide whether to begin, continue, or discontinue PB therapy based on the threat of exposure to nerve agents. The presence of nerve agents in theater or the high probability of their use is the responsibility of the CBRN and intelligence officer. The use of PB as pretreatment against the lethal effects of potential nerve agent poisoning is consistent with the indicated use described in FDA-approved labeling. Since PB is a prescription drug, the command surgeon or another physician should be involved in the decision to initiate therapy. The commander will inform Service members when to begin taking PB tablets. Service members must take one PB tablet every eight hours until ordered to stop. This decision should be based on the threat of exposure to nerve agents. 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.
- Train the Service members to take PB as a directed pretreatment to enhance their survivability if they are exposed to organophosphate nerve agent. Service members must be trained to take PB during the day, at night, and while in MOPP 4, should these procedures become necessary.
- Issue unit standard operating procedures for the retention and decontamination of PB blister pack during personnel decontamination and overgarment exchange.

3-114. Units should—

- Obtain the supplies of PB through medical supply channels.
- Maintain at least a two-week supply of PB per member of the unit. One PB blister pack is issued to each member of the unit. An additional week's supply of PB 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.
- Store PB 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, PB must be replaced every three months.
- Issue PB to the Service members at the time the chemical protective ensemble is expected to be opened for use.

3-115. Unit medical personnel should—

- Recognize the signs and symptoms of PB overdose, adverse reactions, and side effects for determining, on an individual basis, whether or not a Service member is to continue PB based on any adverse reaction to the medication.
- Advise the commander if any serious problems occur.

3-116. The individual Service member should—

- Take PB as directed.
- Cease taking PB if exposed to nerve agent until directed to resume self-administration by higher authority.
- Secure PB supplies against loss and freezing.

# Chapter 4
# Blood (Cyanide) Agents

## GENERAL

4-1. Blood (cyanide) agents 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 (vesicant) agents, nerve agents, and incapacitating agents are also absorbed into the bloodstream and systemically distributed and are in that sense as much blood (cyanide) 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 blood (cyanide) agents. Hydrogen cyanide (AC) and CK are the important agents in this group.

4-2. Cyanogen chloride (CK) also produces central and peripheral pulmonary effects on the respiratory tract because of its chlorine component. These agents can be dispersed by artillery shell, mortar shell, rocket, aircraft spray, and bomb. All blood (cyanide) agents are nonpersistent.

## PROTECTION

4-3. The protective mask with a new filter gives protection against field concentrations of cyanide. Due to its volatility and lack of persistency, a mask only posture can be assumed if cyanide vapors are present.

## HYDROGEN CYANIDE

4-4. 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 that can be detected by only 40 to 60 percent of the population. Moreover, olfactory accommodation to the odor of blood (cyanide) agents 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.

## CYANOGEN CHLORIDE

4-5. 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 a jungle or a forest for up to several hours under suitable weather conditions.

### PATHOLOGY

4-6. Hydrogen cyanide acts by combining with cytochrome oxidase (an enzyme located within mitochondria in cells) and is 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 to produce energy and package it as adenosine triphosphate. 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 CO poisoning. The cherry-red coloration seen in CO poisoning results from the intrinsic color of carboxyhemoglobin, whereas the pink tinge to the skin in cyanide poisoning reflects the high oxygen content of capillary and venous blood.

4-7. 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.

## SYMPTOMS

4-8. 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.

4-9. 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.

## DIAGNOSIS

4-10. The diagnosis of AC poisoning is suggested by the patient's history, including whether the patient smelled an odor, 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.

4-11. 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.

4-12. 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.

## PROGNOSIS

4-13. The next two paragraphs will discuss AC and CK prognosis.

### Hydrogen Cyanide

4-14. At sublethal exposures, many casualties recover within hours without sequelae. Death may occur rapidly after a massive exposure. For those who survive a potentially lethal exposure, there may be prolonged tissue anoxia, residual injury of the CNS may persist for weeks; some of this damage may be permanent.

### Cyanogen Chloride

4-15. 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.

## SELF-AID

4-16. The next two paragraphs will discuss self-aid procedures for AC and CK.

### Hydrogen Cyanide

4-17. If a Service member gets a sudden stimulation to breathe or detect a bitter almond odor during a chemical attack, the Service member must put on the assigned protective mask immediately. Speed in masking is absolutely essential since the effects of this agent are so rapid that within a few seconds the Service member will not be able to put on the mask. The Service member must stop breathing until the mask is on, if possible. This may be very difficult because of the agent's strong respiratory stimulation. Once the mask is on, and sealed, then if the Service member has uncontrolled rapid breathing, the Service member will be protected by the mask filter.

### Cyanogen Chloride

4-18. The Service member must put on the assigned protective mask immediately if the individual experiences any irritation of the eyes, nose, or throat.

## BUDDY AID

4-19. Service members not masked must put on their masks immediately if any AC or CK is present. The mask should be cleared by forcefully exhaling after it is donned and prior to the first inhalation. Service members unable to mask should be masked by the nearest available person (buddy).

## TREATMENT

4-20. 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. 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. For a quick ready reference for the treatment of AC or CK poisoning, refer to Appendix F.

4-21. If amyl nitrite is available, hold one ampule, or capsule, close to a breathing patient's nose, (break mask's seal, if necessary), crush the ampule, and allow the patient two to six breaths (30 seconds) from the ampule. For an apneic patient being treated with "only" amyl nitrite inhalation, you should crush an ampule in the mask and ventilate for 30 seconds, then ventilate for 30 seconds with air/oxygen only. Keep repeating this sequence with a fresh amyl nitrite ampule until the patient responds.

4-22. Intravenously inject one vial (10 milliliter [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 gram) of sodium thiosulfate over a 10-minute eriod. 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.

4-23. A cyanide kit that contains hydoxocobalamin, an antidote indicated for the treatment of known or suspected cyanide poisoning is currently available. If clinical suspicion of cyanide poisoning is high, this antidote should be administered without delay. The starting dose for adults is 5 gram by IV over 15 minutes. Depending upon the severity of the poisoning and the clinical response, a second dose of 5 gram may be administered by IV for a total of 10 grams.

4-24. In 2011, FDA approved a New Drug Application (co-packaged sodium nitrite injection and sodium thiosulfate injection drug product) indicated for the treatment of acute cyanide poisoning that is judged to be life-threatening. Additionally, in 2012, FDA approved New Drug Applications for sodium nitrite injection and sodium thiosulfate injection, which are marketed in separate packaging. Sodium nitrite injection and sodium thiosulfate injection are approved to be used sequentially: Sodium nitrite is injected first, followed immediately by sodium thiosulfate, for the treatment of acute cyanide poisoning that is judged to be life-threatening. The unapproved products on the market compete with these approved products, and pose a direct challenge to the drug approval system.

4-25. 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.

4-26. 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.

4-27. The lung irritant effects of CK are treated according to the presence of pulmonary effects, as in chlorine poisoning.

# Chapter 5
# Blister (Vesicant) Agents

## GENERAL

5-1. Blister (vesicant) agents are likely to be used to produce casualties and to force opposing troops to wear full protective equipment. Blister (vesicant) agents are used to degrade fighting efficiency rather than to kill, although exposure to such agents can be fatal. Blister (vesicant) agents will contaminate terrain, ships, aircraft, vehicles, or equipment and present a persistent hazard. Vesicants include the mustards (generally referred to as H, HD, and HN; arsenicals (primarily L); and the halogenated oximes (primarily CX). The properties and effects of halogenated oximes are different from those of the other vesicants. Although phosgene oxime is a corrosive and an urticant (producing wheals or hives) rather than a vesicant, it is usually grouped with the true vesicants.

5-2. 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.

5-3. Some vesicants have an odor (HD may smell like tar or garlic; L may smell like geraniums); others are odorless. Vesicants can contaminate food and water and make other supplies dangerous to handle. Vesicants can be disseminated by artillery shell, mortar shell, rocket, aircraft spray, and bomb. Although the properties of nitrogen mustard are similar to sulfur mustard, it was not found suitable for use as a weapon but it is a possible TIC.

5-4. The severity of a blister (vesicant) agent burn is directly related to the concentration of the agent, the duration of contact with the skin, and the location on the body. 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.

## SELF-AID

5-5. Assume MOPP 4 whenever liquid or vaporized agents are known to be present.

5-6. Immediately decontaminate the eyes or the skin if exposed to liquid or vapor agents. Follow decontamination procedures as outlined in Appendix D.

## PRECAUTIONS FOR RECEIVING CASUALTIES

5-7. Casualties contaminated with vesicants endanger unprotected attendants. Individuals in contact with these casualties must be at MOPP 4, plus wear a butyl rubber apron.

5-8. Special precautions must be taken when receiving contaminated casualties to prevent injury to others. Contaminated casualties must be decontaminated outside the MTF to prevent vapor accumulation indoors and cross contamination of medical personnel and equipment. 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.

5-9. Vesicants present on casualties' skin surface can present a hazard to individuals receiving or treating these casualties even after several hours, but vesicants that have been absorbed into the skin will not be a surface contact hazard. Blisters caused by mustard agent exposure do not contain active agent and the fluid contained therein poses no contamination risk beyond usual body fluid exposure.

# PROTECTION

5-10. The protective mask protects only the face, eyes, and respiratory tract. The mask protects against both liquid and vapor forms of vesicants.

5-11. Chemical protective overgarments help prevent the vesicant from reaching the skin.

# MUSTARD

5-12. Sulfur mustard contains up to 30 percent impurities and is known as H. Sulfur mustard made by a distillation procedure is almost pure and is known as HD (distilled mustard). In this section, the term "mustard" encompasses both H and HD, unless otherwise noted.

## PHYSICAL PROPERTIES

5-13. Mustard is an oily liquid ranging from colorless when pure (neat) to dark brown. Under temperate conditions, mustard evaporates slowly and is primarily a liquid hazard, but its vapor hazard increases with increasing temperature. At 100°F (37.8°C) or above, it is a definite vapor hazard. Mustard freezes below 58°F (14.4°C) and since a solid is difficult to disperse, mustard is often mixed with substances like Lewisite (the mixture is HL) so that the mixture will remain liquid at lower temperatures. 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 mustard, is only slightly soluble in water, which gradually destroys it, but undissolved mustard may persist in water for long periods. It is most soluble in fats and oils. It is freely soluble in acetone, carbon tetrachloride, alcohol, and liquid fuels (gasoline, kerosene, and diesel); however, these solvents do not destroy mustard. Mustard slowly disappears from contaminated ground or materials through evaporation or hydrolysis.

### Persistence

5-14. The persistence of a hazard from mustard vapor or liquid depends on the degree of contamination by the liquid, type of mustard, nature of the terrain, soil, or material contaminated, type of munitions 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 mustard vapor in the field.

### Cumulative Effect

5-15. Repeated exposures to mustard produce cumulative effects. For example, repeated exposures to vapors from spilled mustard can produce disability by irritating the airways and causing a chronic cough and pain in the chest.

## EFFECTS OF SULFUR MUSTARD ON THE EYES (PATHOLOGY, SYMPTOMS, AND PROGNOSIS)

5-16. The eyes are more susceptible to mustard than either the respiratory tract or the skin. 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.

5-17. Casualties should be reassured. 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:

- Mild conjunctivitis (75 percent of cases in World War I). Recovery takes one to two weeks.
- 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 2 to 5 weeks.
- 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.
- Severe corneal involvement (about 0.1 percent of HD casualties in World War I). Ischemic necrosis of conjunctivae may be seen.
- 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.

## Treatment

5-18. This section will discuss eye treatment of mustard.

### *Self-Aid*

5-19. 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.

5-20. Speed in decontaminating the eyes is absolutely essential. This self-aid procedure is very effective for mustard within the first few seconds after exposure but is of less value after two minutes. Decontamination is done the same as for other vesicants.

### *Treatment of Mustard Conjunctivitis*

5-21. Mild lesions require little medical treatment. The lesions may become secondarily infected. 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 petrolatum or a sterile antibiotic ointment between the eyelids will provide additional lubrication and prevent the eyelids from sticking together.

5-22. 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.

5-23. 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, or as needed, to keep the pupil dilated to prevent later synechiae formation. 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.

5-24. The eye must not be tightly bandaged or the lids allowed to stick together. Prevent the eyelids from sticking together by using petroleum jelly or a similar material. 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.

### *Treatment of Infected Mustard Burns of the Eye*

5-25. 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 2 hours.

5-26. After appropriate cultures, specific antibacterial preparations may be applied. Irrigation should be gentle and employed only to remove accumulated exudate. Control pain as needed. Refer patients with secondary infection or other complications to an ophthalmologist. Local anesthetics should not be used.

## EFFECTS OF MUSTARD ON THE SKIN

5-27. This section will discuss effects of mustard on the skin.

### Pathology

5-28. The severity of the lesions and the rapidity with which they develop are greatly influenced by weather conditions as well as by the degree of exposure. Hot, humid weather strikingly increases the action of mustard. Even under temperate conditions, the warm, moist skin of the perineum, external genitalia, axillae, antecubital fossae, and neck are particularly susceptible.

### Latent Period

5-29. 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.

#### *Erythema*

5-30. Erythema gradually appears (2 to 48 hours postexposure) and becomes brighter, resembling sunburn. 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.

#### *Vesication*

5-31. Except with mild vapor burns, erythema is followed by vesication. 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) mustard 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. This pattern is often not present and blisters may arise indiscriminately in the affected area. Unreacted vesicant on contaminated patients may pose a hazard to other individuals coming in contact with them.

#### *Resorption*

5-32. 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.

#### *Healing*

5-33. 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.

#### *Pigmentation*

5-34. 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. Classic salt-and-pepper pigmentation seen in some healing patients reflects epithelial regeneration arising from hair follicles and gradually spreading to confluence.

***Hypersensitivity***

5-35. Mustard burns may lead to skin hypersensitivity to subsequent exposures.

## Symptoms and Prognosis

5-36. A notable characteristic of the action of mustard is its insidiousness. Exposures to mustard 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.

5-37. Mustard erythema resolves at about the same rate as sunburn of like severity. Healing times for mustard 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. A mustard burn of the skin is usually limited to the epidermis and does not require grafting.

5-38. Active infection of mustard skin lesions with saprophytic bacteria (with inflammation and purulent exudation), may increase the severity of the lesions and delay healing.

## Diagnosis of Mustard Skin Lesions

5-39. Mustard and the HNs produce essentially identical skin burns. Mustard burns are also similar in appearance to those caused by L. Differentiation of mustard lesions from those produced by arsenicals is based upon—

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

## Decontamination of Casualties

5-40. Casualties who have liquid mustard 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 lifesaving by preventing continued absorption. It also can prevent the spread of the 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 mustard. Decontaminate the exposed scalp and exposed skin with RSDL. If not available, use copious amounts of soap and water.

## Treatment of Mustard Erythema

5-41. 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.

### Treatment of Mustard Blisters

5-42. Unless painful, leave the blister intact in a field environment. 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.

5-43. Mustard blisters or deep lesions can be handled in several ways depending on severity, preferences, and available facilities as follows:

- Leave small blisters, 1 centimeter 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.

### Treatment of Denuded Areas

5-44. Contamination of mustard burns with saprophytic bacteria is common and unless careful wound care is given, serious infection may result. If there is no inflammatory reaction, the treatment is the same as for uncontaminated burns.

5-45. 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.

5-46. 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.

5-47. 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 mustard patient. Fluid replacement should be governed by clinical judgment.

## Effects of Mustard on the Respiratory Tract

5-48. Inhalation of mustard vapor causes damage primarily to the laryngeal and tracheobronchial mucosa.

### Pathology

5-49. The lesions in the laryngeal and tracheobronchial mucosa develop slowly after exposure. A single exposure to a small amount of mustard vapor ordinarily does not produce significant injury. Repeated or chronic exposure to low concentrations of mustard vapor may lead to progressive pulmonary fibrosis, chronic bronchitis, and bronchiectasis. Moderate exposures result in hyperemia of the respiratory mucous membrane and necrosis of the lining epithelium. 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.

5-50. 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 mustard but may be seen after massive exposures. The early mortality from mustard among American troops in World War I (slightly more than 2 percent) was due almost entirely to such pulmonary complications following inhalation of vapor.

### Symptoms and Prognosis

5-51. 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 mustard 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.

### Treatment of Respiratory Tract Injury Due to Mustard

5-52. 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 patient's airway, improve oxygenation, and aid in removal of secretions. If evidence of bronchospasm is present, bronchodilators may be of benefit. If a bacterial pneumonia occurs, isolation of the specific organisms with their antibiotic sensitivities should be performed, and then antibiotic therapy can be limited to the specific agents. Administration of prophylactic antibiotics in the absence of culture results is not recommended.

## SYSTEMIC AND GASTROINTESTINAL EFFECTS OF MUSTARD

5-53. Ingestion of mustard produces vacuoles and nuclear swelling of the epithelial cells of the gastrointestinal tract with eventual necrosis and desquamation with hemorrhage.

### Pathology

5-54. Absorption of mustard 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.

5-55. With lesser skin or respiratory exposures to mustard, 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 also involved usually with subsequent lymphocytopenia, but there may be initial lymphocytosis.

5-56. Mustard also damages deoxyribonucleic acid, is mutagenic, and is classified by the World Health Organization, International Agency for Research on Cancer 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 mustard vapor and theoretically could be elevated following a single acute exposure although there is no scientific evidence to support this.

### Symptoms

5-57. Mustard vapor does not significantly contaminate food or water; however ingestion of food or water contaminated by liquid mustard produces nausea, vomiting, pain, diarrhea, prostration, and in severe cases, death.

5-58. Exposure of only the skin to mustard may cause systemic symptoms such as malaise, vomiting, and fever, coming on about the time of onset of the erythema. With severe exposures, particularly by extensive liquid contamination of the skin, these symptoms may be so marked as to result in prostration. Exceptional cases of severe systemic mustard poisoning may also present CNS 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.

5-59. Sufficiently high doses of mustard 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.

## Prognosis

5-60. With mild to moderate field exposures to mustard vapor, deaths rarely occur from systemic effects of absorbed mustard. Death may occur from prolonged exposures to high concentrations of mustard vapor or, in instances of extensive liquid contamination of the skin, where decontamination is neglected or unduly delayed. The 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 mustard 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 leads to sepsis and septic pneumonia. It is the sepsis and septic pneumonia which causes death. Ingestion of mustard is rare but can cause severe injury, including death.

5-61. Never drink water that has been subjected to chemical attack until it has been certified as fit to drink by medical personnel/preventive medicine/public health personnel. Never eat foods that have been exposed to liquid vesicants, unless in sealed cans or aluminum-laminated pouches (field ration meals), until examined by U.S. Army veterinary personnel and certified as safe to eat.

## Treatment of Systemic Effects of Mustard Poisoning

5-62. 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, 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.

5-63. Sulfur donors such as sodium thiosulfate decrease systemic effects and elevate the lethal dose for 50 percent of those exposed ($LD_{50}$) 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 mustard remains in the circulation. Its efficacy is very doubtful if given later.

5-64. One study in nonhuman primates demonstrated that granulocyte colony stimulating factor reduced the severity and duration of mustard -induced pancytopenia.

5-65. Injury due to the ingestion of liquid mustard in food or water may require morphine and atropine for relief of pain.

## Secondary Bacterial Infection in Mustard Burns

5-66. Secondary bacterial infection may result if adequate wound care is not given. Compared to the incidence of infection in thermal and traumatic wounds, the incidence of sepsis in mustard lesions is remarkably low, according to observations made at experimental installations.

5-67. 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.

5-68. 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 injury casualty.

5-69. Secondary infection is an uncommon complication of mild mustard conjunctivitis and normally will not prevent an individual from continuing duty.

### Mild- and Long-Term Sequelae From Acute Exposure to Mustard

5-70. Mild conjunctival burns may be associated with pharyngitis, laryngitis, and tracheitis increasing in severity for several days. Occasionally, more extensive respiratory infection may ensue.

### Long-Term Sequelae From Acute Exposure to Mustard

5-71. Exposure to mustard 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, refer to the Textbook of Military Medicine, Medical Aspects of Chemical and Biological Warfare.

## LEWISITES

5-72. These agents are organic dichloroarsines. The main one is chlorovinyldichloroarsine, also called Lewisite. Others include phenyldichloroarsine, ethyldichloroarsine, and methyldichloroarsine. Lewisite has been combined with mustard to lower the freezing point. These agents have been known to have been stockpiled by other countries.

5-73. All Lewisites 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.

5-74. Lewisite is similar to mustard in that it damages the skin, eyes, and airways; however, it differs from mustard because its clinical effects appear within seconds of exposure. 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.

### EFFECTS OF LEWISITES ON THE EYES

5-75. Lewisites cause severe damage to the eye.

### Pathology, Symptoms, and Prognosis

5-76. Pain and blepharospasm (sustained forced closing of the eyelids due to involuntary muscle spasm) occur within seconds to minutes of eye 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 (an accumulation of pus in the anterior chamber of the eye) may ensue, terminating in necrosis, depigmentation of the iris, and synechia (abnormal adherence of the iris to the cornea or lens) formation.

5-77. Lewisites 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 Lewisites heals in a few days without specific treatment. Severe exposure may cause permanent injury or blindness.

### Treatment

5-78. 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 (dilated pupils) in all cases with corneal erosions, iritis, cyclitis, or with marked photophobia or miosis (constriction of pupils). 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 (such as normal saline). Occlusive dressings and pressure on the globe must be avoided.

## Effects of Lewisites on the Skin

5-79. Liquid Lewisites produce more severe lesions of the skin than liquid mustard.

### *Pathology*

5-80. 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 mustard. 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, Lewisite burns, there may be considerable tissue necrosis, gangrene, and slough. Lewisite damages capillary endothelium (thin wafer-like cells joined at their borders that form the inner lining of the entire blood vascular system) and the resulting increase in capillary permeability leads to local edema at the site of skin contact.

### *Symptoms*

5-81. Stinging pain is felt usually within 10 to 20 seconds after contact with liquid Lewisites. 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 Lewisites usually gives sufficient warning to allow for prompt decontamination and avoidance of deep burns in conscious victims. Pain caused by a Lewisite lesion is immediate and may diminish after blisters form. Erythema is evident within 15 to 30 minutes after exposure to liquid Lewisite, and blisters start within several hours. Lewisite is absorbed by the skin within 3 to 5 minutes (compared to 20 to 30 minutes for an equal amount of mustard) and spreads over a wider area than the same amount of mustard.

### *Prognosis*

5-82. The erythema of Lewisites 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 Lewisites occurs, as with mustard.

### *Treatment*

5-83. The treatment of arsenical skin and eye injury is entirely supportive and similar to that of mustard lesions. The antidote dimercaprol (British anti-Lewisite [BAL]), a heavy metal chelator, is not available through the U.S. military medical supply system, but may be available through coalition forces during international operations.

5-84. Some blistering is inevitable in most Lewisite cases that arrive at MTFs. The treatment of the erythema, blisters, and denuded areas is identical with that for similar mustard lesions. Lewisite blisters may contain a small amount of arsenic (0.8 to 1.3 mg/ml), which can potentially be vesicating, so gloved precautions must be used when managing filled blisters. 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 or when it 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.

## Effects of Lewisites on the Respiratory Tract

5-85. The vapor of Lewisites is so irritating to the respiratory tract that a conscious casualty will tend immediately to put on a mask.

### *Symptoms*

5-86. Severe respiratory injuries are likely to occur only among the wounded that 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 L 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.

### *Prognosis*

5-87. The prognosis is unknown because there have been no known human cases of poisoning by vapors of Lewisites. Extrapolating from animal experiments, the prognosis probably is similar to that for respiratory injury by mustard.

### *Treatment*

5-88. The treatment begins with that for mustard respiratory injury plus preparation for pulmonary edema.

## Systemic Effects of Lewisites

5-89. Absorbed Lewisites may cause systemic poisoning.

### *Pathology and Symptoms*

5-90. A manifestation of systemic poisoning 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 (around a bile duct) 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).

### *Prognosis*

5-91. 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.

5-92. The indications for systemic treatment, following exposure to Lewisites by any route, are—

- A cough with dyspnea and frothy sputum, which may be blood tinged, and other signs of pulmonary edema.
- A skin burn the size of the palm of the hand, or larger, caused by a liquid Lewisite, which was not decontaminated within the first 15 minutes.
- Skin contamination by a Lewisite 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.

### *Types of Treatment*

5-93. Following prompt decontamination with the RSDL or with copious soap and water, follow treatment guidelines for mustard with the addition of attention to the development and treatment of pulmonary edema.

### *Mixtures of Blister (Veiscants) Agents*

5-94. Lewisites such as L or phenyldichloroarsine 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.

### *Radiomimetic Effects of Sulfur Mustard*

5-95. The damage caused by sulfur mustard is in some ways similar to that produced by ionizing radiation (for example, burns, bone marrow suppression). As a result, sulfur mustard is also classified as a radiomimetic agent.

## PHOSGENE OXIME

5-96. Phosgene oxime (chemical name dichloroformoxime) is an example of the class of CW agents called urticants (or nettle agents).

### PROPERTIES

5-97. Urticants 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 pain caused by a severe bee sting. Phosgene oximes were first synthesized in the late 1920s and became recognized as a potential agent for CW. It has a military designation of CX and is one of the least studied CW agents, so specific information is limited.

5-98. 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.

### SYMPTOMS AND COURSE OF LESIONS OF PHOSGENE OXIME

5-99. 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.

### SELF-AID

5-100. A properly-fitting protective mask protects the respiratory system. The individual protective equipment (field protective mask, hood, and chemical protective overgarment) protects the body. After exposure, because of the rapid reaction of CX with tissue, decontamination will not be entirely effective after pain has been produced. Use the RSDL for skin decontamination. If the RSDL is not available, flush the contaminated area as rapidly as possible with copious amounts of soap and water to remove any CX that has not yet reacted with tissue.

### TREATMENT FOR PHOSGENE OXIME INJURY

5-101. 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

## GENERAL

6-1. Incapacitating agents are chemicals designed to temporarily disable an individual, but they do not cause permanent injury or death. The effects of incapacitating agents are 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—

- Highly potent (an extremely low dose is effective) and logistically feasible.
- Able to produce their effects mainly by altering the higher regulatory activity of the CNS.
- Temporary in duration of action, lasting hours or days.
- Unlikely to produce permanent injury in concentrations that are militarily effective.

6-2. Incapacitating agents do not include—

- Lethal agents (such as nerve agents which are incapacitating at sublethal doses).
- Substances which cause permanent or long-lasting injury (such as blister [vesicants] agents, choking [lung-damaging] agents, and those injuring the eyes).
- 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.
- Agents which are transiently effective by producing reflex responses interfering with duty performance (such as vomiting and irritant agents).
- Agents which disrupt basic life-sustaining systems and prevent physical activity (such as agents that lower the blood pressure, paralyzing agents [for example, 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).

6-3. 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:

- Central nervous system depressants—
  - These compounds produce their effects by interfering with cholinergic synapses in the CNS. An example of this type of agent is 3-quinuclidinyl benzilate (BZ) 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.
  - 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.
  - Opioid narcotics have multiple CNS effects, including CNS depression. In sublethal doses, these narcotics can cause listlessness, significant sedation, and affect alertness, attention, and problem solving. Fentanyl derivatives, such as those used by the Russian military to break the siege of a Moscow theater in 2003, cause rapid sedation with an effective dose.

- Central nervous system stimulants—
  - These agents cause excessive nervous system 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.

## DIAGNOSIS

6-4. Currently, field laboratory methods do not permit isolation and identification of specific agents in environmental or body fluids (blood, urine, or cerebrospinal fluid). Refer to Appendix G for more information on technologies, detectors, and levels of identification.

6-5. 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:

- Transport casualties to an uncontaminated area. After initial treatment, resistant or disoriented individuals should be restrained in the triage area.
- 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.

6-6. 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. Very few other pharmaceutical classes can produce delirium in militarily effective doses. Hallucinations produced by psychedelic indoles (such as D-lysergic acid diethylamide [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 are unable to rid themselves of these abnormal perceptions.

**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 3-quinuclidinyl benzilate), indoles (such as D-lysergic acid diethylamide [LSD]), cannabinols (such as marijuana), anxiety reaction, or other intoxications (such as alcohol, bromides, barbiturates, and 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, and lethargy progressing from stupor to 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. |

6-7. Glycolate anticholinergic compounds 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 above 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.

6-8. Since atropine is also an anticholinergic compound, overdose may produce similar signs and symptoms and may be confused with other glycolate anticholinergic poisoning.

## PROTECTION, DECONTAMINATION, AND FIRST AID

6-9. This section will discuss incapacitating agent protection, decontamination, and first aid.

### Protection

6-10. It is likely that such agents will be dispersed by obscurant-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.

### Decontamination

6-11. Complete cleansing of the skin with soap and water should be accomplished at the earliest opportunity. The RSDL 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 a surge in patient numbers 6 to 24 hours after the attack.

### First Aid

6-12. The most important first aid considerations include—

- 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.
- 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.
- 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°F to 75°F (22.3 to 23.9°C) water and air circulation (fanning); applying alcohol or water-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 as this may damage skin. If body temperature cannot be lowered to safe levels, rapid evacuation should be accomplished since treatment with appropriate medication may be lifesaving.
- 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.

6-13. 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.

## ANTICHOLINERGICS

6-14. 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.

6-15. Once a desirable effect is achieved, it should be maintained by oral administration, slow IV injection, or infusion. Physostigmine is a reversible anticholinesterase compound (a carbamate) and controls signs and symptoms of BZ and atropine 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) and, because of reduced chance for overdose, is the preferred method for redosing.

***Notes.*** 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.

# INDOLES

6-16. No true antagonist to the indoles is known. The best treatment known at present for D-lysergic acid diethylamide (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.

# OTHER AGENTS

6-17. Unfamiliar agents or mixtures of agents may be encountered in the future. 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.

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## Chapter 7

# Riot Control Agents (Irritant Agents and Vomiting Agents)

## GENERAL

7-1. Irritant agents 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.

7-2. Irritant agents 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, bromobenzyl cyanide (CA), dibenz(b,f)-1,4-oxazepine (CR) and oleoresin capsicum (OC) (capsaicin). Some pulmonary agents such as CK and chloropicrin also induce lacrimation.

## IRRITANT AGENTS

7-3. They are used primarily in training and in riot control. Under certain conditions and with presidential approval, they may also be used in combat.

### PROTECTION

7-4. Protection against field concentrations of irritant agents is provided by the protective mask and ordinary field clothing secured at the neck, wrists, and ankles. 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.

7-5. 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 personnel. Decontaminate CS-contaminated clothing by airing for a few minutes. Bleach, which produces irritating by-products from these agents, should not be used for decontamination.

### AGENT O-CHLOROBENZYLIDENE MALONONITRILE

7-6. Agent CS is a white crystalline solid that melts at 194°F (90.0°C) and is stable under ordinary storage conditions.

#### Properties

7-7. Agent CS 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.

#### Effects

7-8. When an unmasked person enters a cloud of CS, the effects are felt almost immediately.

##### *Eyes and Respiratory Tract*

7-9. Irritation to the eyes and respiratory tract (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 preexisting pulmonary disease.

***Skin***

7-10. 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.

### Diagnosis

7-11. Diagnosis is made from the pepper-like odor, the presence of intense eye effects, dyspnea, coughing, and rhinorrhea.

## AGENT DIBENZ(B,F)-1,4-OXAZEPINE

7-12. 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.

### Properties

7-13. 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.

### Effects

7-14. Agent CR is similar in effect to CS, but the minimal effective concentration is lower and the lethal dose (lethal concentration) is higher. Thus, the safety ratio is greater than for CS. Symptoms and treatment are similar to those of CS.

### Diagnosis

7-15. Diagnosis is similar to the diagnosis of CS. Agent CR produces a burning sensation in the nose and sinuses.

## AGENTS CHLOROACETOPHENONE AND BROMOBENZYL CYANIDE

7-16. Agent CN and Agent CA may appear as bluish-white clouds at points of release.

### Properties

7-17. 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. Agent 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. 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.

### Effects

7-18. The vapors or smokes of these agents cause basically the same reactions as does CS.

#### *Eyes and Respiratory Tract*

7-19. Agent CN and agent CA's effectiveness as irritants are generally lower than CS; that is, higher concentrations of CN or CA are required to produce an irritant effect equivalent to that of CS. Recovery is quick if exposure is brief, but prolonged exposure may cause conjunctivitis and photophobia. 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.

#### *Skin*

7-20. 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.

### Diagnosis

7-21. Diagnosis is made from their odors and from the marked coughing and dyspnea in addition to the eye effects. Headache and depression may also appear as late effects of CN exposure.

## AGENT OLEORESIN CAPSICUM (CAPSAICIN)

7-22. There are some 20 species and 300 varieties of the agent oleoresin capsicum (capsaicin).

### Properties

7-23. Agent OC, commonly called pepper spray, is derived from the *Capsicum* plant family, which includes chili peppers, red peppers, jalapeno and paprika, but not black pepper. The *Capsicums* are hardy and adaptable, sometimes developing new characteristics of shape, color, size, and pungency.

### Effects

7-24. Exposure to OC results in irritation and inflammation of the mucous membranes. The OC dilates the capillaries and causes temporary blindness. It causes instant inflammation of the breathing tissues, restricting all but life support breathing.

### Diagnosis

7-25. Diagnosis is made from the pepper-like (hot cayenne) odor, dyspnea, coughing, and intense burning eye sensation.

## SELF-AID

7-26. Put on the protective mask, clear it, and keep eyes open as much as possible. Move out of the contaminated environment, if possible. When vision clears, go on with assigned 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 blisters (rarely) may develop. The cutaneous reaction can be prevented by immediately flushing the skin with copious amounts of water. Do not use bleach.

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***Note.*** Water will at first exacerbate the burning or stinging sensation of CS and pepper spray; however, decontamination should be continued.

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### TREATMENT

7-27. The effects on the eyes and skin do not necessary require treatment.

#### Eyes

7-28. 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 and OC 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.

#### Skin

7-29. 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 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.

#### Pulmonary

7-30. 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).

### PROGNOSIS

7-31. 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.

## VOMITING AGENTS

7-32. 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, diphenylaminechloroarsine (adamsite), and diphenylcyanoarsine. They are dispersed as aerosols and produce their effects by inhalation or by direct action on the eyes. Today diphenylaminechloroarsine is considered obsolete as a riot control agent and has no other application; however, it may still have an environmental impact near former production, storage, and disposal sites.

### PROTECTION

7-33. The protective mask gives adequate protection against field concentrations of vomiting agents. No protective clothing is required.

### PROPERTIES

7-34. All three agents (diphenylchloroarsine, diphenylcyanoarsine, and diphenylaminechloroarsine) are crystalline solids and are usually dispersed by heat as fine particulate smokes. When concentrated, diphenylaminechloroarsine obscurants are canary yellow; diphenylchloroarsine and diphenylcyanoarsine obscurants 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. Agent diphenylaminechloroarsine is different than the other riot control agents; it is more toxic, the effects do not seem to appear immediately and more prolonged systemic effects (for example, headaches, chills, and mental depression).

## PATHOLOGY

7-35. Vomiting agents produce local inflammation of the upper respiratory tract, the nasal accessory, and sinuses. At doses known to induce irritation in other sensory tissues, the eyes show minimal effects.

## SYMPTOMS

7-36. 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.

7-37. The onset of symptoms may be delayed for several minutes after initial exposure (especially with diphenylaminechloroarsine). Therefore, an exposure may occur that can produce mild symptoms before the presence of the obscurants are 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.

## DIAGNOSIS

7-38. 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.

## SELF-AID

7-39. 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 the mask. Continue with assigned 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.

## TREATMENT

7-40. Few cases should reach the MTF because recovery is usually prompt. The exception is with high doses of these agents particularly diphenylaminechloroarsine which can have more systemic effects of malaise, cramping, vomiting, and diarrhea. Symptomatic relief may be obtained by using antiemetics intramuscularly, intravenously, orally, or rectally. Aspirin or acetaminophen may be given to relieve headaches and general discomfort.

## PROGNOSIS

7-41. 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.

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# Chapter 8
# Obscurants

## GENERAL

8-1. Obscurants obscure vision and are used to hide troops, equipment, and areas from detection. Chemicals used to produce obscurants include hexachloroethane, grained aluminum, and HC mixture, special petroleum oils (fog oil [standard gas fuel]), diesel fuel, red phosphorus in a butyl rubber matrix, and white phosporous (WP) plasticized or impregnated in wool felt wedges. There are several newer obscurants, such as phthalic acid and graphite-based obscurants, now used to defeat infrared and millimeter and microwave technologies. Sulfur trioxide-chlorosulfonic acid (FS) solution and titanium tetrachloride are seldom used in current operations. The chemical composition of the petroleum-based and colored obscurants 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 CG and 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 obscurants composed of highly concentrated (60 to 80 percent) polyphosphorus acids.

8-2. High concentrations of obscurant generated in closed spaces are extremely dangerous. High concentrations of HC obscurant generated under these conditions have caused fatalities. In training, terephthalic-acid obscurant should be substituted for HC obscurant. Never use HC munitions indoors or in closed compartments. Should oil obscurant be generated in closed spaces, personnel must immediately evacuate the area or wear self-contained air supply equipment.

## PROTECTION AGAINST OBSCURANTS

8-3. The protective mask gives the respiratory tract and the eyes adequate protection against all obscurants. The protective mask should always be worn when smoke screens are in use. Both FS and titanium tetrachloride 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 obscurants 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 obscurants by rolling down their sleeves. Showering and laundering clothing following exposure to obscurants will also reduce the risk of skin irritation and sores.

## PETROLEUM OIL OBSCURANTS

8-4. These obscurants are produced by vaporizing fuel oils in obscurant generators or engine exhausts.

### PHYSICAL PROPERTIES

8-5. The generator or engine exhaust vaporizes either standard gas fuel or diesel fuel and forces it into the air where it condenses into a dense white obscurant.

### PHYSIOLOGICAL PROPERTIES

8-6. Petroleum oil obscurants are the least toxic obscurants. They seldom produce ill effects. Even prolonged exposure to these obscurants has not been known to cause lipoid pneumonia.

## ZINC OXIDE MIXTURES

8-7. Zinc oxide mixture is a combination of hexachloroethane, grained aluminum powder, and zinc oxide.

## PROPERTIES

8-8. On burning, zinc oxide mixture produces zinc chloride that rapidly absorbs moisture from the air to form a grayish white obscurant. The more humid the air, the more dense the HC obscurant. This obscurant can be dispersed by grenades, candles, pots, artillery shells, and special air bombs. The obscurant of HC has a sharp, acid odor, even at moderate concentrations. The obscurant 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 obscurant are at risk of developing late-onset pulmonary fibrosis (cryptogenic organizing pneumonia).

## PATHOLOGY

8-9. 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 obscurant 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.

## SYMPTOMS

8-10. The obscurant 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.

8-11. 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.

8-12. Finally, case reports of accidental exposure to moderate and high concentrations of HC obscurant 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.

## SELF-PROTECTION

8-13. Put on the mask at once in all concentrations of HC obscurant. 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 feeling shortness of breath.

### TREATMENT

8-14. 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.

### PROGNOSIS

8-15. 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 early use of steroids will prevent fibrosis for HC obscurant and NOx. The severely exposed patient may develop marked progressive dyspnea, cyanosis, fibrosis, and may die.

## SULFUR TRIOXIDE-CHLOROSULFONIC ACID

8-16. Sulfur trioxide-chlorosulfonic acid is a standard obscurant mixture for aircraft spray tanks.

### PROPERTIES

8-17. Sulfur trioxide-chlorosulfonic acid is a heavy, strongly acidic liquid that 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 U.S. military use.

### PATHOLOGY

8-18. Local inflammation of the eyes, respiratory tract (central pulmonary effects), and skin may be seen after severe exposures to the obscurant. Contact with liquid FS produces acid burns.

### SYMPTOMS

8-19. 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.

### SELF-AID

8-20. Wear the mask in all concentrations of FS obscurant 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.

### TREATMENT

8-21. The treatment protocol for sulfur trioxide-chlorosulfonic acid is discussed below.

#### Eye

8-22. 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.

### Skin

8-23. 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.

### Respiratory Tract

8-24. Administer warm, moist air. Use bronchodilators as clinically indicated.

### PROGNOSIS

8-25. The skin burns, conjunctival lesions, and respiratory irritation heal readily. Corneal erosions are more serious and may lead to residual scarring.

## TITANIUM TETRACHLORIDE

8-26. Liquid titanium tetrachloride may be dispersed as an aircraft spray or by explosive munitions, but it is not commonly used.

### PROPERTIES

8-27. Liquid titanium tetrachloride is a corrosive that decomposes on contact with moist air, yielding a dense white obscurant composed of titanium dioxide, titanium oxychloride, and hydrochloric acid.

### PATHOLOGY

8-28. Liquid titanium tetrachloride produces acid burns of the skin or eyes. It may also cause irritation of the upper (central) airways.

### SYMPTOMS

8-29. 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 obscurant may provoke bronchospasm in individuals with underlying reactive airway disease.

### SELF-AID

8-30. Wear the mask in all concentrations of titanium tetrachloride obscurant 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.

### TREATMENT

8-31. Treatment is similar to that for FS.

### PROGNOSIS

8-32. The prognosis is good except in rare instances in which corneal erosions lead to some permanent scarring.

## WHITE PHOSPHORUS OBSCURANT

8-33. The WP obscurant irritates the eyes and nose in moderate concentrations.

### PROPERTIES

8-34. White phosphorus is a pale yellow waxy solid that ignites spontaneously on contact with air. The flame produces a hot, dense white obscurant composed of particles of phosphorus pentoxide. The particles

are converted by moist air into phosphoric acid. White phosphorus is usually dispersed by explosive munitions. 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 reignite if the felt is crushed and the unburned WP is exposed to the atmosphere.

### PATHOLOGY

8-35. For WP burns, see Chapter 9. Inhaled obscurant irritates the upper respiratory tract.

### SYMPTOMS

8-36. Field concentrations of the obscurant may irritate the eyes, nose, and throat.

### SELF-AID

8-37. Wear the protective mask in all concentrations of WP obscurant 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 Chapter 9.

### TREATMENT

8-38. Generally, treatment of WP obscurant irritation is unnecessary. Spontaneous recovery is rapid.

### PROGNOSIS

8-39. No permanent injury has been reported from exposure to WP obscurant at usual field concentrations.

## RED PHOSPHORUS OBSCURANT

8-40. This obscurant is similar to WP obscurant.

## COLORED OBSCURANTS

8-41. These obscurants are produced by explosive dissemination of dyes.

### PHYSIOLOGICAL PROPERTIES

8-42. There are no reports of serious effects produced by exposure to these obscurants. Anecdotally, discoloration of the urine has been noted.

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# Chapter 9
# Incendiary Agents

## GENERAL

9-1. Incendiary agents are used to burn supplies, equipment, and structures. The main agents in this group are thermite, magnesium, WP, and combustible hydrocarbons (including oils and thickened gasoline). Chemical fire extinguishers containing carbon dioxide should not be used in confined spaces to extinguish thermite or magnesium incendiaries. When carbon tetrachloride is in contact with flame or hot metal, it produces a mixture of CG, chlorine, CO, and hydrochloric acid. The field protective mask *does not* protect against some products of combustion such as CO.

## THERMITE

9-2. 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.

## MAGNESIUM AND ITS ALLOYS

9-3. Magnesium burns at about 3600°F (1982.2°C) with a scattering effect similar to that of thermite. Its particles produce deep burns. Healing is slow unless these particles are removed quickly. Removal is usually possible under local anesthesia. When explosive charges have been added to a magnesium bomb, the fragments may be embedded deep in the tissues, causing the localized formation of hydrogen gas and tissue necrosis.

## WHITE PHOSPHORUS

9-4. 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 obscurant irritates the eyes and the nose in moderate concentrations.

### SELF-AID

9-5. 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.

9-6. 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.

9-7. 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.

9-8. Report for treatment as soon as the mission permits.

### TREATMENT

9-9. 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.

9-10. At the earliest opportunity, all WP particles must be removed from the skin.

9-11. 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 obscurant when air strikes them, or by their phosphorescence in the dark. In dark surroundings, fragments are seen as luminescent spots.

9-12. 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.

9-13. 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.

9-14. 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 U.S. 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 WP.

# COMBUSTIBLE HYDROCARBON INCENDIARIES

9-15. Burns may be produced by flame weapons (such as napalm), 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.

## FLAME WEAPON ATTACK

9-16. 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 obscurant is produced. The coolest and least contaminated air is found at floor level.

### Casualties

9-17. 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.

### Protection

9-18. 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 obscurant but is not protective against CO.

### Treatment

9-19. 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.

## FIREBOMB ATTACK

9-20. 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.

### Casualties

9-21. 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.

### First Aid

9-22. Rapidly remove burning clothing and brush off burning fuel with a gloved hand or with several layers of other material. The flames can also be smothered with a wet/damp cover to deprive it of oxygen for combustion.

### Treatment

9-23. In general, treatment is similar to that used after flame weapon attacks.

### Replacement of Body Fluids

9-24. In severe burns, lost body fluid must be replaced quickly to prevent shock.

#### *Intravenous Replacement*

9-25. 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.

#### *Oral Replacement*

9-26. 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:

- Do not remove the casualty's protective clothing or mask.
- 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.
- Remove the casualty's canteen from its carrier. Check the canteen for contamination. If it is contaminated, decontaminate it before using.
- 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.
- Insert the protruding end of the drinking tube into the protective canteen cap. Be sure the seal is tight.
- 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.
- Arrange for the evacuation of the casualty to an uncontaminated area as rapidly as possible.

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# Chapter 10
# Toxic Industrial Chemicals

## GENERAL

10-1. Many TICs' 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, railheads, and ports) facilities. Releases may be by accident or from deliberate acts by enemy forces, terrorists, or belligerents.

10-2. Some of the most widely encountered TICs are ammonia, CO, chlorine gas, hydrogen sulfide, oxides of nitrogen, and hydrazine. Most TICs do not have a specific antidote or treatment. The TICs in this chapter are listed in the International Task Force-40 as military priority industrial chemical hazards. A *chemical hazard* is any chemical manufactured, used, transported, or stored that can cause death or other harm through toxic properties of those materials, including chemical agents and chemical weapons prohibited under the Chemical Weapons Convention as well as toxic industrial chemicals (JP 3-11). The TICs are categorized and discussed in this chapter to a specific medical protocol. For more information on medical protocols, refer to the *Hazardous Materials Injuries: A Handbook for Pre-Hospital Care,* Fourth Edition. See also table 10-1 on page 10-3 for a list of TICs discussed in this chapter.

10-3. Low level exposures or prolonged exposures to occupational and environmental health hazards that may result in some clinically relevant adverse health outcomes to exposed individuals as determined by an appropriate medical/health professional should be reported in the Defense Occupational and Environmental Health Readiness System or DOEHRS. Copies of the report should be submitted to the Military Exposure Surveillance Library for archiving. Refer to Technical Guide 230, DODIs 6055.1, 6055.05, and 6490.03 for more information.

## PROTECTION

10-4. The field protective mask and collective protection systems may have limited protection capabilities against TICs (Service members and MWDs). The field protective mask should be considered an escape device only and personnel exposed to unidentified TICs should egress the contaminated area as rapidly as possible. The self-contained breathing apparatus 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. A health risk assessment is required in order to employ the best available protective equipment (for example, CBRN protective mask, self-contained breathing apparatus) in support of the operation or response. See Appendix H for more information on MWD protection and treatment and ATP 5-19 for more information on risk management.

10-5. The Emergency Response Guidebook recommends first responders enter a site in Level A until the chemical concentration can be evaluated and measured. Selection of personal protective equipment does not solely depend on the type of chemicals. Personal protective equipment should be determined based on following factors: Chemical concentration/amount, chemical phase (liquid, solid, gas), chemical type, and many other factors (weather, physical characteristics, chemical compatibility).

10-6. The filter element/canister of the field protective mask provides only limited protection against obscurant caused by TICs. Duration of the protection depends upon the type of obscurant and its concentration. The filter element/canister does not generate oxygen but filters obscurant 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 19.5 percent oxygen.

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

10-7. There are four levels of protection (Levels A, B, C, D) established by the U.S. Environmental Protection Agency according to U.S. Code of Federal Regulations Title 29, *Labor*, Subtitle B, Chapter XVII, Part 1910, Subpart H, Section 1910.120, *Hazardous Waste Operations and Emergency Response.* The Occupational Safety and Health Administration (OSHA) has also adopted these four levels. The level of skin and respiratory protection provided by the selected protection ensemble determines the protection furnished to personnel. The four OSHA levels of personal protective equipment are as follows:

- Level A is to be selected when the greatest level of skin, respiratory, and eye protection is required. The following constitute Level A equipment:
  - Positive pressure, full face-piece self-contained breathing apparatus.
  - Totally encapsulating chemical-protective suit.
  - Gloves (outer and inner), chemical resistant.
  - Boots, chemical resistant, steel toe, and shank.
- Level B is worn when the highest level of respiratory protection is necessary but a lesser level of skin protection is needed. The following constitute Level B equipment:
  - Positive pressure, full face-piece self-contained breathing apparatus.
  - Hooded chemical resistant clothing.
  - Gloves (outer and inner), chemical resistant.
  - Boots, chemical resistant, steel toe, and shank.
  - Boot cover, outer, chemical resistant.
- Level C is worn when the concentration and type of airborne substance is known and the criteria for using air purifying respirators are met. Level C protection is similar to that of MOPP Level 4. The following constitute Level C equipment:
  - Full-face or half-mask, air purifying respirators.
  - Hooded chemical resistant clothing.
  - Gloves (outer and inner), chemical resistant.
  - Boots, chemical resistant, steel toe, and shank.
  - Boot cover, outer, chemical resistant.
- Level D is a work uniform affording minimal protection. It is worn for nuisance contamination only. The following constitute Level D equipment:
  - Coveralls.
  - Boots, chemical resistant, steel toe, and shank.

10-8. Toxicity level is indicated for each agent (see table 10-1). The following descriptions summarize the toxicity level:

- Level 1—Materials only slightly hazardous to health. Wear self-contained breathing apparatus.
- Level 2—Materials hazardous to health. Wear self-contained breathing apparatus.
- Level 3—Materials extremely hazardous to health. Full protective clothing, including self-contained breathing apparatus, rubber gloves, boots and bands around legs, arms, and waist are required. No skin surface should be exposed.
- Level 4—Minimal contact required to cause death. Normal protective clothing not adequate. Special protective clothing designed against the hazard is required.

**Table 10.1. List of military priority industrial chemical hazards**

| *Chemical name* | *Toxicity level* | *Personal protective equipment level* |
|---|---|---|
| Hydrogen Chloride | 4 | A |
| Sulfuric Acid | 4 | A |
| Hydrogen Fluoride | 4 | A |
| Nitric Acid | 4 | A |
| Hydrogen Bromide | 4 | A |
| Ammonia | 3 | A |
| Carbon Monoxide | 1 | B |
| Phosgene | 3 | B |
| Hydrogen Cyanide | 3 | B |
| Chlorine | 4 | A |
| Cyanogen Chloride | 4 | A |
| Ethylene Oxide | 2 | C |
| Formaldehyde | 2 | B |
| Methyl Bromide | 3 | B |
| Ethyline Dibromide | 3 | B |
| Mercury | 3 | C |
| Nitrogen Dioxide | 2 | C |
| Octamethyl Pyrophosphoramide | 4 | B |
| Sulfur Dioxide | 3 | B |
| Hydrogen Sulfide | 2 | C |

# ACIDS

10-9. Hydrogen chloride, sulfuric acid, hydrogen fluoride, nitric acid, and hydrogen bromide are categorized as acids.

## PROPERTIES

10-10. Acids may be encountered as solids, liquids, gases, or aerosols. Liquids may also evaporate to form vapor. Vapors and gases usually have a characteristic pungent odor. The most common acids are hydrochloric, nitric, and sulfuric.

## RELEVANCE TO MILITARY OPERATIONS

10-11. 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 released 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.

## PATHOLOGY

10-12. 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 increases, however, peripheral pulmonary effects (pulmonary edema) may also be seen.

## SYMPTOMS

10-13. Signs and symptoms may include severe burns with pain; destruction of the cornea that 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.

## DIAGNOSIS

10-14. 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 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 that is important initially to rule out is nerve agent exposure, easily differentiated by the papillary changes, sweating, muscular fasciculations, and mental status changes.

## PROTECTION

10-15. The toxicity level for acids is Level 4. Responders 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.

## TREATMENT

10-16. Combat medics/Corpsmen/Air Force medics care is as follows:

- 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.
- Cover burns with sterile gauze (loosely).
- Evacuate casualty.

10-17. Medical treatment facility care is as follows:

- 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, if available.
- Watch for secondary pneumonitis, and treat with antibiotics once a causative organism has been identified.
- Maintain patient at enforced bed rest (semiseated if tolerated by patient) if estimated inhaled dose of acid is high.
- 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.

### PROGNOSIS

10-18. 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.

## AMMONIA

10-19. Ammonia 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.

### PROPERTIES

10-20. 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.

### RELEVANCE TO MILITARY OPERATIONS

10-21. 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.

### PATHOLOGY

10-22. 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.

### SYMPTOMS

10-23. 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 can lead to pulmonary edema.

### DIAGNOSIS

10-24. 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 should be presumed to have been exposed to a caustic vapor or gas, which would include $NH_3$ 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.

### PROTECTION

10-25. The toxicity level for ammonia is Level 3. Responders 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.

### TREATMENT

10-26. Combat medics/Corpsmen/Air Force medics care is as follows:

- Remove casualty from contamination zone and decontaminate.
- Thoroughly flush exposed eyes with water.
- Administer oxygen, 10-12L using nonrebreather mask.
- Administer one or two glasses of water in cases of ingestion, if casualty is conscious.
- Monitor and treat for shock, as necessary.

10-27. Medical treatment facility care is as follows:

- Initiate IV, sodium lactate (or normal saline).
- Observe for and manage pulmonary edema.
- Observe for airway compromise.
- Administer beta agonists to manage bronchospasm, if available.
- Irrigate eye; apply the scleral lens with attached tube for ocular irrigation.

### PROGNOSIS

10-28. The mortality is high following severe exposure. With low concentrations, recovery is usually rapid, although bronchitis may persist.

## ASPHYXIANTS

10-29. Asphyxiants are gases or vapors of volatile liquids. An asphyxiant is a substance that can cause unconsciousness or death by suffocation (asphyxiation).

### PROPERTIES

10-30. Some asphyxiants may be flammable or even explosive. One of the asphyxiants that is of military concern is CO. Pure CO is a colorless, tasteless, odorless gas. It is lighter than air, into which it diffuses rapidly.

### RELEVANCE TO MILITARY OPERATIONS

10-31. Carbon monoxide is formed by gun blasts, bursting shells, internal combustion engines, fires in confined spaces, and the incomplete combustion of fuels. It can also be a metabolic by-product of chemicals like the industrial solvent methylene chloride.

### PATHOLOGY

10-32. Tissue hypoxia is caused chiefly by displacement of oxygen from binding sites on blood hemoglobin. Carbon monoxide has an affinity for these sites that is 200 times that of oxygen, and it forms carboxyhemoglobin, 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.

### SYMPTOMS

10-33. Carbon monoxide is insidious in its actions, and poisoning may occur without 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.

### DIAGNOSIS

10-34. The diagnosis is made from the circumstances of exposure and the appearance of cherry-red skin and mucous membranes. Exposure to 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 carboxyhemoglobin. Both cyanide and CO poisoning will produce lactic acidosis.

### PROTECTION

10-35. The toxicity level for asphyxia is Level 1. 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. At a minimum, OSHA Level B affords the greatest protection and should be worn if the substance or concentrations are unknown, especially in a confined space.

### TREATMENT

10-36. Combat medics/Corpsmen/Air Force medics care is as follows:

- Remove casualty from contamination zone and decontaminate.
- Thoroughly flush exposed eyes with water.
- Administer oxygen, 10-12L using nonrebreather mask.

10-37. Medical treatment facility care is as follows:

- Use hyperbaric oxygen if chamber is available.
- Initiate IV, sodium lactate (or normal saline).
- Alleviate seizures by administering diazepam.
- Administer phenobarbital if diazepam is ineffective.
- Observe for circulatory collapse.
- Observe for airway compromise.
- Administer beta agonists to manage bronchospasm, if available.
- Irrigate eye; apply the scleral lens with attached tube for ocular irrigation.

### PROGNOSIS

10-38. 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.

## BLISTERING AGENTS

10-39. Phosgene and AC are both in the same protocol as blister (vesicant) agents. A general class of compounds that is extremely irritating to the skin and mucous membranes. Blistering agents may be solids, liquids, or gases. Mustard and Lewisites are oily liquids (colorless to dark brown) with varying odors as follows:

- Mustard—a garlic or horseradish odor.
- Nitrogen mustard—no odor or fishy, irritating odor.
- Lewisite—fruity to geranium-like smell.

10-40. For the rest of the information on blister (vesicant) agents, refer to Chapter 5.

# CHLORINE

10-41. Chlorine is an irritant and blistering agent.

## PROPERTIES

10-42. 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.

## RELEVANCE TO MILITARY OPERATIONS

10-43. 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.

## PATHOLOGY

10-44. Because chlorine is intermediate in both aqueous solubility and chemical reactivity, it exhibits both central pulmonary effects and also peripheral pulmonary effects 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).

## SYMPTOMS

10-45. 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.

## DIAGNOSIS

10-46. 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.

## PROTECTION

10-47. The toxicity level for blistering agents is Level 3. Responders 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, self-contained breathing apparatus) and fully encapsulated chemically protective suits (OSHA Level A) should be worn when entering a contaminated confined space. Although MOPP 4 will usually be adequate in open-air contaminated areas, the standard mask filter will not filter out chlorine.

## TREATMENT

10-48. Combat medics/Corpsmen/Air Force medics care is as follows:

- 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.

10-49. Medical treatment facility care is as follows:

- 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.

### PROGNOSIS

10-50. Individuals with a mild or short-term exposure have excellent prognosis. 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.

## CYANOGEN COMPOUNDS

10-51. Cyanogen compounds have strong irritant and choking effects.

### PROPERTIES

10-52. Cyanogen compounds specifically CK, are a highly volatile and toxic chemical asphyxiant that interferes with the body's ability to use oxygen. Exposure to CK can be rapidly fatal. It has whole-body (systemic) effects, particularly affecting those organ systems most sensitive to low oxygen levels such as—

- The CNS (brain).
- The cardiovascular system (heart and blood vessels).
- The pulmonary system (lungs).

10-53. Cyanogen chloride's vapors are extremely irritating and corrosive. Cyanogen chloride is a CW agent (military designation CK). It is used commercially in chemical synthesis and fumigation. Cyanogen chloride's boiling point is 57°F (13.8°C) and its melting point is 21°F (-6.1°C).

### RELEVANCE TO MILITARY OPERATIONS

10-54. The major sources of CK during military operations are water discharges from some metal mining processes, organic chemical industries, iron and steel plants or manufacturers, and publicly owned wastewater treatment facilities. Other cyanide sources include vehicle exhaust, releases from certain chemical industries, burning of municipal waste, and use of cyanide-containing pesticides.

### PATHOLOGY

10-55. Cyanogen chloride acts in two ways. Its systemic effects are similar to those of hydrocyanic acid, but it also has local irritant effects on the eyes, upper respiratory tract, and lungs. Cyanogen chloride damages the respiratory tract, resulting in severe inflammatory changes in the bronchioles, and congestion and edema in the lungs. The fluid in the lungs may accumulate much more rapidly than phosgene poisoning. All concentrations of cyanogen chloride produce eye irritation and lacrimation.

## SYMPTOMS

10-56. Early symptoms of cyanide poisoning include lightheadedness, giddiness, rapid breathing, nausea, vomiting (emesis), feelings of neck constriction and suffocation, confusion, restlessness, and anxiety. Accumulation of fluid in the lungs (pulmonary edema) may complicate severe intoxications. Rapid breathing is soon followed by respiratory depression/respiratory arrest (cessation of breathing). Severe cyanide poisonings progress to stupor, coma, muscle spasms (in which head, neck, and spine are arched backwards), convulsions (seizures), fixed and dilated pupils, and death. The CNS is the most sensitive target organ of cyanide poisoning. Cardiovascular effects require higher cyanide doses than those necessary for CNS effects. In serious poisonings, the skin is cold, clammy, and diaphoretic. Blue discoloration of the skin may be a late finding. Severe signs of oxygen deprivation in the absence of blue discoloration of the skin suggest cyanide poisoning.

## DIAGNOSIS

10-57. In an acute setting, diagnosis is based on characteristic signs and symptoms, coupled with an index of suspicion based on environmental setting. Consider the diagnosis of CK poisoning in patients with irritation of the eyes, nose, respiratory tract followed by rapid collapse or seizures accompanied by metabolic acidosis and decreased oxygen consumption.

## PROTECTION

10-58. The toxicity level for cyanogen compounds is Level 4. Positive-pressure, self-contained breathing apparatus with OSHA Level A protective suit are recommended in response situations that involve exposure to CK.

## TREATMENT

10-59. Combat medics/Corpsmen/Air Force medics care is as follows:

- 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 to all casualties.
- Administer amyl nitrite by inhalation, if available.
- Monitor and treat for shock, if necessary.
- Evacuate casualty.

10-60. Medical treatment facility care is as follows:

- Manage airway.
- Initiate IV.
- Administer a cathartic (such as magnesium sulfate) if ingestion exposure has occurred.
- Inject sodium nitrite.
- Administer IV; a solution of sodium thiosulfate immediately following sodium nitrite.
- Administer diazepam to alleviate seizure.
- Treat eye injuries.

## PROGNOSIS

10-61. The prognosis in patients with CK poisoning is better in those with low-level exposures whose symptoms resolve after they are removed from exposure. However, the prognosis is generally poor in patients who suffer cardiac arrest secondary to cyanide toxicity, even if antidotes are administered promptly.

# EPOXY COMPOUNDS

10-62. Uses for epoxy compounds include medical sterilizers, pharmaceuticals, protective coatings, road paving, caulking compounds, plasticizers, and vinyl resin stabilizers.

## PROPERTIES

10-63. Epoxy compounds are liquids or solids. Some compounds may be flammable. One of the epoxy compounds that is of military concern is ethylene oxide. It 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 Service members is at 800 parts per million and the lethal concentration for 50 percent of those exposed is 4350 parts per million. The vapors are flammable and explosive.

## RELEVANCE TO MILITARY OPERATIONS

10-64. Ethylene oxide is used to sterilize surgical instruments, as an agricultural fungicide, to fumigate food items and textiles, and in organic synthesis.

## PATHOLOGY

10-65. 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.

## SYMPTOMS

10-66. 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 well as mental changes indicative of encephalopathy.

## DIAGNOSIS

10-67. 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.

## PROTECTION

10-68. The toxicity level for epoxy compounds is Level 2. Responders must determine the concentration level in the contaminated area and assume the appropriate protective level before attempting to rescue or care for casualties. The protection level should be at least OSHA Level C (a respirator with full 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.

## TREATMENT

10-69. Combat medics/Corpsmen/Air Force medics care is as follows:

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

10-70. Medical treatment facility care is as follows:

- Continue IV and oxygen therapy, as needed.
- Administer 30 to 100 gram of activated charcoal as a suspension in 1 cup of water (12.5 to 25 gram for children), if ingestion has occurred.
- Administer a cathartic such as magnesium sulfate following the activated charcoal. Give 10 to 15 gram in a glass of water (5 to 10 gram for children).
- Irrigate the eyes, as needed.

### PROGNOSIS

10-71. 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 CNS 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.

## FORMALDEHYDE

10-72. Formaldehyde is used as a disinfectant, preservative, and fumigant.

### PROPERTIES

10-73. Formaldehyde is a colorless, pungent irritant gas (created by oxidation of methyl alcohol). The gas is very soluble in water, alcohol, and ether. Formaldehyde usually is produced and marketed as a 37 percent (weight) solution in water.

### RELEVANCE TO MILITARY OPERATIONS

10-74. Formaldehyde is an important chemical used widely by industry to manufacture building materials and numerous household products. Sources of formaldehyde include pressed wood products (hardwood, plywood, wall paneling, particleboard, fiberboard, and furniture made with these pressed wood products), combustion sources, durable press drapes, other textiles, and glues and adhesives.

### PATHOLOGY

10-75. Formaldehyde is irritating to the skin, eyes, and respiratory tract. Poisoning can occur by ingestion, inhalation, and skin absorption. Severity of effects following exposure is dependent on the concentration.

### SYMPTOMS

10-76. Symptoms may include irritation of the skin, eyes, nose, throat, and upper respiratory tract with episodes of coughing. Abdominal pain, nausea, and vomiting may be seen following ingestion. Burning sensation in chest, dizziness, exhaustion, convulsions, shock, and coma may also occur.

### DIAGNOSIS

10-77. 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.

### PROTECTION

10-78. The toxicity level for formaldehyde is Level 2. Responders must determine the concentration level in the contaminated area and assume the appropriate protective level before attempting to rescue or care for casualties. Protection level should be at least OSHA Level B (chemical resistant impervious clothing, boots, and gloves). A self-contained breathing apparatus should be worn as well.

### TREATMENT

10-79. Combat medics/Corpsmen/Air Force medics care is as follows:

- Remove casualty from the contamination zone and decontaminate.
- Flush exposed eyes with water.
- Administer oxygen, as needed.
- Administer one or two glasses of water in cases of ingestion, if casualty is conscious.
- Administer activated charcoal following emesis.

10-80. Medical treatment facility care is as follows:

- Initiate IV and continue oxygen therapy, as needed.
- Administer a cathartic such as magnesium sulfate, if ingestion exposure has occurred.
- Administer diazepam to alleviate seizure.
- Administer phenobarbital sodium IV.
- Observe for pulmonary edema and airway compromise.
- Administer a beta agonist, if bronchospasm occurs.

### PROGNOSIS

10-81. Those with short-termed, acute exposure usually have a prompt resolution of symptoms after removal to an uncontaminated environment. Those who ingested formaldehyde may suffer severe injury to the upper gastrointestinal tract. Deaths have occurred due to ingestion of very little amount of water but with higher concentration of formaldehyde.

## HYDROCARBONS, HALOGENATED ALIPHATIC

10-82. Methyl bromide and ethylene dibromide are categorized as halogenated aliphatic hydrocarbon compounds.

### PROPERTIES

10-83. Halogenated aliphatic hydrocarbon compounds are practically odorless and colorless liquids with excellent solvent properties and low flammability. Halogenated aliphatic hydrocarbons are used in industry as solvents, chemical intermediates, fumigants and insecticides. They are found in the chemical, paint and varnish, textile, rubber, plastics, dye-stuff, pharmaceutical and dry-cleaning industries.

### RELEVANCE TO MILITARY OPERATIONS

10-84. Methyl bromide and ethylene dibromide have been used as broad spectrum fumigants and as pesticides used in the control of pest insects, nematodes, weeds, pathogens, and rodents. Studies in humans indicate that the lung may be severely injured by the acute (short-term) inhalation of methyl bromide. Acute and chronic (long-term) inhalation of methyl bromide can lead to neurological effects in humans. Neurological effects have also been reported in animals. Animal studies indicate that chronic exposure to ethylene dibromide may result in toxic effects to the liver, kidney, and the testis, irrespective of the route of exposure. Limited data on men occupationally exposed to ethylene dibromide indicate that long-term exposure to ethylene dibromide can impair reproduction by damaging sperm cells in the testicles.

### PATHOLOGY

10-85. Halogenated aliphatic hydrocarbon intoxication may occur by inhalation, ingestion, or skin contact. These compounds are highly irritating and may cause permanent eye damage following exposure. Halogenated hydrocarbons will depress the CNS. Many of these compounds are toxic to the liver, kidneys, and heart.

### SYMPTOMS

10-86. The symptoms may include severe skin, eye, and mucous membrane irritation and associated pain. Dilation of pupils, sluggishness, confusion, headache, abdominal pain, nausea, vomiting, and loss of consciousness may also occur.

### DIAGNOSIS

10-87. No specific test is available; however, elevated levels in serum might indicate that an exposure has occurred. Detection of bromide below toxic levels does not rule out poisoning.

### PROTECTION

10-88. The toxicity level for Halogenated aliphatic hydrocarbon is Level 3. Responders should wear OSHA Level B protection (chemical resistant impervious clothing, boots, and gloves). A self-contained breathing apparatus should be worn as well. Medical personnel caring for contaminated casualties should be at the same protective posture.

### TREATMENT

10-89. Combat medics/Corpsmen/Air Force medics care is as follows:

- Remove casualty from contamination zone and thoroughly flush eyes with water.
- Administer 1 to 2 glasses of water to patient following ingestion exposure.
- Administer activated charcoal- recommended dosage is 30 to 100 grams as a suspension in 1 cup of water.
- Start an IV or saline lock.
- Administer 100 percent oxygen if available.

10-90. Medical treatment facility care is as follows:

- Initiate IV (5 percent dextrose in water).
- Administer a cathartic such as magnesium sulfate with a recommended dosage of 10 to 15 gram in a glass of water, if ingestion occurred.
- Observe for pulmonary edema, circulatory collapse, and airway compromise.
- Administer a beta agonist if bronchospasm occurs.
- Irrigate properly and apply the scleral lens with attached tube for ocular irrigation.

### PROGNOSIS

10-91. Prognosis will be dose and time/exposure dependent. Organ systems that may be affected are pulmonary, cardiac, neurologic, gastrointestinal, renal, hepatic, and hematologic. The dermatologic system will likely be involved. The pulmonary system will most commonly be involved.

## MERCURY COMPOUNDS

10-92. Mercury poisoning can occur from ingestion of seafood from contaminated waters.

### PROPERTIES

10-93. Mercury is a heavy, silver-white metallic element used in thermometers, paints, lamps, explosive and electrical apparatus.

### RELEVANCE TO MILITARY OPERATIONS

10-94. The most important organic mercury compound, in terms of human exposure, is methyl mercury. This type of mercury exposure occurs primarily through the diet, with fish and fish products as the dominant

source. Sources of past exposure to methyl mercury include fungicide-treated grains and meat from animals fed such grain.

### PATHOLOGY

10-95. All forms of mercury are poisonous due to the high toxicity of the mercuric ion. Elemental mercury exhibits most of its toxicity following inhalation of its vapor. Although skin absorption of liquid mercury does occur, the rate at which it does so is negligible when compared to that for inhalation exposure of mercury vapor.

### SYMPTOMS

10-96. The major systems impacted by human inhaling or ingesting mercury are the kidneys and CNS. Acute exposure to high levels of mercury in humans causes metallic taste, nausea, vomiting, tremors, irritability, insomnia, memory loss, neuromuscular changes, headaches, slowed sensory and motor nerve functions, reduction in cognitive functions, dyspnea, cough, and in some rare cases, blindness and deafness.

### DIAGNOSIS

10-97. Diagnosis will initially be empiric and based on clinical (for example, blood, urine, and scalp hair analysis) 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.

### PROTECTION

10-98. The toxicity level for mercury is Level 3. Responders should wear OSHA Level C protection. Medical personnel caring for contaminated casualties should be at the same protective posture.

### TREATMENT

10-99. Combat medics/Corpsmen/Air Force medics care is as follows:

- Remove casualty from contamination area.
- Administer water (1 cup) to victims following ingestion exposure.
- Monitor for shock.
- Administer activated charcoal following emesis.

10-100. Medical treatment facility care is as follows:

- Initiate IV (sodium lactate or normal saline).
- Administer a cathartic such as magnesium sulfate to victim if ingestion exposure has occurred.
- Observe for airway compromise.
- Administer a beta agonist if bronchospasm occurs.

### PROGNOSIS

10-101. Prognosis is dependent upon the severity of exposure and type of mercury compound. Recovery is possible if exposure is mild. Severe exposure to mercury may have long-term neurological residual effects.

## NITROGEN COMPOUNDS

10-102. Two of the most common NOx are nitric oxide and nitrogen dioxide.

### PROPERTIES

10-103. The oxides of nitrogen are formed by a variety of chemical and natural biological reactions, including combustion and metabolism. Most are gases. The solid form will evaporate under ambient conditions; liquid form is usually compressed. Some compounds may be flammable or explosive. Nitrogen oxides mixed with water form nitric and nitrous acids.

## RELEVANCE TO MILITARY OPERATIONS

10-104. Nitrogen dioxide is used to produce rocket fuels, explosives, and other chemicals. Nitric oxide is used to bleach rayon and produce nitric acid.

## PATHOLOGY

10-105. Nitrogen oxide exposure can occur when breathing air that contains it especially in industrial countries that burn coal, oil, diesel fuel, and natural gas. Although NOx are only slightly irritating in low concentrations, higher concentrations may cause chemical burns. Acute exposure to high concentrations may be fatal within hours. Severe effects may take 24 hours to manifest, and follow a symptom-free period of several hours. Some may act as CNS depressants.

## SYMPTOMS

10-106. Exposure symptoms may include red inflamed skin, inflamed eyes and mucous membranes, cyanosis, coughing, burning sensation of the throat and chest, shortness of breath, restlessness, insomnia, pulmonary edema and in some cases, coma. Exposure to high level of NOx can cause unconsciousness, mental confusion, difficulty breathing, dizziness, fatigue, and even death.

## DIAGNOSIS

10-107. 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.

## PROTECTION

10-108. The toxicity level for nitrogen oxide is Level 2. Responders should wear OSHA Level C protection. Medical personnel caring for contaminated casualties should be at the same protective posture.

## TREATMENT

10-109. Combat medics/Corpsmen/Air Force medics care is as follows:

- Remove casualty from contamination area.
- Flush eyes with water thoroughly.
- Administer oxygen using a nonrebreather mask.
- Administer 1-2 glass of water if ingestion exposure has occurred.
- Administer activated charcoal.

10-110. Medical treatment facility care is as follows:

- Initiate IV (5 percent dextrose in water).
- Administer a cathartic such as magnesium sulfate to victim if ingestion exposure has occurred.
- Observe for pulmonary edema.
- Observe for airway compromise.
- Administer a beta agonist if bronchospasm occurs.

## PROGNOSIS

10-111. Prognosis is dependent upon the severity of exposure. Recovery is possible if exposure is mild. Severe or long-term exposure to NOx may have long-term effects such as respiratory problems and reduction in lung function.

# ORGANIC PHOSPHORUS COMPOUNDS

10-112. The organophosphorus compounds (often incorrectly called *organophosphate* compounds) are solids or liquids used as pesticides.

## PROPERTIES

10-113. Some organophosphorus compounds 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.

## RELEVANCE TO MILITARY OPERATIONS

10-114. 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.

## PATHOLOGY

10-115. 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.

## SYMPTOMS

10-116. Symptoms are the same as for nerve agent poisoning (see Chapter 3).

## DIAGNOSIS

10-117. 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.

## PROTECTION

10-118. The toxicity level for organic phosphorus is Level 4. Responders 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 B protection, depending upon concentration. Mission-oriented protective posture 4 will usually be adequate in open-air contaminated areas.

## TREATMENT

10-119. Combat medics/Corpsmen/Air Force medics care is as follows:

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

10-120. Medical treatment facility care is as follows:

- 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 gram 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.

### PROGNOSIS

10-121. 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.

## INORGANIC SULFUR COMPOUNDS

10-122. Sulfur dioxide is highly soluble in water.

### PROPERTIES

10-123. Sulfur dioxide will immediately form a corrosive acid when it reacts with water. It 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). Concentrations above 39 parts per million can cause severe respiratory tract injury.

### RELEVANCE TO MILITARY OPERATIONS

10-124. Sulfur dioxide is a widely used and readily available industrial compound. Sulfur dioxide gas may be released in hazardous quantities during a sulfur pile fire. Sulfur piles are commonly found at fertilizer plants and petroleum refineries.

### PATHOLOGY

10-125. 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.

### SYMPTOMS

10-126. Sulfur dioxide exposure will result in immediate symptoms due to its high water solubility. Symptoms include eye irritation, headache, irritation to mucous membranes and to upper (central) airways (with concomitant coughing, sneezing, hoarseness, wheezing, stridor, or laryngospasm), and dyspnea (shortness of breath, chest tightness) indicative of incipient pulmonary edema, shock, circulatory collapse, seizures, and coma.

### DIAGNOSIS

10-127. Diagnosis in the acute setting is usually empiric and includes other TICs or CW agents that produce eye irritation and acute pulmonary symptoms.

### PROTECTION

10-128. The toxicity level for inorganic sulfur compounds is Level 3. Responders should wear at least OSHA Level B to enter the contaminated area. Medical personnel caring for contaminated casualties should be at the same protective posture.

### TREATMENT

10-129. Combat medics/Corpsmen/Air Force medics care is as follows:

- Mask casualty, remove casualty from the contaminated area, or both.
- Manage airway.
- Decontaminate casualty.
- Administer oxygen using a nonrebreather mask.
- Irrigate casualty's eyes with copious amounts of water or, preferably, sterile isotonic saline.
- Start an IV or saline lock.
- Administer CANA or other forms of diazepam to control seizures.

10-130. Medical treatment facility care is as follows:

- 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.

### PROGNOSIS

10-131. 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.

## SULFUR COMPOUNDS

10-132. Sulfides are widely used as fungicides.

### PROPERTIES

10-133. Sulfides may be solids or liquids. One of the sulfur compounds that is of military concern is hydrogen sulfide. 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 parts per million. Olfactory fatigue occurs at 100 parts per million.

### RELEVANCE TO MILITARY OPERATIONS

10-134. Hydrogen sulfide may exist in petroleum products, natural gas, and maybe a by-product of certain processes that occur in leather tanning and the production of rayon fibers. It may also be generated from bacterial action in the environment and occur in soil, sewage material, and manure collections.

### PATHOLOGY

10-135. Hydrogen sulfide ranks with CO and cyanide in terms of inhalational toxicity. In low concentrations (less than 0.15 mg per liter), hydrogen sulfide may produce inflammation of the eyes, nose, and throat if inhaled 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 Chapter 4). 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.

## SYMPTOMS

10-136. 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, 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.

## DIAGNOSIS

10-137. Diagnosis is initially empiric and similar to that for acid or chlorine exposure. 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.

## PROTECTION

10-138. The toxicity level for sulfur compounds is Level 2. Responders should wear OSHA Level C protection. Medical personnel caring for contaminated casualties should be at the same protective posture.

## TREATMENT

10-139. Combat medics/Corpsmen/Air Force medics care is as follows:

- Remove casualty from contamination zone and decontaminate with soap and water.
- 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.
- Administer 100 percent oxygen if available.

10-140. Medical treatment facility care is as follows:

- 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. Sodium nitrite therapy is the primary pharmaceutical treatment for severe cases but its efficacy has never been conclusively demonstrated. It should be considered for severe cases that present soon after exposure. 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.

- 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.

## PROGNOSIS

10-141. 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.

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## Appendix A

# Recognition of a Chemical Agent Casualty

## RECOGNITION

A-1. 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. When CW 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.

## TYPES OF CASUALTIES

A-2. On the battlefield, the following types of casualties may be seen:

- Conventional casualties—
  - Conventional casualties with no chemical injury and with no contamination of their clothing and equipment.
  - Conventional casualties with no chemical injury but with contamination of their clothing and equipment.
- Direct chemical casualties—
  - Chemical casualties with no other injury.
  - Mixed casualties with conventional and chemical injuries. Since chemical munitions often include burst charges, such injuries may occur as part of a CW 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 CW agents. 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 preexisting medical conditions. Whenever mixed casualties are encountered, the nature of the interactions, or synergism, of the coexisting diagnoses must be considered. For example, radiation casualties who are also exposed to HD are at far greater risk of medical complications than casualties exposed to just radiation or just HD.
- Indirect chemical casualties—
  - Casualties suffering combat and operational stress reactions occur often in warfare, but may be more frequent where the CW agents 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 CW agents. The differential diagnosis between the patients with psychological effects and chemical patients may sometimes be difficult. Combat and operational stress reaction patients could outnumber all others.
  - Some CW 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.

- 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). The possibility of heat injury and the psychological effects of wearing the protective ensemble may degrade mission effectiveness.

# RECOGNITION OF CHEMICAL CASUALTIES

A-3. Under operational conditions, the medical situation may be complicated by the psychological effects of an incapacitating agent. To determine if the casualty has been caused by a CW 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 obscurant?
- Was anyone else affected and if so, what effects and were those effects similar?
- Did the casualty notice any unusual smell?

A-4. To recognize a chemical casualty, the identity of the agent must be determined.

- 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.
- The patient should also be questioned concerning a delay between the onset of symptoms and exposure or contamination as follows:
  - 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?
- To assess the dose of a CW 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

# Handling of Contaminated Clothing and Equipment at Medical Treatment Facilities

## CONTAMINATED CLOHING AND EQUIPMENT

B-1. 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. Contaminated clothing and equipment are removed from the casualty as soon as possible. Clothing removal must not compromise the individual's medical condition.

### DISPOSITION OF CONTAMINATED CLOTHING AND BLANKETS

B-2. 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 contaminated dump site should be at least 75 meters from the drop-off/arrival point; downwind from the patient decontamination site of the MTF and living quarters. The contaminated waste dump should be clearly marked with standard chemical contamination markers (see ATP 4-02.7/MCRP 4-11.1F/NTTP 4-02.7/AFTTP 3-42.3 and ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37).

B-3. 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.

B-4. The medical officer should notify designated authority of the—

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

### REPLACEMENT OF CONTAMINATED BLANKETS

B-5. 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 replacement requirements are known. Disposable foil blankets may be used in place of cloth blankets.

B-6. If the tactical situation permits, replacements are requisitioned through the normal medical logistics channels. Emergency resupply may be requested from the nearest general supply support unit.

## THE CHEMICAL PROTECTIVE ENSEMBLE

B-7. All personnel handling or treating chemically contaminated casualties must be at MOPP 4. Personnel must also be at MOPP 4 while decontaminating litters, ambulances, and other equipment.

B-8. The chemical protective overgarment is not removed until the danger of contamination has been eliminated. Contaminated chemical protective overgarments/Joint Service Lightweight Integrated Suit technology may be worn safely in a contaminated environment for 24 hours. The uncontaminated suit may be worn for 45 days or as prescribed in ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37. This publication gives

further guidance on individual protection using the complete ensemble, and 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 collective protection or clean areas be located for the provision of medical care.

## DISPOSITION OF CONTAMINATED GLOVES AND CHEMICAL PROTECTIVE OVERGARMENTS DURING AIR, LAND, AND NAVAL OPERATIONS

B-9. Contaminated gloves and overgarments are placed in a closed plastic bag and segregated for further disposal.

B-10. 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-11. 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 millimeters thick) and stored for later transfer to a shore facility hazardous material team. For ships in port, double-bag contaminated suits for turn-in to shore-based disaster preparedness/hazardous material teams for disposal, see NTTP 3-20.31 and Office of the Chief of Naval Operations Manual (OPNAV M-) 5090.1.

## IMPERMEABLE PROTECTIVE CLOTHING, APRONS, GLOVES, AND BOOTS

B-12. 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).

B-13. The ratio of a slurry mix is 1:5 (1 gallon hot water to 5 pounds of super tropical bleach). For more information on slurry mix see ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37. 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: aeration, soap and water or slurry.

### Aeration

B-14. 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.

### Soap and Water

B-15. 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.

### Slurry

B-16. 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.

## PROTECTIVE MASKS, WEB, CANVAS, AND LEATHER EQUIPMENT

B-17. This section will discuss protective masks, web, canvas, and leather equipment decontamination.

### Protective Masks

B-18. Masks that have been exposed to droplets or vapor may be decontaminated. If the mask is decontaminated immediately after contamination (thus avoiding absorption of the agent into the rubber), the following methods may be used:

- Decontaminate the mask with hot soapy water and rinse with clear water or decontaminate with decontamination kit. 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.
- Mask and carriers lightly contaminated by vapor only may be decontaminated by airing in sunlight and wind.

### Web and Canvas Equipment

B-19. 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 (see ATP 3-11.32/MCWP 3-37.2/NTTP 3- 11.37).

### Leather Equipment

B-20. Leather quickly absorbs liquid CW agents. Initial decontamination should be done as rapidly as possible by using the M295 Decontamination Kit, Individual Equipment. 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 ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37 for additional information on decontamination of leather equipment.

## PROTECTION AND CARE OF LITTERS

B-21. Provide emergency protection of canvas litters by covering them with materials such as ponchos, plastic sheeting, or shelter halves.

### DECONTAMINATION OF LITTERS

B-22. If possible, take litters apart and decontaminate components.

### Canvas

B-23. 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.

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***Note.*** Sodium carbonate is not readily available or fielded in the medical equipment set, Patient Decontamination and Chemical Treatment.

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### Wood

B-24. 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.

### Metal (Unpainted)

B-25. Use soap and water or available decontamination solution.

***Note.*** The only place where 5 percent hypochlorite (full strength liquid bleach) solution is used is to decontaminate (plastic mesh) litters that are designed to be decontaminated. Allow the litter to air dry. Litters should be rinsed with water before use. See ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37 for more information.

B-26. If the litter cannot be taken apart, decontaminate it by flushing it with copious amounts of hot soapy water. Then aerate the litter outdoors until dry or discard.

## DECONTAMINABLE LITTER

B-27. Apply a 5 percent hypochlorite solution to the entire surface of the litter and handles/poles. Allow the solution to remain on the litter for 10 to 15 minutes and then rinse thoroughly with fresh water. If the 5 percent hypochlorite solution is not available, remove gross contamination by scraping with a stick or other object, then use the M295 Decontamination Kit, Individual Equipment. Litters must be removed from the patient care area of the patient decontamination station for decontamination.

B-28. Determining proper concentration of decontaminant solution can be a constant challenge to the Service members. To help reduce the amount of human calculation errors, an automated tool was created by the U.S. Air Force. The Automated Decontaminant Calculator is a user-friendly database that allows the Service member to make a predetermined percentage concentration of chlorine solution without dealing with complicated chemical formulas. This tool can determine the volume of water needed for a set amount of decontaminant and vice versa. In addition, the user will be able to choose from the most standard chlorine-based decontaminants, for example- calcium hypochlorite and sodium hypochlorite. A representative screen shot of the calculator is shown at figure B-1. To use the online automated decontaminant calculator, go to the ccc.apgea.army.mil link found in the reference section.

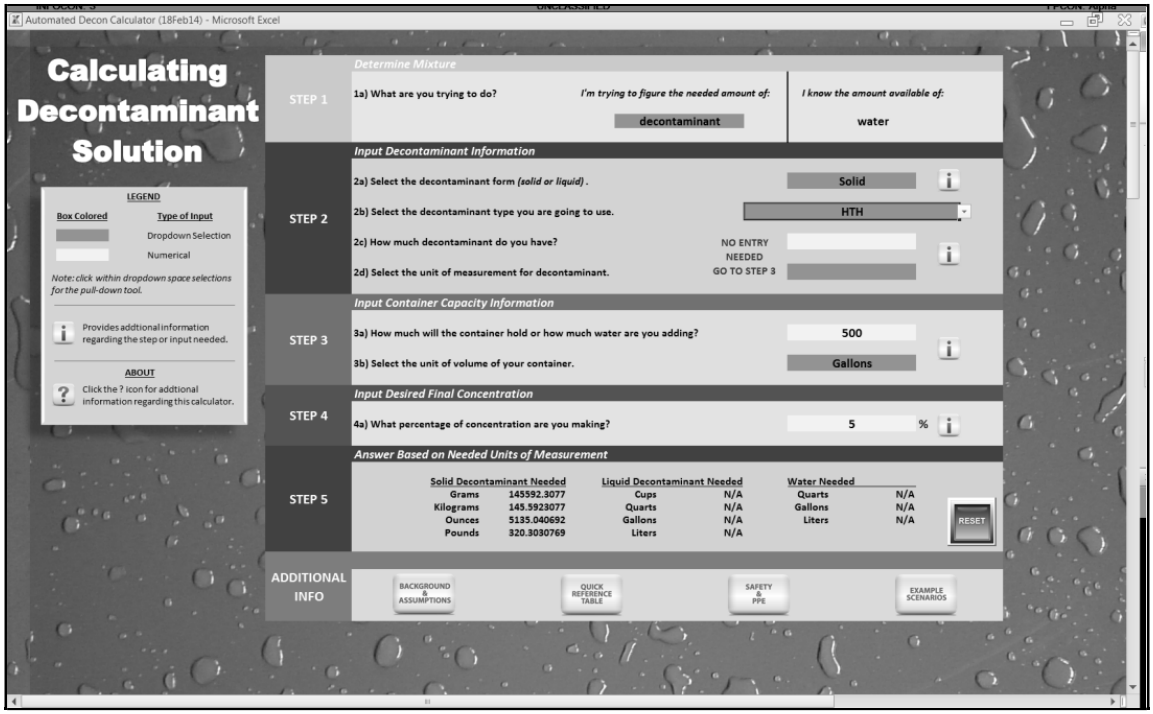


**Figure B-1. Automated decontaminant calculator**

## VERIFY COMPLETENESS OF DECONTAMINATION

B-29. Decontamination has to be monitored and verified to ensure that there are no residual hazards left.

### Monitor Decontaminated Equipment

B-30. Use a chemical agent monitor/detector to check each item prior to its being placed into the general supply area. If time allows, complete the following:

- 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 chemical agent monitor/detector into the opening, and observe for any indication of residual vapor hazard.
- If residual contamination is found, repeat the decontamination process. If it is still contaminated, remove from area then follow unit's standard operating procedure and commander's guidance for disposition of the equipment.

### Residual Hazards

B-31. 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.

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## Appendix C

# Medical Management and Treatment in Chemical Environment Operations

## MEDICAL MANAGEMENT

C-1. All MTFs must be prepared to receive mass casualties caused by exposure to CW agents. A mass casualty situation exists when the number and types of casualties exceed the local medical support capabilities for their care. For more information on medical support in a CBRN environment, refer to STANAG 2873. If the unit follows conventional operational standard operating procedures, 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.

C-2. The recommended minimal staffing for one cycle at a patient decontamination site is 30 to 39 personnel. At U.S. Army MTFs, Roles 1 and 2 (unit and at echelons above brigade), the supported unit commander must provide a minimum of eight nonmedical personnel to perform patient decontamination. At Role 3 combat support hospitals, a minimum of 20 nonmedical personnel must be provided to perform patient decontamination for one work cycle. The base cluster commander or units within the geographical area of the combat support 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.

C-3. At U.S. Air Force MTFs, casualty decontamination is performed by the U.S. Air Force Wartime Medical Decontamination Team.

C-4. At U.S. Navy MTF afloat, nonmedical personnel perform casualty decontamination procedures.

C-5. At MTFs supporting U.S. Marine Corps units, casualty decontamination is performed by personnel as designated by the commander.

### OBJECTIVES OF HEALTH SERVICE SUPPORT IN CHEMICAL ENVIRONMENT OPERATIONS

C-6. The objectives of HSS in chemical environment operations are to—

- Return to duty the maximum number of personnel as soon as possible.
- Protect persons handling contaminated casualties or persons working in contaminated areas.
- Avoid spreading contamination in ambulances, other evacuation vehicles, MTFs, and adjoining areas.
- Manage casualties so that CW agent injuries are minimized and any other injuries or illnesses are not aggravated.
- Provide postdeployment health assessments, aftercare, and continued treatment as indicated and directed by DOD and component service guidance.

### PLANNING FOR THE MANAGEMENT AND TREATMENT OF CHEMICALLY CONTAMINATED CASUALTIES

C-7. The initial management and treatment of casualties contaminated with a CW agent will vary with the tactical situation and the nature of the contaminant. Refer to STANAGs 2478 and 2553 for information on medical planning support and casualty estimation in a CBRN environment. 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, or in a leeward exposed topside position afloat (Navy). This ensures medical supervision of casualty decontamination is available.

C-8. Specifics on management of chemically contaminated casualties at the MTF are found in multi-Service tactics, techniques, and procedures for HSS in a CBRN environment. Although currently at the Department of the Army level for approval, the capability of the U.S. Army Medical Equipment Set Chemical Agent Patient Treatment will be combined with the medical equipment set Chemical Agent Patient Decontamination. This new assemblage will have the capability of treating and decontaminating 60 patients. The basis of issue is the same—

- One set per medical treatment team or squad at battalion level (battalion aid station).
- Three sets per medical treatment squad in the medical company (except medical treatment squad area).
- Three sets per combat support hospital.

### EMERGENCY MEDICAL TREATMENT OF CHEMICALLY CONTAMINATED CASUALTIES

C-9. Chemical warfare 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.

C-10. Triage of the arriving casualties is extremely important. A decision must be made whether emergency medical treatment 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-11. 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.

C-12. 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:

- Administer CW agent antidote, if available.
- Control respiratory failure (provide assisted ventilation) and/or massive hemorrhage.
- Decontaminate the casualty.
- Administer additional emergency medical treatment for shock, wounds, and life- or limb-threatening illnesses.
- Evacuate the casualty as soon as possible, if necessary.

## RESUSCITATION DEVICE, INDIVIDUAL CHEMICAL

C-13. To control respiratory failure in a contaminated environment, the *resuscitation device, individual chemical* is what is currently used. The purpose of the *resuscitation device, individual chemical* is to ventilate an apneic patient while in individual protective equipment/personal protective equipment. The *resuscitation device, individual chemical* (see figure C-1) should not be used in a contaminated environment without a filter attached to the inlet of the resuscitator. The *resuscitation device, individual chemical* is a ventilator system comprised of the following:

- Compressible butyl rubber bag.
- NATO-standard designated canister filter.

- Nonrebreathing valve.
- Cricothyroid cannula adapter.
- Flexible hose connected to an oropharyngeal mask.
- Other materials (extension tube, tube connectors, head harness, and face shield).

***Note.*** When supplemental oxygen is given through the filter adaptor, the protective black cap must be removed from the inlet nipple before oxygen supply tube is attached.

**Figure C-1. Resuscitation device, individual chemical**

# STERNAL INTRAOSSEOUS INFUSION SYSTEM

C-14. To mitigate the effects of often critical delay in treatment to the next role of care, sustaining treatment such as IV in a CBRN environment may be required. It will be hard to access the vascular system of the patient while in full individual protective equipment/personal protective equipment unless the patient is in a PPW where IV ports are available. Currently, the recommended device to use for fluid resuscitation while in full individual protective equipment/personal protective equipment is the sternal intraosseous infusion system. This is a specialized medical procedure (insertion site is the manubrium) performed by trained medical personnel. The sternal intraosseous infusion system includes (figure C-2 on page C-4):

- Target/strain relief patch.
- Introducer with infusion tube.
- Remover.
- Protector dome.
- Sharps protector cap.
- Foam filled sharps plug.
- Packaged syringe.
- Iodine prep pad.
- Alcohol prep pad.

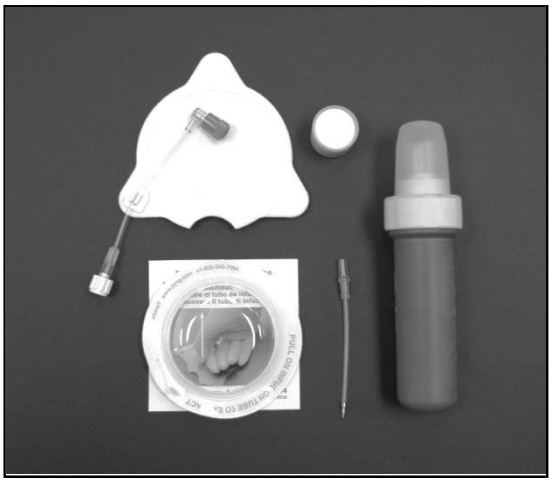

**Figure C-2. Sternal intraosseous infusion system**

C-15. The following are the steps to take when initiating the sternal intraosseous infusion system:

- Gather, inspect, and prepare flush and equipment.
- Take body substance isolation.
- Locate suprasternal notch landmark.
- Clean site with alcohol.
- Place target patch at landmark.
- Recheck the location of the target patch.
- Place bone needle cluster into the target zone of the target patch. Maintain perpendicular aspect of the introducer to the sternal surface.
- Apply increasing pressure along introducer axis until release is felt and heard.
- Gently remove the introducer by pulling straight back.
- Connect the infusion tube to the right angle connector on the target patch.
- Connect syringe and flush the infusion tube with 5 cubic centimeters of flush.
- Connect the IV infusion tubing, initiate flow of fluids.
- Attach the protective dome to target patch and secure with tape.

***Note***. The above procedures have to be completed within 3 minutes.

# CASUALTY DECONTAMINATION METHODS

C-16. Casualty decontamination serves two purposes—it prevents the casualty's system from absorbing additional contaminants and 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.

C-17. 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-18. 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.

C-19. Also, forward MTFs do not have replacement protective overgarments, protective masks, or filter canisters. Any ambulatory patient decontaminated during clothing removal becomes a litter patient; this individual 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.

C-20. Patients not requiring treatment at a forward MTF, but requiring evacuation to the next level MTF, must initiate immediate decontamination techniques on their MOPP gear and equipment and the integrity of their MOPP gear restored, such as taping over tears or rips. Immediate decontamination will remove gross contamination, reducing the hazard to the casualty and evacuation personnel.

C-21. Every person entering the decontamination area (including casualties) must wear their protective mask or have other respiratory protection in place. Most contaminants are removed by carefully removing all clothing. The following items are removed from the casualty- protective mask hood (the protective mask will be worn by the casualty at all times) overgarments, overboots, boots, uniform, and undergarments. For step-by-step procedures in performing casualty decontamination, refer to multi-Service tactics, techniques, and procedures for HSS in a CBRN environment. For more information on levels of decontamination (immediate, operational and thorough), refer to ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37.

C-22. After patients have been decontaminated, exercise rigid control to prevent exposing their unprotected skin to a vapor or liquid CW agent. 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.

C-23. 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, immediate decontamination must be performed on their overgarment to remove gross contamination. When possible, these personnel should proceed in groups of two or three to facilitate control. Ambulatory patients require constant observation and periodic assistance during the decontamination process. The combat medic/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 walks across the hot line, through a shuffle pit if established, 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 a Role 1 MTF; however, immediate decontamination techniques on the individual's MOPP gear must be performed to remove gross contamination before evacuation to the Role 2 or 3 MTF.

# MEDICAL LOGISTICS

C-24. Medical treatment requirements increase when operating in a chemically contaminated environment. Military Health System personnel reinforcement or replacement may be necessary. Plans for HSS following a CBRN incident must include efforts to conserve available Military Health System personnel and ensure their best use.

C-25. Provisions must be made to ensure that medical personnel are supplied and equipped to manage and treat contaminated casualties. The MTF should operate in a contaminated environment only until medical personnel have the time and means to move to a clean area. 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.

C-26. Patient protective wraps (see figure C-3, on page C-6) 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 ensembles (if available) or be placed in PPWs before

they are moved to the evacuation point if they are to be transported with dirty patients or through a contaminated area.

**Figure C-3. Patient protective wrap**

C-27. Contaminated environments may have a profound effect on medical evacuation. There are three basic modes of evacuating casualties— ground vehicles, watercraft, and aircraft. If operating forces are in a contaminated area, most or all of the medical evacuation assets will operate there. However, efforts should be made to keep some ambulances free of contamination.

# TRAINING

C-28. Commanders must ensure that medical personnel and decontamination team members (provided by the supported unit) are trained to manage, decontaminate, and treat CW agent casualties. Personnel must be trained to protect themselves from CW agent injuries. Refer to STANAG 2954 for more information on training of medical personnel for CBRN defense operations.

C-29. In addition, provisions must be made for training activities and practice exercises to enable them to accomplish their responsibilities with speed and accuracy. For example, decontaminating a casualty with speed is achieved through practice. Air Force medical personnel training for handling CBRN contaminated casualties is established in AFI 41-106. *First Receivers* training is required as a minimum. Training emphasis should be placed on the following subjects:

- Employing individual protection.
- Practicing immediate decontamination.
- Using CW agent detection paper and the improved chemical agent monitor 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 emergency medical treatment while in MOPP ensemble.
- Performing casualty decontamination.
- Lifting and transferring patient techniques.
- Evacuating decontaminated casualties.
- Evacuating contaminated casualties.

# CASUALTY EVACUATION

C-30. Immediate decontamination should be performed on contaminated casualties as close to the areas where they were contaminated as possible. Their MOPP gear and clothing should not be removed until they are at the patient decontamination site near an MTF. Upon arrival at the patient decontamination site, all contaminated clothing and equipment (except the protective mask) are removed and the skin and protective mask are decontaminated. After decontamination, 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 at all times. Patients will keep their protective masks on until they are in the clean treatment area (away from liquid and vapor contamination) or have entered the CPS through the airlock (see multi-Service tactics, techniques, and procedures for HSS in a CBRN environment).

C-31. Once treated, the patient is provided a new MOPP ensemble (if available) 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. The individual's mask is bagged after decontamination and stays with the patient. A plastic window in the PPW permits patient observation. A patient in a PPW that is left in a sunny area is subject to excessive heat build-up. Casualties in PPWs must be in a shaded area for maximum protection from heat injury.

C-32. 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.

C-33. A special consideration when evacuating patients is to determine the specific routes that will be used by dirty medical evacuation vehicles to get to the patient decontamination site at an MTF. The routes used by the dirty ground vehicles to cross between contaminated and clean areas are considered dirty routes and are not crossed by clean vehicles.

C-34. If immediate decontamination cannot be performed on contaminated casualties, they should be evacuated by ground ambulance where feasible. This will allow for easier decontamination of transport assets. Before contaminated casualties are evacuated by air ambulance or watercraft, immediate decontamination techniques should be conducted. The CW agent vapor from contaminated casualties may endanger the crew and other personnel, as ventilation is poor in aircraft compartments and other enclosed spaces. These crafts should be designated as dirty evacuation assets. 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.

C-35. Provide as much ventilation during transport as the weather and other conditions permit as follows:

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

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## Appendix D

# Immediate Decontamination Procedures

## PROCEDURES FOR DECONTAMINATING THE EYES

D-1. Any suspected CW agent contamination of eyes or face must be removed immediately. In most cases, Service members will not be able to identify the agent before decontamination. Service members should quickly obtain overhead shelter to protect them while they—

- Remove and open canteen.
- Take a deep breath and hold it.
- Lift mask away from face but not take the mask off.
- Flush (irrigate) eye or eyes immediately with copious amounts of water or irrigate the eyes with water (from a canteen or other container of uncontaminated water). To flush eye or eyes properly, Service member should—
  - Tilt 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 face to avoid spreading the contamination.
  - Not use fingers or gloved hand to hold the eyelids apart. Instead, open eyes as wide as possible and pour the water as indicated. Eyes must be irrigated despite the presence of toxic vapors in the atmosphere.
  - Hold breath and keep mouth closed to prevent contamination and absorption through the mucous membranes. Decontaminate the CW agent residue along the flush path on the face.

D-2. If the skin is contaminated while flushing eyes, then decontaminate the face.

## SKIN DECONTAMINATION

D-3. Immediate decontamination of the skin to remove suspected agent should be accomplished at the time of exposure, utilizing the currently fielded RSDL. The RSDL, a Joint Service Personnel Decontamination System replaced M291 decontamination kit. The RSDL is effective against cutaneous nerve and blister (vesicant) agents, such as mustard, sarin, and VX. It breaks down chemicals in seconds leaving a nontoxic liquid that can be removed with water. When exposed to CW agents, the Service member wipes the exposed skin with the lotion. The RSDL is safe for use on all intact skin surfaces.

D-4. The RSDL acts within seconds of being applied to the skin, neutralizing the toxicity of CW agents by breaking down their molecules. The lotion should be applied within 1 minute of contamination. The RSDL reacts rapidly, providing full removal and destruction of CW agents within two minutes, enabling efficient decontamination of casualties.

D-5. The RSDL is a bright yellow viscous liquid broad spectrum CW agent and vesicating toxin decontaminant that is spread onto skin that is exposed to CW agents or toxins. It is impregnated in a sponge pad packaged as a single unit in a heat-sealed, compact, easy to use tear-open foil pouch. The packet can be carried for use by Service members to protect themselves and aid victims of a chemical attack.

D-6. The RSDL was invented by the Canadian Defence Research Establishment. The RSDL is registered with the FDA and has been cleared for use by the U.S. military based on studies conducted by the U.S. Army (see figure D-1 on page D-2). It is available in three formats of which two are approved in the U.S. A training simulant is also available which allows realistic training and incorporation of human decontamination into training scenarios.


**Figure D-1. Reactive skin decontamination lotion**

# INDIVIDUAL EQUIPMENT DECONTAMINATION

D-7. The M295 Decontamination Kit, Individual Equipment (see figure D-2) is designed for use in decontamination of individual equipment. Individual equipment decontamination reduces the probability of cross contamination to the Service member and other personnel.

D-8. Use a stick or other object to remove any thickened spots of CW agent from the equipment.

D-9. Open the packet, remove the pad, and place fingers through the slot in the pad.

D-10. Rub all surface areas of the equipment with the pad.

**WARNING**

**The M295 is not approved for use on the skin by the FDA. Only use the M295 kit on equipment. Keep the decontaminating material out of the eyes; it may be slightly irritating to the skin and eyes.**


**Figure D-2. Decontamination kit, individual equipment**

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## Appendix E

# Pretreatment Regimen and Nerve Agent Antidotes Administration

## PRETREATMENT REGIMEN

E-1. The PB tablet blister pack (figure E-1, below and figure E-2 on page E-2) contains the pretreatment medication to be taken within 8 hours prior to exposure to organophosphate nerve agent. After exposure, atropine and 2-PAM Cl are 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).

E-2. 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 PB 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.

E-3. Orders to start taking PB will be issued by the proper line authority within the chain of command, under the advice of qualified medical personnel. It is not a medical decision.

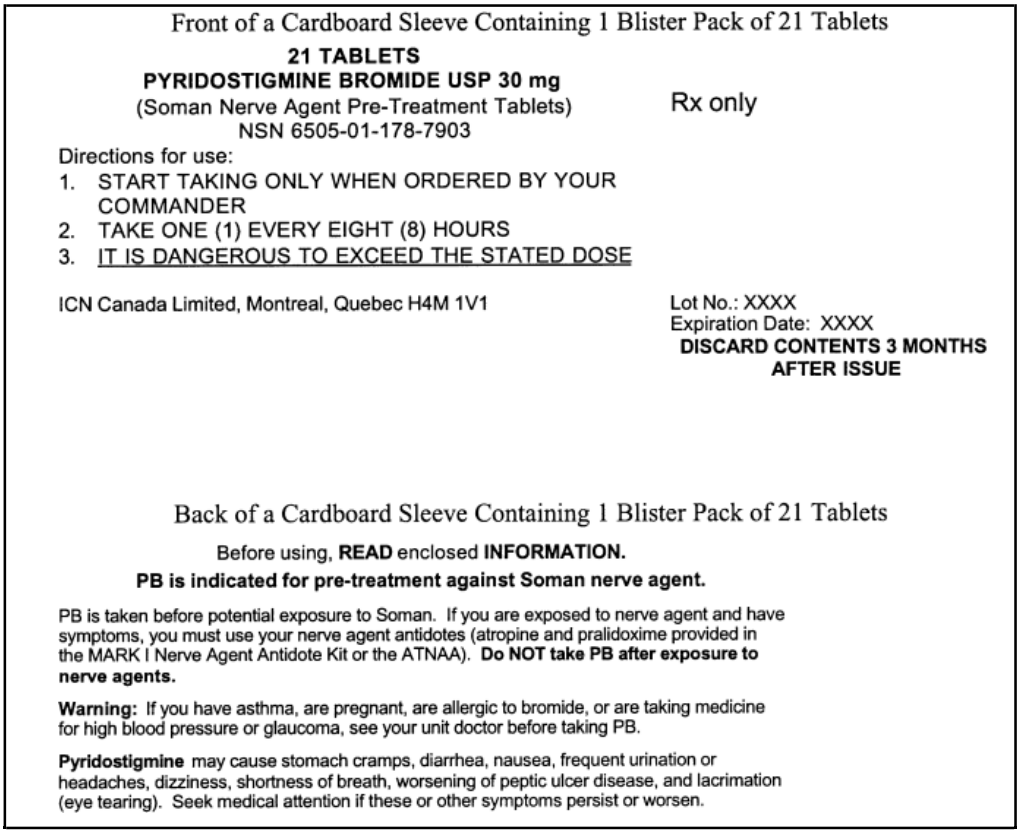


**Figure E-1. Pyridostigmine bromide tablet cardboard sleeve labels**

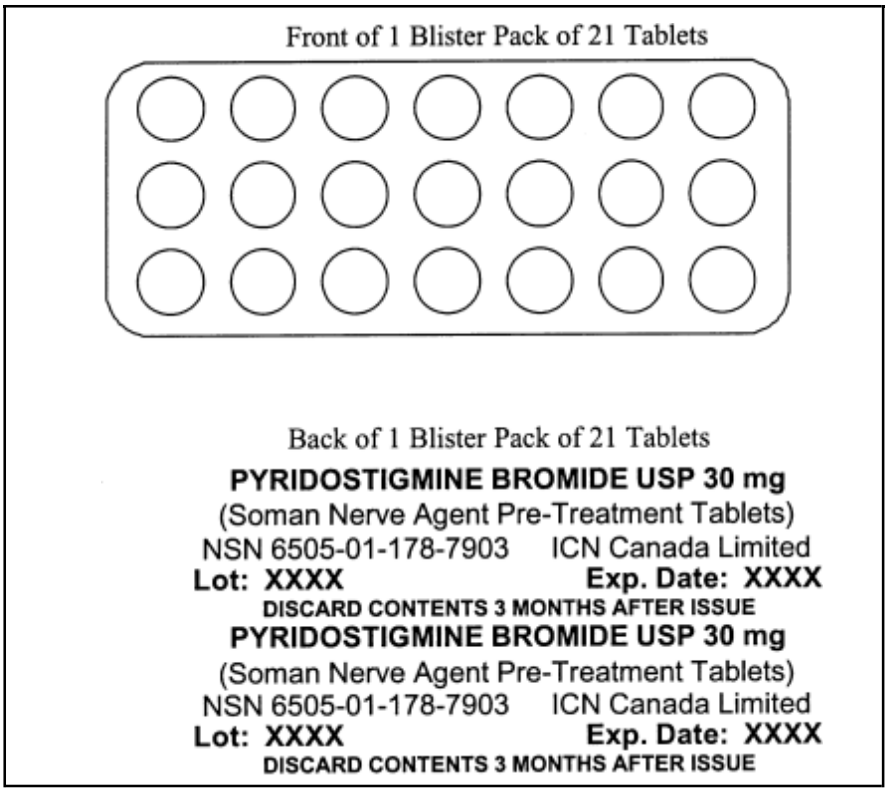


**Figure E-2. Pyridostigmine bromide blister pack front and back label**

## PRINCIPLES FOR THE USE OF PYRIDOSTIGMINE BROMIDE

E-4. To be very effective, one PB tablet should be taken every eight hours on a continuous and consistent basis prior to exposure to a nerve agent until all 21 tablets in the blister pack have been taken, or the individual has been directed to discontinue taking the medication. If PB 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 consistently every eight hours, the beneficial effect of PB as a pretreatment significantly diminishes after eight hours from the last tablet.

E-5. Individuals exposed to nerve agents require antidotes in addition to the PB received prior to exposure.

***Note.*** Do not attempt to give a PB tablet to a casualty with organophosphate nerve agent symptoms. If PB is taken immediately before exposure or at the same time as poisoning by nerve agent, it is not expected to be effective and may make the effects of a sublethal exposure to nerve agents worse.

E-6. At times, a commander may defer administration of PB on schedule. Examples of this would be when Service members—

- 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.
- 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. In any case, the benefits versus the risks should be carefully weighed before a decision is reached.

E-7. As long as the risk is elevated, it is desirable to continue the PB pretreatment. The PB pretreatment should continue regardless of individual protective equipment since the protective posture could be breached at any time. Command guidelines should be developed for situations such as—

- 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.
- Taking the tablets while in MOPP 4 could be hazardous (for example, 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-8. Pyridostigmine bromide should be used by pregnant Service members only if clearly needed.

## ADMINISTRATION OF PYRIDOSTIGMINE BROMIDE PRETREATMENT IN AN UNCONTAMINATED ENVIRONMENT

E-9. 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 the commander. If an individual missed a dose, the Service member should not make it up. The Service member should not take two tablets at once because of a missed dose—this individual should 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.

## SIGNS AND SYMPTOMS OF PYRIDOSTIGMINE BROMIDE OVERDOSE, ADVERSE REACTIONS, AND CONTRAINDICATIONS

E-10. Signs and symptoms of overdose, adverse reactions, or side effects are—

- Abdominal cramps.
- Nausea and vomiting.
- Diarrhea.
- Blurring of vision, miosis.
- Increased bronchial secretions.
- Cardiac arrhythmias, hypertension.
- Weakness, muscle cramps, and muscular twitching.
- Skin rash.

E-11. 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 PB.

## CONTRAINDICATIONS

E-12. Since PB may increase bronchial secretions and aggravate bronchiolar constriction, caution should be used in its administration to personnel with bronchial asthma.

E-13. Pyridostigmine bromide is contraindicated in mechanical intestinal or urinary obstructions.

E-14. Pyridostigmine bromide should not be administered to personnel with known hypersensitivity to anticholinesterase agents.

E-15. Additional relative contraindications include hyperthyroidism, sensitivity to bromide, peptic ulcer disease, and low serum acetylcholinesterase.

E-16. 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.

**WARNING**

**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.**

E-17. If any of the above signs/symptoms occur, the Service member should consult unit medical personnel as soon as possible.

# NERVE AGENT ANTIDOTES

E-18. The injection site for administering the ATNAA and CANA (see figure E-3) 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 (see figure E-4). 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 (see figure E-5). Injecting in the buttocks of thinly built individuals avoids injury to the thighbone.

***Note.*** The ATNAA replaced the MARK I.


**Figure E-3. Nerve agent antidotes (ATNAA and CANA)**

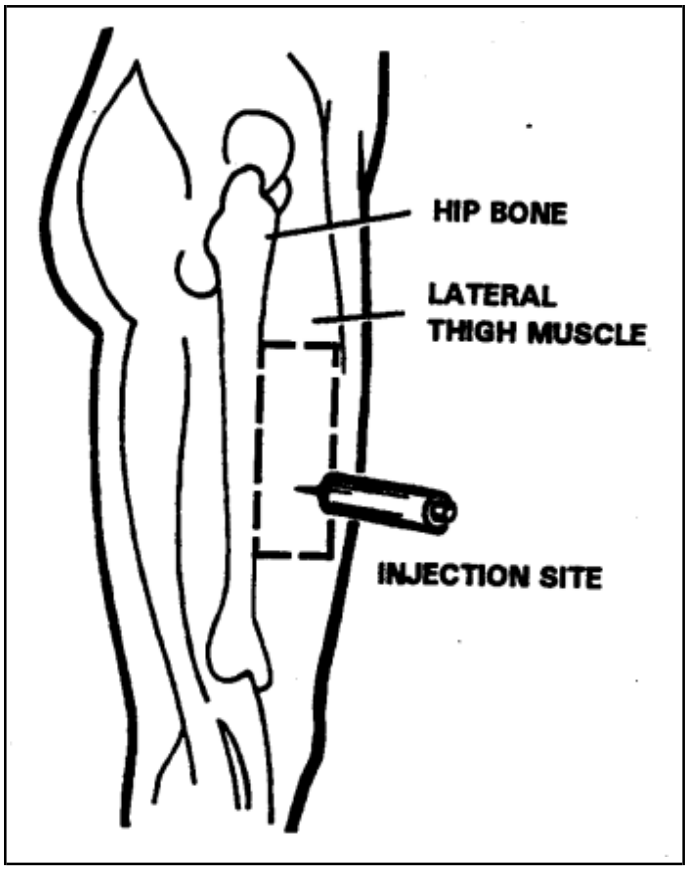


**Figure E-4. Thigh injection site**

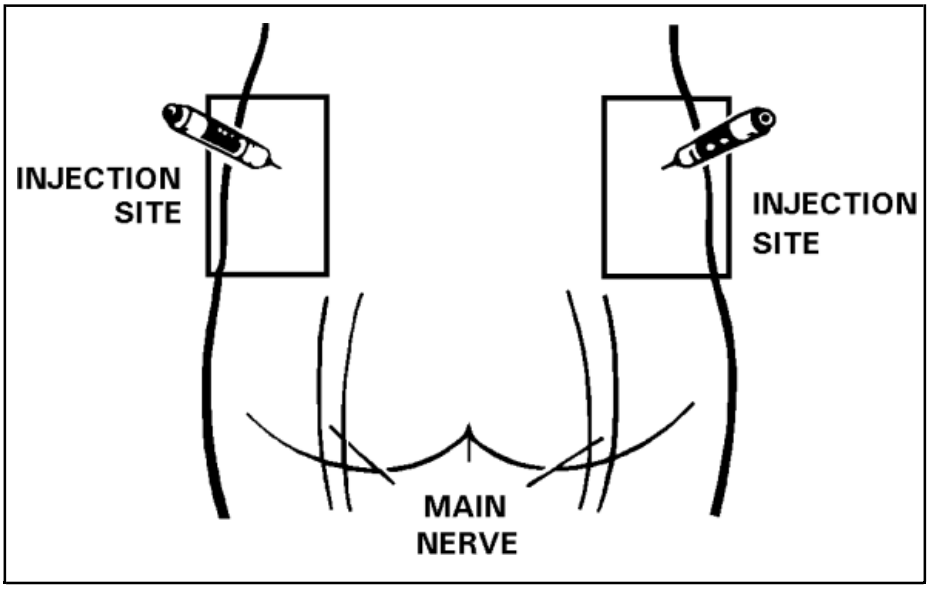


**Figure E-5. Buttocks injection site**

## SELF-AID ADMINISTRATION OF ANTIDOTE TREATMENT-NERVE AGENT AUTOINJECTOR

E-19. If an individual experiences any or all of the nerve agent mild exposure effects, the individual must immediately put on the assigned protective mask and self-administer one ATNAA (see figures E-7 and E-8). Follow the procedure given in table E-1.

**Table E-1. Self-aid for nerve agent poisoning**

| *Antidote Treatment Nerve Agent, Autoinjector* |
|---|
| STEP 1. Obtain one ATNAA*. |
| STEP 2. Check injection site. |
| STEP 3. Hold ATNAA with dominant hand (see figure E-6). |
| STEP 4. Grasp safety cap with nondominant hand and remove from injector. Drop the safety cap to the ground (see figure E-6). |
| STEP 5. Clear hard objects from injection site. |
| STEP 6. Inject ATNAA at injection site (holding the injector in the fist or holding it like a pen) applying even pressure to the injector (do not jab). Hold in place for 10 seconds (see figures E-7 or E-8). |
| STEP 7. Bend needle of used injector by pressing on a hard surface to form a hook. |
| STEP 8. Attach used injector to blouse pocket flap of JSLIST (see figure E-9). |
| STEP 9. Massage injection site, mission permitting. |
| *Only administer one ATNAA as self-aid. Do not self-administer CANA. |
| **Legend:**<br>ATNAA antidote treatment nerve agent, autoinjector<br>JSLIST Joint Service Lightweight Integrated Suit Technology |

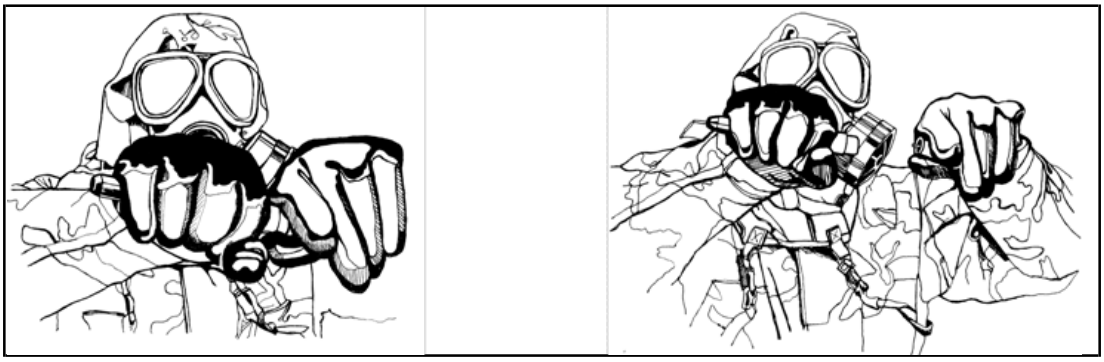

**Figure E-6. Preparing ATNAA or CANA for injection**

**Figure E-7. Self-aid thigh injection**

**Figure E-8. Self-aid buttocks injection**

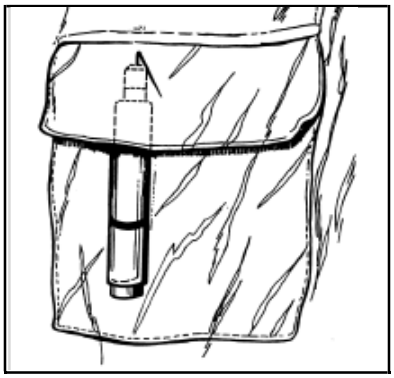

**Figure E-9. Used ATNAA attached to clothing**

E-20. The ATNAA is carried in the individual's protective mask carrier, pocket of the MOPP overgarment, or other location as specified in the unit tactical standard operating procedure. In cold weather, the ATNAA should be stored in an inside pocket of Service member's clothing to protect the antidote from freezing. A frozen ATNAA cannot be immediately used to provide the individual with antidote, when needed. The ATNAA can still be used after complete thawing.

***Note***. The ATNAA is packaged in a chemically hardened pouch. The ATNAA needs to be stored in the chemically hardened pouch and not removed until it is needed for use.

E-21. After administering the first injection, wait 10 to 15 minutes. After administering one ATNAA, the individual should decontaminate the skin (Appendix D), if necessary, and put on any remaining protective clothing.

E-22. If the heart beats very rapidly and the mouth becomes very dry, the individual received enough antidote to overcome the dangerous effects of the nerve agent. The individual should not self-administer another ATNAA. If a Service member is able to walk without assistance (ambulate), and is not confused or disoriented, then the Service member does not need the second ATNAA. If not needed, injecting a second ATNAA injection may create a nerve agent antidote overdose, which could cause incapacitation.

E-23. If the Service member continues to have symptoms of nerve agent poisoning, the Service member (if able) should seek someone else (a buddy) to check the his/her symptoms and administer the remaining antidotes, if required.

## BUDDY AID/COMBAT LIFESAVER AID

E-24. Service members may seek or require further assistance after self-aid (self-administering one ATNAA). A buddy or combat lifesaver 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 exposure effects of nerve agent poisoning; 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/combat lifesaver aid, determine if one ATNAA autoinjector has already been used. No more than three sets (total) of the antidote are to be administered. Buddy/combat lifesaver aid also includes administering the CANA with the third 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 CW agent into or through the protective clothing.**

**CAUTION**

Service members should not use their own issued ATNAA on a casualty. If they use their own, they may not have any antidote for self-aid.

**Table E-2. Buddy aid/combat lifesaver aid for nerve agent casualty**

| *ATNAA* | *CANA* |
|---|---|
| STEP 1. Mask the casualty and position casualty on his side (swimmer's position). | STEP 1. Obtain casualty's CANA. |
| STEP 2. Assume a proper position (squat, do not kneel) near the casualty's thigh (see figure E-10). | STEP 2. Check injection site. |
| STEP 3. Obtain casualty's three or remaining ATNAAs. | STEP 3. Hold CANA in a closed fist with dominant hand. |
| STEP 4. Check injection site. | STEP 4. Grasp safety cap with nondominant hand and remove from injector. Drop safety cap to the ground. |
| STEP 5. Hold ATNAA in a closed fist with dominant hand. | STEP 5. Clear hard objects from injection site. |
| STEP 6. Grasp safety cap with nondominant hand and remove from injector. Drop the cap to the ground. | STEP 6. Inject CANA at injection site (holding the injector in the fist or holding it like a pen) by applying even pressure to the injector (do not jab). Hold in place for 10 seconds. |
| 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 ATNAA at injection site (holding the injector in the fist or holding it like a pen) by applying even pressure to the injector (do not jab). Hold in place for 10 seconds (see figures E-10, below, and E-11 on page E-10). | STEP 8. Attach used injector to blouse pocket flap of JSLIST. |
| 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. Attach all used injectors to blouse pocket flap of JSLIST (see figure E-12 on page E-10). | |
| STEP 11. Massage injection site, mission permitting. | |
| **Legend:**<br>ATNAA antidote treatment nerve agent, autoinjector<br>CANA convulsant antidote for nerve agent<br>JSLIST Joint Service Lightweight Integrated Suit Technology | |

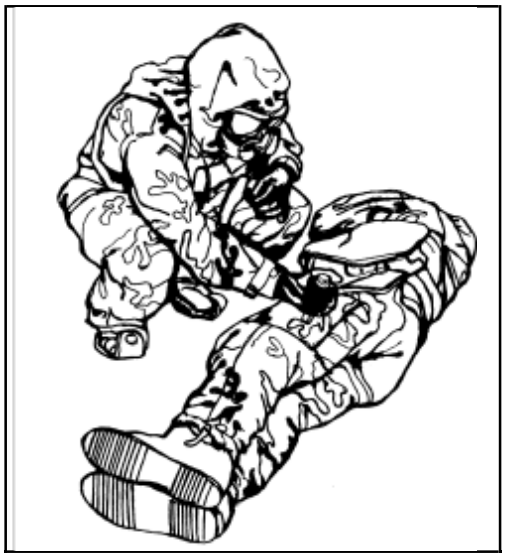

**Figure E-10. Injecting the casualty's thigh**

**Figure E-11. Injecting the casualty's buttocks**

***Note.*** Attach used autoinjectors to the casualty's protective overgarment by lifting the pocket flap and pushing the needles (one at a time) through the pocket flap fabric. Bend each needle to form a hook. Be careful not to tear the casualty's protective garments or Service member's gloves with the needles.

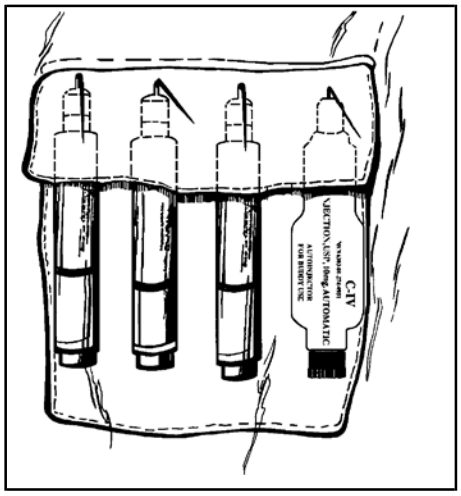


**Figure E-12. Three used ATNAA autoinjectors and one CANA autoinjector attached to clothing**

## Appendix F

# Chemical Warfare Agents and Toxic Industrial Chemical Immediate/Emergency Treatment Ready Reference

This appendix provides an immediate/emergency treatment ready reference for the treatment of casualties contaminated by CW agents and TICs (see table F-1).

**Table F-1. Emergency treatment ready reference**

| ***Choking (lung-damaging) agents*** | |
|---|---|
| AGENTS | Phosgene, diphosgene, chlorine, and chloropicrin. |
| SIGNS AND SYMPTOMS | Eye and airway irritation.<br>Dyspnea, coughing, choking, chest tightness, and respiratory distress.<br>Pathophysiology:<br>central agents: laryngospasm, loss of airway.<br>peripheral agents: noncardiogenic pulmonary edema. |
| DETECTION | Odor: newly mown hay or freshly cut grass or corn.<br>Sensors: Air monitoring system, chemical agent detector, chemical agent monitor, and standoff detector.<br>Other: Some monitors and detectors are not designed to identify phosgene. |
| PROTECTION | Military chemical protective mask. |
| DECONTAMINATION | Vapor: removal of victim to uncontaminated/fresh air.<br>Liquid: copious amounts of water or soap/water solutions/irrigation. |
| FIRST AID/BUDDY AID | Termination of exposure.<br>Enforced rest, warmth, and observation. |
| MEDICAL MANAGEMENT | Termination of exposure.<br>Basic life support: airway control, oxygenation, and ventilation, and circulatory support, as needed.<br>Enforced rest, warmth, and observation.<br>Supplemental oxygen with/without positive airway pressure.<br>More aggressive supportive therapy (pulmonary and airway management, antibiotic and/or steroid treatment), if required. |

**Table F-1. Emergency treatment ready reference (continued)**

| | |
|---|---|
| ***Blood (cyanide) agents*** | |
| AGENTS | Hydrogen cyanide and cyanogen chloride. |
| SIGNS AND SYMPTOMS | Low threshold between initial symptoms and severe physiological distress.<br>After exposure to high concentration: brief period of rapid breathing followed by convulsions, respiratory and cardiac arrest. |
| DETECTION | Odor: peach kernels or bitter almonds (absent in 50 percent), pink color of skin.<br>Sensors: Chemical agent detector kit.<br>Other: Chemical agent monitor and chemical detector paper/tape do not detect cyanide. |
| PROTECTION | Military chemical protective mask (vapor); mission-oriented protective posture 4 (liquid). |
| DECONTAMINATION | Usually unnecessary.<br>Remove wet, contaminated clothing and decontaminate underlying skin with water or soap/water solutions/irrigation. |
| FIRST AID/BUDDY AID | Mask others who are unable to don their mask.<br>Termination of exposure.<br>Fresh, uncontaminated air. |
| MEDICAL MANAGEMENT | Termination of exposure.<br>Basic life support: airway control, oxygenation, and assisted ventilation, and circulatory support, as needed.<br>Antidotes: amyl nitrite inhalation ampules if available, followed by intravenous sodium nitrite and sodium thiosulfate.<br>Supportive: administer oxygen, correct metabolic acidosis. |
| **Vesicants** | |
| AGENTS | Sulfur mustard, nitrogen mustard, Lewisites (Lewisite, phenyldichloroarsine, ethyldichloroarsine, methyldichloroarsine), and phosgene oxime. |
| SIGNS AND SYMPTOMS | Initial: asymptomatic (except Lewisite).<br>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.<br>Late: bone marrow suppression, generalized sepsis (sulfur mustard). |
| DETECTION | Odor: Garlic or tar (sulfur mustard), geraniums (Lewisite), others faint or no odor.<br>Sensors: Air monitoring system, chemical agent detector kit, chemical agent monitor, standoff detector, and chemical detector paper/tape. |
| PROTECTION | Mission-oriented protective posture 4.<br>Occupational Safety and Health Administration Levels A, B, or C depending on concentration. |
| DECONTAMINATION | Skin decontamination kit, copious water or soap/water solutions/irrigation. |
| FIRST AID/BUDDY AID | Termination of exposure/immediate decontamination.<br>Protect blisters and open wounds. |
| MEDICAL MANAGEMENT | Termination of exposure/immediate decontamination.<br>Basic life support: airway control, oxygenation, and ventilation, and circulatory support, as needed. Morphine may be needed to control pain.<br>Supportive care: correct fluid losses, protective bandages for bullae, open lesions. |

**Table F-1. Emergency treatment ready reference (continued)**

| Nerve agents | |
|---|---|
| AGENTS | Tabun, sarin, soman, cyclosarin, and O-ethyl methyl phosphonothiolate. |
| SIGNS AND 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.<br>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 | Chemical agent detector kit, chemical agent monitor, chemical agent paper/tape, and chemical agent alarm systems. |
| PROTECTION | Mission-oriented protective posture 4, and Levels A, B, and C.<br>Occupational Safety and Health Administration A or B depending on concentration. |
| PRETREATMENT | Pyridostigmine bromide. |
| DECONTAMINATION | Skin decontamination kit, copious water or soap/water solutions/irrigation. |
| FIRST AID/BUDDY AID | Termination of exposure/immediate decontamination.<br>Antidotes: atropine and 2-pralidoxime chloride by autoinjector; diazepam by autoinjector (self-aid—one Antidote Treatment-Nerve Agent Autoinjector; buddy aid or combat life support—up to three sets of Antidote Treatment-Nerve Agent Autoinjector plus one convulsant antidote for nerve agent). |
| MEDICAL MANAGEMENT | Termination of exposure/immediate decontamination.<br>Antidotes: atropine and 2-pralidoxime chloride; diazepam (severe exposure or seizures/convulsions).<br>Basic life support: airway control, oxygenation, and ventilation, and circulatory support, as needed.<br>Ventilation and suction of airways for respiratory distress.<br>Ocular symptoms: atropine sulfate ophthalmic ointment. |
| ***Incapacitating Agents*** | |
| AGENTS | 3-quinuclidinyl benzilate. Others include 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 | M50 chemical mask.<br>Air purifying respiratory mask. |
| DECONTAMINATION | Gentle, but thorough flushing of skin and hair with soap and water is required.<br>Reactive skin decontamination lotion can be used if washing is not possible.<br>Remove clothing. |
| FIRST AID/BUDDY AID | Termination of exposure. No immediate decontamination is usually necessary. Effects are self-limiting. |
| MEDICAL MANAGEMENT | Termination of exposure/immediate decontamination.<br>Antidote: physostigmine.<br>Supportive: monitoring of vital signs, especially core temperature. Ice should not be used for skin cooling. Use water or alcohol soaked cloth to cool patients.<br>Convulsant antidote for nerve agent/diazepam may be used to control seizures/convulsions. |

**Table F-1. Emergency treatment ready reference (continued)**

| | |
|---|---|
| ***Riot control agents (irritants)*** | |
| AGENTS | O-chlorobenzylidene malononitrile, chloroacetophenone in chloroform, bromobenzyl cyanide, dibenz(b,f)-1,4-oxazepine, and chloroacetophenone. |
| SIGNS AND SYMPTOMS | Burning and pain on exposed mucous membranes and skin, eye pain and tearing, burning in the nostrils, respiratory discomfort, coughing and dyspnea, and tingling of the exposed skin. |
| DETECTION | Chemical agent detector kit, chemical agent alarm systems, and chemical agent paper/tape. |
| PROTECTION | Military chemical protective mask with hood; field clothing. Individuals handling O-chlorobenzylidene malononitrile should wear rubber gloves, rubber boots, and rubber apron. |
| DECONTAMINATION | Eyes: thoroughly flush with water, saline, or similar substance.<br>Skin: flush with copious amounts of soap and water.<br>Bleach should not be used for decontamination because it produces irritating by-products from these agents,<br>Decontaminate O-chlorobenzylidene malononitrile-contaminated clothing by airing for a few minutes. |
| FIRST AID/BUDDY AID | Termination of exposure, no immediate decontamination.<br>Is usually necessary; effects are self-limiting. |
| MEDICAL MANAGEMENT | Termination of exposure/immediate decontamination.<br>Usually none is necessary; effects are self-limiting. |
| ***Vomiting agents*** | |
| AGENTS | Diphenylchloroarsine, diphenylaminechloroarsine (Adamsite), and diphenylcyanoarsine. |
| SIGNS & SYMPTOMS | Fullness in the nose and sinuses, severe headache, intense burning in the throat, and chest tightness; eye irritation and lacrimation; intense coughing, sneezing and rhinorrhea. Nausea and vomiting are prominent. With high doses there is prolonged period of malaise. |
| DETECTION | None available to field units. |
| PROTECTION | The protective mask provides adequate protection. No protective clothing is required (briefly lift mask from face to permit vomiting when needed). |
| DECONTAMINATION | Eyes: thoroughly flush with water, saline, or similar substance.<br>Skin: flush with copious amounts of soap and water.<br>Bleach should not be used for decontamination because it produces irritating by-products from these agents.<br>Decontaminate O-chlorobenzylidene malononitrile-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. |

**Table F-1. Emergency treatment ready reference (continued)**

| | |
|---|---|
| ***Toxic industrial chemicals*** | |
| AGENTS | Wide range of chemicals. Those most commonly encountered include ammonia, carbon monoxide, chlorine vapor, hydrogen sulfide, and oxides of nitrogen. |
| SIGNS AND SYMPTOMS | Signs and symptoms primarily due to chemical burns of eyes, airways, and skin. In enclosed spaces, secondary effects due to displacement of oxygen may be fatal within minutes. Carbon monoxide binds to hemoglobin and causes symptoms similar to cyanogens. |
| DETECTION | Many toxic industrial chemicals can only be detected by commercial, industrial chemical detectors, such as organic vapor analyzers (photoionization detectors and flame ionization detectors) and gas chromatographic analyzers. Typical chemical agent detectors fielded by the military services will not detect or identify many hazardous toxic industrial chemicals. |
| PROTECTION | Self-contained breathing apparatus. Military chemical protective masks in general provide no protection against toxic industrial chemicals; however, the joint service general-purpose mask will provide improved protection for selected toxic industrial chemicals. Occupational Safety and Health Administration 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.<br>Monitor and treat for shock.<br>Supportive/symptom-based treatment.<br>Agents with pulmonary effects may require supplemental oxygen, suctioning, and airway control. |
| ***Obscurants*** | |
| AGENTS | Hexachloroethane, grained aluminum, and zinc oxide containing mixtures, fog oil (smoke generator fog number 2), diesel fuel, sulfur-trioxide chlorosulfonic acid, titanium tetrachloride, red phosphorus, and white phosphorus. |
| SIGNS AND SYMPTOMS | Eye irritation, burning, lacrimation.<br>Dyspnea, coughing, stridor. |
| DETECTION | Not applicable. |
| PROTECTION | Military chemical protective mask, field clothing. |
| DECONTAMINATION | Eyes: saline or water.<br>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.<br>Bronchial constriction to zinc oxide obscurant can be treated with epinephrine hydrochloride, as required. |

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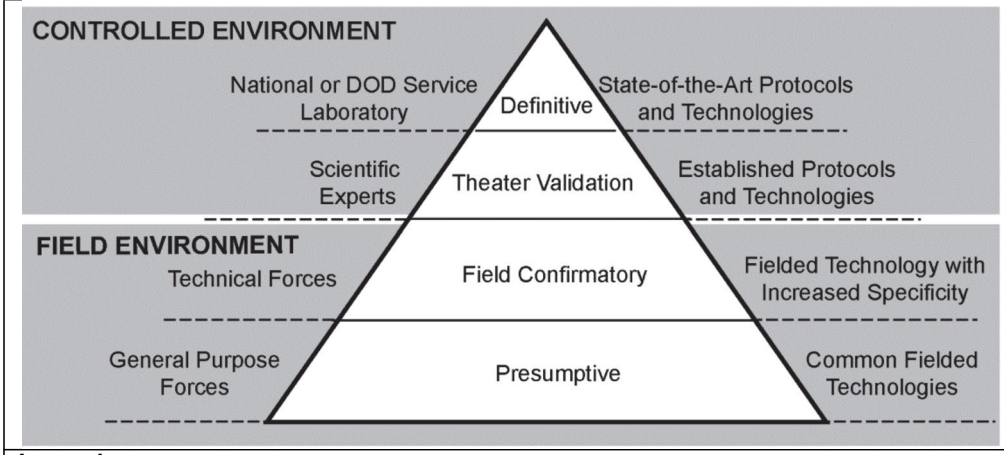
## Appendix G

# Levels of Identification

## FOUR LEVELS OF IDENTIFICATION

G-1. The four levels of CBRN hazard identification are—presumptive; field confirmatory; theater validation; and definitive. For more information on CBRN levels of identification, refer to ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44 and ATP 4-02.84/MCRP 4-11.1C/NTRP 4-02.23/AFMAN 44-156_IP. This appendix will only concentrate on the CW agents/target hazards levels of identification descriptors. See figure G-1 for overview of the four CBRN levels of identification.

***Note.*** Samples may not require analysis at all four levels of identification depending on the agent and the decisions and actions taken after identification.




**Legend:**
DOD Department of Defense

**Figure G-1. Overview of the four CBRN levels of identification**

***Note***. In figure G-1, *Technical Forces* are those specially trained and equipped forces that possess a higher degree of CBRN detection and sampling capability compared to conventional forces.

## PRESUMPTIVE IDENTIFICATION

G-2. *Presumptive identification* is the employment of technologies with limited specificity and sensitivity by general-purpose forces in a field environment to determine the presence of a chemical, biological, radiological, and/or nuclear hazard with a low level of confidence and the degree of certainty necessary to support immediate tactical decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

G-3. Presumptive identification is obtained using commonly fielded devices/materials/technologies available to general purpose forces to indicate/warn of the possible presence of a CW agent/target hazard. It provides important information to support warning decisions and actions, such as taking avoidance, protection, and decontamination measures. Table G-1 on page G-2 provides further presumptive identification descriptors.

**Table G-1. Presumptive identification descriptors**

| | |
|---|---|
| ***Who*** | General purpose forces. |
| ***Where*** | Field environment. |
| ***Capabilities*** | • Detector paper using chemical sensitive dyes.<br>• Photo-ionization detection.<br>• Ion mobility spectroscopy.<br>• Electrochemical sensors.<br>• Flame spectrophotometry.<br>• Surface acoustic wave.<br>• Reagents. |
| ***Why*** | Determine presence/absence of chemical warfare agents or hazards including toxic industrial chemicals to support immediate tactical decision such as avoidance, protection, or decontamination. |
| ***Example actions*** | • Assuming higher protection posture.<br>• Warning.<br>• Sampling.<br>• Reporting.<br>• Further assessments/exploitation. |

# FIELD CONFIRMATORY IDENTIFICATION

G-4. *Field confirmatory identification* is the employment of technologies with increased specificity and sensitivity by technical forces in a field environment to identify chemical, biological, radiological, and/or nuclear hazards with a moderate level of confidence and the degree of certainty necessary to support follow-on tactical decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

G-5. Field confirmatory identification is obtained using fielded devices/materials/technologies available to specially trained personnel and units in a field environment that includes collection and analyses of samples to substantiate the presence and type of a CW agent/target hazard at a given area/location. Field confirmatory identification can be used to prove (or disprove) previous presumptive results. It results in higher confidence levels to support tactical decisions regarding avoidance, protection, decontamination measures, and immediate treatment. Table G-2 provides further field confirmatory identification descriptors.

**Table G-2. Field confirmatory identification descriptors**

| ***Field confirmatory*** | |
|---|---|
| ***Who*** | Technical forces. |
| ***Where*** | Field environment. |
| ***Additional capabilities not available at lower levels of identification*** | • Fourier transform infrared spectroscopy.<br>• Gas chromatography/mass spectroscopy. |
| ***Why*** | To substantiate the presence and type of CW agents or hazards/toxic industrial chemicals at a given area/location to support follow-on tactical decisions such as avoidance, protection, or decontamination. |
| ***Example actions*** | • Reporting.<br>• Sample evacuation to theater validation laboratories.<br>• Further assessments/exploitation.<br>• Determining appropriate treatment and prophylaxis. |

# THEATER VALIDATION IDENTIFICATION

G-6. *Theater validation identification* is the employment of multiple independent, established protocols and technologies by scientific experts in the controlled environment of a fixed or mobile/transportable laboratory to characterize a chemical, biological, radiological, and/or nuclear hazard with a high level of confidence and the degree of certainty necessary to support operational-level decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

G-7. Theater validation identification is using accepted quality assurance measures, theater validation quantifies the CW agent/target hazard. It provides additional critical information to support timely and effective decisions regarding avoidance, protection, and decontamination measures, and medical prophylaxis and treatment for affected units and personnel. It can also support preliminary attribution to implicate or support trace analytics for the source of the identified CW agent/target hazard. Table G-3 provides further theater validation identification descriptors.

**Table G-3. Theater validation identification descriptors**

| | ***Theater validation*** |
|---|---|
| ***Who*** | Scientific experts applying multiple independent, established protocols and technologies. |
| ***Where*** | Fixed or mobile laboratory with constant temperature and humidity controls; stable power supply. |
| ***Additional capabilities not available at lower levels of identification*** | • Gas chromatograph-flame ionization detector.<br>• Gas chromatograph-electron capture detector.<br>• Gas chromatograph-mass spectrometer. |
| ***Why*** | To support timely and effective operational-level decisions including medical prophylaxis and treatment for affected units and personnel; and other avoidance, protection, and decontamination measures. |
| ***Example actions*** | • Reporting.<br>• Sample evacuation to definitive laboratories.<br>• Further technical assessments/exploitation. |

# DEFINITIVE IDENTIFICATION

G-8. *Definitive Identification* is the employment of multiple state-of-the-art, independent, established protocols and technologies by scientific experts in a nationally recognized laboratory to determine the unambiguous identity of a chemical, biological, radiological, and/or nuclear hazard with the highest level of confidence and degree of certainty necessary to support strategic-level decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

G-9. Definitive identification supports attribution to implicate or point to the source of the identified material. It uses the highest level quality assurance measures. Table G-4 on page G-4 provides further definitive identification descriptors.

**Table G-4. Definitive identification descriptors**

| *Definitive* | |
|---|---|
| ***Who*** | Scientific experts using multiple independent, state-of-the-art established protocols and technologies. |
| ***Where*** | National or Department of Defense Service laboratories. |
| ***Additional capabilities not available at lower levels of identification*** | Technologies are similar to theater validation level, but with higher controls at fixed accredited facilities. |
| ***Why*** | To support strategic-level decisions; to support attribution; to implicate or point to the source of the identified material. |
| ***Example actions*** | Reporting.<br>Further technical assessments/exploitation. |

## Appendix H

# Treatment of Military Working Dogs Exposed to a Chemical Environment

## CHEMICAL AGENT PROTECTION

H-1. Chemical protective doctrine for animals is incomplete, and there is no chemical protective equipment in the current inventory for MWDs. Equipment and doctrine for animals are under development but pending its availability, any degree of protection of the MWD in a CW agent environment will, at best, be extremely difficult. The information given herein applies particularly to the MWD, although these principles can be applied to other animals.

### PROTECTION OF MILITARY WORKING DOG RATIONS AND EQUIPMENT

H-2. Bagged MWD food and MWD equipment such as leather leashes, collars, and leather or plastic muzzles are subject to contamination and may be difficult to decontaminate or replace in a timely manner. One set of MWD handling equipment and a short-term supply, 1 to 4 weeks, of food should be stored in an impervious and easily decontaminated container for each MWD. Tightly sealed plastic cans (National Stock Number 7240-01-094-4305) may be used or these items may be stored in a nearby chemical protective shelter or protected vehicle.

### PROTECTIVE SHELTER FOR THE INDIVIDUAL MILITARY WORKING DOG AT THE DUTY SITE

H-3. In the absence of MWD protective garments or shelters, it will be difficult to protect a MWD if it cannot be placed in a field expedient protective shelter or in an available CPS. If chemical attack (to include toxic industrial materials) is likely, the only reliable method of MWD protection is movement from the area.

H-4. For immediate field expedient protection, the MWD can be covered with wet weather gear, tarp or similar impervious materials. This will provide some protection while the MWD is evacuated from the area.

H-5. If the MWD must remain on-site to perform necessary duty, limited protection may be provided by—

- Moving the MWD into an existing structure or vehicle that has been sealed with tape, tarps, or tentage to prevent inflow of contaminated air.

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***Note***. The risk of heat injury for an MWD in a sealed vehicle may be higher than the risk of chemical injury during a potential attack.

---

- Placing the MWD in its transport kennel and covering the transport kennel with tarps, tent, or plastic sheets to limit contamination by droplet or liquid agent.
- Placing the MWD in a chemical protective shelter with the handler and other personnel when space is available. This is the preferred method when possible.
- Placing chemical impervious barriers on the MWD's paws, after the paws have been decontaminated and thoroughly dried, if the dog must walk through a contaminated area. Ideally, the MWD should not be walked through any area with ground contamination but this may be necessary in some circumstances. If this occurs, the following items may provide limited protection if placed over the feet and taped at the carpus or tarsus:
  - Specimen bags (polyethylene terephthalate).
  - Outer bag from field rations meal.
  - Extra butyl-rubber protective gloves from Joint Service Lightweight Integrated Suit technology gloves.

***Note.*** None of these items are of a design to be walked on, so the ground contact surface may need to be protected with a more durable material such as tape or canvas over wrap.

- In the absence of specific MWD kennel facility CPS, the principles of field expedient protection covered in ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37 should be followed. If available, MWDs should be housed in chemical biological protective shelter or Joint Expeditionary Collective Protection shelters.

### PRETREATMENT OF MILITARY WORKING DOGS TO LIMIT CHEMICAL AGENT ABSORPTION AND TOXICITY

H-6. There is no specific preexposure therapy that has been evaluated in MWDs; however, some of the protective measures for military personnel may be implemented.

### PROPHYLACTIC MEDICATION

H-7. The effectiveness of the PB tablet is not well-documented in dogs and the effect of this medication on the performance of MWDs has not been evaluated. The DOD MWD Veterinary Service does not recommend the use of PB in MWDs because its effect on MWD detection performance has not been evaluated; however, the use of PB in the MWD may be authorized by the responsible veterinarian and MWD unit commanders.

- If PB is used, the handler must evaluate the ability of the MWD to perform in assigned tasks prior to performance of assigned duties. Treated MWDs should be identified as under the influence of the PB prior to entry into a contaminated environment and other protective measures should be taken when possible. When used, the recommended PB regimen is ½ tablet (15 mg) every 8 to 12 hours. All precautions regarding PB utilization as discussed in Chapter 3 should be followed in MWDs.
- If the MWD on PB is unable to perform its mission due to adverse effects of the medication, the dose and frequency should be reduced. If the performance decrement continues on the reduced dose, the MWD must be removed from duty and from the high risk area, or the PB treatments must be discontinued.
- Adverse effects of PB may mimic organophosphate toxicity including salivation, nausea, vomiting, abdominal pain, diarrhea, miosis and lacrimation, increased respiratory secretions, weakness, muscle twitching, and respiratory distress. If any of these are seen, the dose and frequency of PB must be decreased or the PB must be discontinued.

H-8. Initial issue guidelines for MWD medical, CBRN defense materiel can be found in the Department of the Army Supply Bulletin (SB), 8-75-S7, Army Medical Department Supply Information (page 5-3, table 5-2, National Stock Number 7610-01-564-2341, MWD Handler's Guide to Medical, CBRN Defense Materiel).

## NERVE AGENTS EFFECTS ON MILITARY WORKING DOG AND FOOD AND WATER

H-9. Nerve agents dispersed either by aerosol, vapor, or spray can be absorbed through a dog's respiratory tract, eyes, mouth, gastrointestinal tract, and skin. Currently, there is no means of protecting a MWD's respiratory tract. Respiratory absorption may occur after dispersal of aerosol, vapor or liquid agents and is of greatest concern because of the speed of absorption and toxicity. Absorption of nerve agent through the mouth may occur simultaneously with respiratory exposure. However, oral and gastrointestinal absorption is of greater concern when a dog ingests nerve agent by eating contaminated food, drinking contaminated water, or licking its own hair or paws that are contaminated with a nerve agent. Because of the combination of hair covering and lack of sweat glands, the risk of nerve agent absorption through the skin is of less concern in dogs than in people; however, the risk is still significant. Absorption through the skin via the MWD's paws is of the greatest concern since pads of the MWD's paws have sweat glands, no hair, and will absorb nerve agents.

H-10. Liquid nerve agents or vapors of nerve agents can poison food and water. Military working dogs should not drink from waterholes or trenches in contaminated areas or drink surface water that has run off from contaminated areas. Water suspected of being contaminated should be tested by preventive medicine/public health personnel and only water found to be safe should be used for consumption. Contaminated food or food that is suspected of being contaminated should not be fed to MWDs unless approved by veterinary personnel.

H-11. Food and water packaged in sealed, airtight cans, bottles, or other impermeable containers can be decontaminated according to procedures in ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37.

## SIGNS OF NERVE AGENT INTOXICATION IN MILITARY WORKING DOGS

H-12. All nerve agents generally produce similar effects, although the onset and severity of signs may vary depending upon the route and degree of exposure.

### Local Ocular And Respiratory Effects

H-13. Exposure to nerve agent vapors produces local ocular and respiratory effects before other effects. These signs usually appear within five minutes after exposure.

H-14. The initial ocular effect is miosis. More severe exposures may cause eye pain and visual impairment.

H-15. Respiratory exposure is manifested by a rapid, heavy panting (respiration), and an increase in upper respiratory secretions resulting in watery nasal discharge. Increased upper respiratory secretions with bronchoconstriction will cause coughing, rattling sounds in the throat, wheezing, and respiratory distress.

### Systemic Effects

H-16. Systemic absorption of enough nerve agent through the respiratory or gastrointestinal system will increase the severity of local effects and will also cause generalized systemic effects. Respiratory distress becomes marked due to profuse bronchial secretions, bronchoconstriction, and airway obstruction. The distressed animal will gasp and the mucous membranes of the mouth will become blue (cyanotic) as a result of decreased oxygenation. Other effects which may occur are slowing of the heart rate, profuse salivation and frothing, loss of fecal and urinary control, defecation and/or vomiting, and abdominal pain. Muscular effects occur with other systemic effects and the animal will exhibit muscular weakness, twitching muscles, and trembling. As weakness and paralysis of the respiratory muscles progress, breathing becomes increasingly labored, shallow, rapid, and finally intermittent, with the animal quickly becoming oxygen deficient. In severe exposures, the onset and progression of signs are very rapid. The animal may tremble violently, become uncoordinated, collapse, and go into generalized convulsive seizures. Loss of consciousness may ensue with a total loss of reflexes. Convulsions may become intermittent with the animal showing a rapid panting respiration between convulsive episodes. Marked generalized convulsions are usually followed by complete flaccid paralysis, central respiratory and circulatory depression, asphyxiation, and death.

### Local Skin Effects

H-17. The symptoms of cutaneous exposure to liquid nerve agents are similar to respiratory exposure to nerve agent vapors. One difference is that the initial signs take longer to develop and the transition from mild to severe symptoms may be slower. With fatal cases, the survival period may be hours, whereas in inhalation poisoning, most deaths occur in a few minutes. Cutaneous exposure causes local twitching at the site of contamination, increased gastrointestinal activity, salivation, miosis, generalized tremors, prostration, and convulsions. Dyspnea is not a pronounced symptom of early cutaneous poisoning, which differs from the inhalation route. Hypopnea occurs during the prolonged convulsive phase. A lethal factor in cutaneous poisoning is the very rapid rise in body temperature to heatstroke levels caused by the prolonged convulsions. For more information on signs and symptoms, refer to Chapter 3.

## DECONTAMINATION

H-18. Following contamination of the hair coat, skin, paws, or eyes, the animal should be decontaminated as quickly as possible to prevent or reduce any further absorption of the agent.

**CAUTION**

All persons who handle animals contaminated with nerve agents must be in MOPP 4 or proper civilian personal protective equipment as determined by Incident Commander.

### Hair and Skin

H-19. Since the hair coat delays penetration of liquid agents to the skin and cutaneous absorption requires several minutes, effective decontamination of the hair and skin may be carried out before any significant absorption has occurred. Decontamination is not a substitute for treatment. When the animal shows signs of exposure to a nerve agent, specific therapy should be initiated.

H-20. The entire animal (except eyes and periocular area) may be decontaminated by using RSDL and/or with soap and water.

***Note.*** The initial Medical CBRN Defense Materiel issue of RSDL for MWDs is one pouch (contains three packets). The MWD handlers should carry several extra RSDL for decontamination of the MWD and an extra M295 Individual Equipment Decontamination Kit for decontamination of MWD equipment.

H-21. Initial MWD decontamination with RSDL should be completed as soon as possible after nerve agent exposure. The entire MWD should be wiped down using RSDL, except for eyes and the area around the eyes, which should be rinsed with water. For other contaminates, soap and warm water can be used for MWD decontamination. The eyes should be flushed with large amounts of water, ophthalmic solution, or saline. If soap is not available, rinsing with large amounts of water is the next best method of decontamination. Allow the MWD to shake off excess water and dry with clean towel or other absorbent cloth.

H-22. Definitive decontamination of the MWD should be completed by thoroughly washing the hair coat and the skin with soap (Castile Soap Liquid [National Stock Number 8520-01-519-0776] or available nonmedicated veterinary shampoo) and water. It is important that all body surface areas are saturated with the soap and water and gently scrubbed and washed. After the washing is completed, the hair coat and skin should be rinsed and the soap residue removed from the dog. If soap is not available, rinsing with large amounts of water is the next best method of decontamination. The preferred method of decontaminating the MWD is by first using RSDL then thoroughly washing and rinsing the MWD to ensure all contaminants are removed.

### Eyes

H-23. Any amount of agent getting into the eyes of an animal requires prompt action to prevent conjunctival absorption, which can occur very rapidly. Personnel should prevent RSDL or soap from getting into the MWD's eyes as it could cause further injury to the eyes. The eyes should be decontaminated by irrigation with copious amounts of water, saline, or ophthalmic solution until all contaminates have been removed. After decontamination is complete, the eyes should be thoroughly evaluated and treated with appropriate ointments.

***Note.*** Eye ointments must not be placed in the eyes prior to completion of decontamination process and thorough eye examination after decontamination as it may absorb and concentrate agents and cause additional eye damage and toxicity.

**CAUTION**

Personnel performing the decontamination of the MWD must be careful and prevent any of the RSDL and soap residue from getting into the MWD's eyes. The decontamination solution could cause injury to the eyes and should not be used on or around the eyes. Ocular contamination should be removed with copious water irrigation of the eyes.

### MILITARY WORKING DOG DECONTAMINATION PROCEDURES

H-24. Decontamination procedures are as follows:

- Rinse the MWD thoroughly with plain water beginning at the head along the back and to the tail; then rinse down the MWD's sides, chest, stomach, legs, and paws.
- Work the soap into the hair starting the head, along the back and to the tip of the tail, then work down the MWD's sides, chest, and abdomen, legs, and paws. Ensure the soap reaches the MWD's skin. If the MWD has erect ears, flush the ears with otic solution or water.

***Note.*** Special attention should be paid to the MWD's stomach, face, ears, eyes, under tail, paws and in between legs to ensure all contamination is removed.

- Flushed the eyes with copious amounts of water, ophthalmic solution, or saline.
- Rinse with plain water using the same pattern as the initial rinse (head to back to tail, then down sides, chest, stomach, legs, and paws).
- Allow the MWD to shake off excess water. A tarp or other impervious materiel may be placed around the MWD while it shakes off excess water to prevent contaminating of other people, MWDs, or equipment.

***Note.*** Steps 1 to 5 may need to be repeated until all contaminants are removed.

### MILITARY WORKING DOG EQUIPMENT DECONTAMINATION

H-25. The leash, collar, and muzzle should be removed from the MWD and decontaminated as soon as possible. They may be decontaminated using the M295 Individual Equipment Decontamination Kit wipe-down mitts or by using a 5 percent hypochlorite solution or with 5 percent sodium carbonate solution (G-agents only). Additional guidance for decontamination of equipment is contained in ATP 3-11.32/MCWP 3-37.2/NTTP 3-11.37.

## TREATMENT OF MILITARY WORKING DOG CASUALTIES OF NERVE AGENTS

H-26. Treatment of MWD nerve agent casualties is discussed below.

### EMERGENCY THERAPY PROCEDURES

H-27. Initial first aid provided by the MWD handler depends on the severity of the poisoning and the type of nerve agent antidote kit that is issued to the MWD handler.

***Note***. The initial Medical CBRN Defense Materiel MWD basis of issue for nerve agent treatment is three ATNAA, five additional atropine autoinjectors and four CANA. Keep the autoinjectors used for the MWD with the MWD during evacuation.

H-28. For mildly exposed MWDs, administer a total of two ATNAA injections (atropine and 2-PAM Cl in a single autoinjector) (carried by the MWD handler) into the back of the thigh of the dog. The initial dosage of atropine is 4 mg and the dosage for 2-PAM Cl is 1200 mg.

H-29. For severely exposed MWDs, administer three ATNAA and one CANA. This is similar to the buddy aid a Service member provides another Service member suffering from severe nerve agent exposure.

**CAUTION**

In general, MWDs should not need additional 2-PAM Cl injections.

## FOLLOW UP HANDLER FIRST AID FOR SEVERE NERVE AGENT EXPOSURE

H-30. Single atropine injections may be given every 10 to 20 minutes until the nerve agent effects have subsided or signs of atropinization appear. This is equivalent to combat lifesaver aid or enhanced first aid for Service members with severe nerve agent exposure. The MWD must be monitored for heat stress. Atropine dries the mucous membranes thus preventing the MWD from expelling body heat.

H-31. The initial dosage of 2-PAM Cl in the dog is 20 mg/kilogram. Three ATNAA injectors should provide sufficient amount of 2-PAM Cl.

H-32. If a MWD is still showing signs of seizure after initial treatment, the handler may give up to 3 additional CANA autoinjections at 5 to 10 minute intervals until the seizures are gone.

H-33. Maintain a clear airway by removing respiratory secretions and saliva obstructing the airway. Loosen or remove the muzzle.

**CAUTION**

When clearing the MWD airway, the handler and veterinary personnel must use great care to avoid being bitten. Even a minor MWD bite could compromise personnel's MOPP status resulting in human nerve agent exposure.

H-34. In severe nerve agent exposure, the animal's respiration is markedly depressed and extreme muscular weakness or paralysis is present. In such cases, assisted ventilation is required to effectively resuscitate the animal.

H-35. Adequate atropine and 2-PAM Cl should bring about an improvement or restoration of spontaneous respiration and also improve blood circulation. However, the effectiveness of 2-PAM Cl is lost after a short period of time. The 2-PAM Cl varies in its effectiveness against nerve agents. It is least effective against GD nerve agent. In some cases, severe nerve agent symptoms may persist or recur and require veterinary personnel to administer additional 2-PAM Cl autoinjectors every 8 to 12 hours for up to 3 days.

H-36. Signs of effective atropinization include dry mouth and mucous membranes, increased heart rate, and increased body temperature. Signs of excessive atropinization and atropine toxicity may include vomiting, thirst, difficulty eating, constipation, difficulty urinating, altered mental status which may be either depression or excessive stimulation, ataxia, seizures, decreased breathing rate, increased heart rate with possible arrythmias, and abnormal blood pressure (decreased with shock and circulatory collapse or increased). Atropine administered systemically may not overcome local ocular effects so that the absence of pupillary dilation does not necessarily indicate the need for further atropine administration. Canine nerve agent casualties can tolerate much greater doses of atropine than would a normal dog that has not been exposed to a nerve agent. However, repeated doses of atropine will markedly increase its effects, especially in animals that have received only a minimal exposure.

H-37. Supportive therapy procedures are as follows:

- Maintain a clear, unobstructed airway. Assisted ventilation may be required.
- Complete decontamination if not already performed.
- Provide supportive treatment, as indicated.

***Note***. Atropine is usually sufficient to control CNS signs, but if convulsions persist or occur intermittently and further interfere with respiration, they may be controlled by the administration of CANA intramuscularly.

# PROTECTION AGAINST INCAPACITATING AGENTS (3-QUINUCLIDINYL BENZILATE TYPE, FENTANYL DERIVATIVES)

H-38. Significant absorption of BZ, an incapacitating agent, is most likely to occur through the animal's respiratory tract, but effective percutaneous and gastrointestinal absorption can occur. The protective measures for nerve agent exposure can be applied to incapacitating agents.

## SIGNS OF EXPOSURE

H-39. The incapacitating agent BZ is an anticholinergic agent with pharmacological effects similar to those of atropine, although it has a greater effect on the CNS than atropine. The onset of signs following a moderate respiratory exposure can be expected to occur within 10 to 20 minutes. In general, the greater the dose, the shorter the time for the onset of symptoms.

H-40. In the MWD, early effects of moderate exposures to BZ include increased heart rate, pupillary dilation, impaired vision, dry mouth, and a decrease in physical endurance while working. Marked rises in body temperature do not usually occur. The agent's predominant effects are on the CNS, resulting in incoordination, behavioral changes, confusion, and a lack of normal responses to commands. These exposures can be expected to incapacitate animals and make them unfit for service.

H-41. There is a large margin of safety between incapacitating and lethal exposures to BZ. Overwhelming exposures, however, can result in prostration and convulsions, with death occurring rapidly. Moderate exposures may cause altered mental status, failure of the MWD to follow commands, and spontaneous aggressive behavior.

H-42. Fentanyl derivatives can cause decreased respirations or breathing to stop, lethargy, listlessness, sleepiness, confusion, and unconsciousness.

## DECONTAMINATION

H-43. Decontamination for BZ type agents is large amounts of water warm and soap. Decontamination for fentanyl derivatives is not required as they evaporate quickly.

## TREATMENT

H-44. After a MWD has had a moderate exposure to BZ, effects may persist 24 hours or more. Although the MWD's life is not immediately threatened, therapy can be administered to hasten recovery and return the animal to duty as quickly as possible. However, the MWD should be examined and its work performance evaluated before it is returned to duty.

***Note.*** Do not use anesthetics, tranquilizers, and sedatives in the treatment of MWDs exposed to BZ as they tend to potentiate the effects of incapacitating agents.

H-45. General therapy for BZ exposure should include decontaminating the hair and the skin with warm soapy water, restricting activity, and provide clean drinking water.

H-46. Physostigmine salicylate (0.02 to 0.025 mg/kilogram) 1 to 1.5 mg per MWD is given by slow IV or IM injections. Repeated doses of physostigmine can be given at intervals of 1 to 2 hours until effective, then

redosed every 2 to 4 hours if signs of BZ exposure persist or recur. Continuous therapy may not be necessary since the effects of the exposure gradually disappear. If continuous administration is required, it should be carried out at reduced dosage levels to avoid an overdose of physostigmine. The signs of physostigmine overdose include pupillary constriction, muscle weakness, twitching, vomiting, diarrhea, respiratory distress, slowed heart rate, and convulsions. If toxicity is noted, further administration of physostigmine should be discontinued and one atropine injector should be given intramuscularly to control severe effects of overdose.

H-47. Fentanyl derivatives will cause death if respiratory system is compromised. Maintaining an open and clear airway is essential. Supplemental oxygen and intubation may be needed. The opioid antagonist naloxone (0.04 mg/kg IV, IM or subcutaneous) is an effective antidote.

# PROTECTION AGAINST BLISTER (VESICANT) AGENTS

H-48. The terms blister (vesicant) agents or vesicant are misnomers when applied to MWDs since vesiculation (blistering) generally does not occur in dogs or in most other animal species. Despite the lack of blistering, these agents do injure any part of the body they contact. The preventive measures used for nerve agents can also be used for blister (vesicant) agents. If a MWD must transit a contaminated area, it is best if it is placed in the transport kennel and carried. If a MWD must transit a contaminated area, its paws should be protected to prevent the blister (vesicant) agents from reaching the skin as described in the Chemical Agent Protection paragraph above.

H-49. Distilled HD is a colorless to a dark brown oily liquid with a garlic-like odor. It is used as a delayed-action casualty agent. The persistency depends upon the munitions used and the weather. Although HD is not persistent at high temperatures (100°F to 120°F), mustard vapor becomes a major hazard. In addition, with an increase in temperature (90°F) and humidity, there is a marked decrease in the effective dosage. Also, wet skin absorbs more mustard than dry skin.

## EFFECTS

H-50. Liquid mustard or mustard vapors produce delayed effects on the skin and eyes following exposure. The long hair of dogs does not prevent injury to the skin, but it does impede the penetration of liquids and vapors.

### Skin

H-51. Contamination of the skin is followed by a latent period, which varies in length with the degree of exposure. Within 1 hour after exposure, piloerection (erection of the hair) occurs at the site of exposure and may last for an hour or more. Two to three hours after that, redness and edema of the skin develop, increasing in intensity for 24 hours and then subsiding. In mild exposures, edema is followed by exfoliation of the epidermis of the skin. Severe exposures form ulcerated lesions. The lesions heal if secondary infection can be prevented or treated adequately. The skin of the abdomen, axilla, face, and feet are more susceptible to damage from HD and this sensitivity is not directly related to the length of hair protecting the rest of the MWD's body.

### Ocular

H-52. The eye is most sensitive to mustard's corrosive effects. Liquid mustard or heavy vapor exposures can be extremely damaging to the entire eye. Mild ocular exposures are followed by conjunctivitis and conjunctival edema, usually appearing within 1 or 2 hours, edema of the eyelids, corneal opacity and inflammation of the cornea, corneal roughening, and pain. More severe exposures can produce more serious lesions, resulting in necrotic conjunctivitis, corneal erosions or deep ulcerations, deep ophthalmic inflammation, and permanent corneal opacification due to scarring. These lesions predispose the eye to secondary bacterial infections.

### Respiratory Tract

H-53. Mild to severe exposures to mustard vapor damage the respiratory tract. Inhalation of blister (vesicant) agents vapors will produce sloughing and ulceration of the tracheobronchial mucosa first. Profuse

inflammatory exudation and edema may cause respiratory distress. More severe exposures produce involvement of the lung tissue, pulmonary edema, and acute pulmonary alveolar emphysema, and may become complicated by secondary purulent bronchopneumonia. The effects of respiratory exposures tend to develop over several days. The signs of respiratory involvement include cough, nasal discharge, respiratory difficulty, fever, and tracheal and pulmonary rales.

### Gastrointestinal Tract

H-54. Ingestion of contaminated food and water or the licking of contaminated body areas may produce ulceration of the alimentary mucous membranes, resulting in oral ulceration, abdominal pain, vomiting, bloody diarrhea, and prostration.

### Systemic

H-55. Systemic absorption of mustard can result from extremely high skin or respiratory exposures or from

absorption of the agent from the intestines. It may produce systemic effects involving the CNS, cardiovascular system, and hematopoietic system. The possibility of severe leukopenia and susceptibility to infection also exists. These effects are manifested by excitation, salivation, slowed heart rate, decreased count of white blood cells and platelets, bloody diarrhea, and shock.

## DECONTAMINATION

H-56. All persons who receive and handle contaminated MWDs must be in MOPP 4.

H-57. Because of the insidious action of mustard vesicants (where effects are not immediately apparent), decontamination may not be entirely effective. Yet, it is essential to decontaminate MWDs promptly after exposure to prevent more serious injuries and to mitigate the effects of exposure where possible. Decontamination should be carried out within the first minute or two after contamination with vesicants to prevent injury and before treatment is begun.

**CAUTION**

Decontamination should be accomplished as soon as possible to prevent contamination of handlers and treatment area.

H-58. Before redness and edema appear, localized areas of the skin can be decontaminated by using RSDL (as described in Chapter 3 and Appendix D) and washing the MWD with soap and water. Collars, muzzles, and leashes are also decontaminated by using the M295 Individual Equipment Decontamination Kit wipe-down mitt or by using a 5 percent hypochlorite solution.

H-59. The eyes must be decontaminated by copious water irrigation immediately after exposure. The RSDL should not be used in or around the eyes as it may cause additional ocular injury. Ophthalmic ointments should not be applied to the eye until after decontamination is completed and the eyes have been examined as they may absorb mustard agents and prolong corneal exposure thus increasing eye injury.

## TREATMENT

H-60. The treatment for either local or systemic effects of mustard blister (vesicant) agents is primarily symptomatic and similar to the treatment described in Chapter 5 for human casualties. Specific systemic and/or topical antibiotic therapy should be administered when indicated. Supportive therapy may be required to maintain the animal's nutritive and fluid status. With eye injuries, the degree of corneal damage should be determined with fluorescein stain and treated accordingly with antibiotic or antibiotic steroid ointments. The possibility of leukopenia, lung damage, sepsis, or others injuries may also exist.

### NITROGEN MUSTARDS

H-61. Nitrogen mustards is a colorless liquid when pure with a faint fishy or soapy odor. It is used as a delayed-action casualty agent that has a delay of hours or more before skin-damaging symptoms are felt. The eyes are very susceptible to low concentrations of HN, while a high concentration is required to significantly damage the skin or respiratory tract insofar as single exposure is concerned. Liquid and vapor exposures to HN are less damaging to the skin of MWDs than are equal concentrations of mustard or arsenical blister (vesicant) agents. Exposures of the eye to HN, however, produce more serious lesions than HD exposures do. The respiratory, gastrointestinal, and systemic effects of HN are similar to those effects caused by HD. Decontamination and therapy for HN are similar to those for HD.

## PROTECTION AGAINST LEWISITE AGENTS

H-62. Lewisite agents are more damaging as liquids than as vapors. Exposure to liquid arsenical blister (vesicant) agents is immediately painful and the exposed MWD becomes very restless. Lesions produced by these agents are more severe and develop faster than those produced by mustard. Liquid arsenicals on the skin and their inhaled vapors are readily absorbed into the systemic circulation, producing signs of arsenic exposure manifested by restlessness, vomiting, bloody diarrhea, shock, weakness, anemia, and pulmonary edema.

### DECONTAMINATION

H-63. Procedures for decontamination are the same as those applied for mustard.

### TREATMENT

H-64. The treatment protocol provided below is based on the availability of BAL ointment and BAL injectable (dimercaprol) that are not currently in the chemical agent patient treatment set but efforts are underway to procure these items for future sets. If available, the treatment protocols for the ointment and the BAL injectable are provided below.

H-65. The treatment of lesions induced by arsenical blister (vesicant) agents is similar to that for other blister (vesicant) agents. To treat localized skin exposures, BAL ointment can be rubbed into the contaminated areas, allowed to remain 5 minutes, and then washed off. Any other protective ointment on the skin must be removed before application of BAL ointment. When BAL ointment is applied, it will penetrate and neutralize arsenical blister (vesicant) agents.

H-66. Systemic treatment for arsenical blister (vesicant) agents is indicated when there is extensive skin exposure which has not been decontaminated within 15 minutes, when a very rapid onset of effects follows exposure, or when systemic signs of arsenic exposure appear. Systemic therapy consists of the administration of BAL injectable (dimercaprol) at 2.5 to 5.0 mg/kilogram by IM injection. Dosage can be repeated every 4 hours for 2 days and then two times per day for the next 10 days or until recovery is apparent. Supportive therapy should also be administered, as indicated.

## CHOKING (LUNG-DAMAGING) AGENTS

H-67. Chemical warfare agents that primarily cause pulmonary edema by attacking lung tissue have traditionally been classified as choking (lung-damaging) agents, or pulmonary edematogenic agents. They include CG, diphosgene, chlorine, ammonia, chloropicrin, HC obscurant, and NOx. Best known of these agents is CG.

H-68. The effects of choking (lung-damaging) agents in MWDs are similar to its effects in humans (delayed signs of breathing difficulty, coughing, wheezing, sneezing, and collapse). One difference is cyanosis (which is so prominent in human casualties of CG) is masked in MWDs. Decontamination is large amounts of warm water. For exposed MWDs, extreme exertion is dangerous, especially when pulmonary edema develops. Military working dogs in shock should be kept comfortably warm and given oxygen, if available. If pneumonia develops, treatment with antibiotics is indicated.

# IRRITANT AGENTS (RIOT CONTROL)

H-69. Under field conditions, the irritant agents CA, CN, and CS have little effect on MWDs. O-chlorobenzylidene malononitrile may cause increased respiration and hyperactivity. Liquid or solid agents in direct contact with the eyes will cause severe irritation; the eyes should, therefore, be flushed with saline or water. For skin decontamination, a 0.25 percent solution of sodium sulfite is more effective than saline or water in dissolving and neutralizing the irritant agent and should be used if available.

# OBSCURANT AND INCENDIARY AGENTS

H-70. Burning particles of WP cause deep burns on contact with the skin. The obscurant is generally not toxic. Since WP burns spontaneously when exposed to air, oxygen must be excluded to stop the burning. This may be done by submerging the burn or wound in water or by covering it with a water-soaked dressing. At the earliest opportunity, all WP should be removed from the skin as follows: bathe the affected part in a bicarbonate solution (no more than a 0.5 percent solution of sodium bicarbonate) to neutralize phosphoric acid, which then allows removal of visible WP. Remaining fragments will be observed in dark surroundings as luminescent spots. The burn should be debrided promptly if the MWD's condition will permit, to remove bits of phosphorus which might be absorbed later and possibly produce systemic effects. An ointment with an oily base should not be applied until it is certain that all phosphorus has been removed. Further treatment should be carried out as for thermal burns. Treatment with ultraviolet light is both palliative and therapeutic. If the eyes are affected, treatment should initially be commenced by irrigation, using water or saline. The lids must be separated and a local anesthetic instilled to aid in the removal of all imbedded particles. In eyes with severe ulceration, atropine ophthalmic ointment should be instilled once all particles have been removed.

# SULFUR TRIOXIDE-CHLOROSULFONIC ACID SOLUTION, TITANIUM TETRACHLORIDE, AND A CHEMICAL MIXTURE HEXACHLOROETHANE

H-71. Field concentrations of these agents usually are not harmful to MWDs, but the liquid may cause burns on the skin and in the eyes. After the eyes are irrigated, they are treated the same as for thermal burns.

# CYANIDE COMPOUNDS (BLOOD AGENTS)

H-72. Cyanide compounds (blood agents) affect bodily functions by inactivating the cytochrome oxidase system; this prevents cell respiration and the normal transfer of oxygen from the blood to body tissues. Hydrogen cyanide and CK are the important agents in this group. Cyanogen agents are highly volatile and, therefore, nonpersistent even at very low temperatures. Exposure at high concentrations cause effects within seconds and death within minutes in unprotected personnel and MWDs. Cyanogen chloride also produces central and peripheral pulmonary effects on the respiratory tract because of its chlorine component.

## ABSORPTION

H-73. These agents produce toxic effects after absorption. Inhalation is the usual route of entry. Artillery shells, mortar rounds, rockets, a sprayer mounted on aircraft, or bombs can disperse these agents.

## EFFECTS, DECONTAMINATION, AND TREATMENT

H-74. Hydrogen cyanide causes asphyxiation of the tissues, especially the respiratory center of the CNS. In addition to cyanide effects, CK causes marked local irritant effects on the respiratory system that can lead to pulmonary edema.

H-75. Decontamination for cyanide compounds is usually not necessary because the agents evaporate quickly. If MWD's hair is wet use large amounts of soap and water.

H-76. Treatment is difficult under field conditions. It should consist of oxygen therapy under positive pressure ventilation and injections of antidote medications.

H-77. Initial treatment for MWDs under 85 pounds is IV injection of one sodium nitrite 10 ml (3 percent) ampule containing 300 mg of sodium nitrite, followed by one 50 ml (25 percent) ampule containing 12.5 grams of sodium thiosulfate IV. Military working dogs over 85 pounds should receive an initial dose of two ampules of sodium nitrite, a total of 600 mg, and two ampules of sodium thiosulfate, a total of 25 grams. Sodium nitrite and sodium thiosulfate must be administered slowly through an IV (over 3 to 5 minutes).

H-78. If signs of intoxication continue additional medication may be needed. A small amount of venous blood should be examined visually. If the blood is chocolate brown, give an additional injection of sodium thiosulfate at one-half the initial dose quantity. If the venous blood is red, give additional injections of both sodium nitrite and sodium thiosulfate at one-half the initial dose quantity.

***Note***. The initial dosage of sodium nitrite is approximately 4 to 7 mg/pound (1.8-3.2 mg/kilogram) IV, followed immediately by sodium thiosulfate at approximately 150 to 300 mg/pound (68-136 mg/kilogram).

# TOXIC INDUSTRIAL CHEMICALS/TOXIC INDUSTRIAL MATERIALS OF CONCERN FOR MILITARY WORKING DOGS

H-79. Military Working Dogs are highly likely to come into contact with TICs and toxic industrial materials both during an incident as well as during their daily duties. Military Working Dogs are at greater risk of being exposed to TICs/toxic industrial materials due to breathing the air closer to the contaminated ground, walking across contaminated areas, and licking or grooming their paws or hair.

## HYDROCARBONS

H-80. Hydrocarbons include such compounds as gasoline, diesel fuel, motor oil, transmission fluid, general cleaners and degreasers, and lubricants. Exposure can occur via oral, ocular, inhalation (vapors, aerosols, particles, or dust), or dermal routes. Clinical signs include gastrointestinal (vomiting, diarrhea, abdominal pain), ocular (conjunctivitis, corneal irritation or necrosis), respiratory (nasal discharge, nasal bleeding, respiratory distress), dermal (burns, skin eruptions, dermatitis), and CNS (ataxia, seizures, coma). Hydrocarbon exposure may also lead to liver and kidney damage.

## POLYCHLORINATED BIPHENYLS

H-81. Polychlorinated biphenyls have been banned in the U.S. since 1979. However, older buildings and equipment may still contain polychlorinated biphenyls which could be released during a disaster or fire. Polychlorinated biphenyls can be aerosols, vapors, oily liquids or solids. Exposure can occur via oral, inhalation, or dermal routes and leads to liver and kidney damage. Additional effects including but not limited to gastric injury, thyroid suppression, anemia, skin lesions, reproductive effects, and neurobehavioral abnormalities are also possible.

## HAZARDOUS METALS

H-82. Hazardous metals are of concern when buildings or equipment are damaged or destroyed. Explosions and fires may increase the risk for release of particles or fumes, such as lead fumes from lead piping or arsenic from burning pressure-treated lumber. Some of the more likely hazardous metals MWDs may encounter are arsenic, lead, zinc, cadmium, chromium, thallium, mercury, nickel, cobalt, and beryllium. Exposure is mainly via inhalation but can also be orally. Clinical signs include nonspecific respiratory (coughing, respiratory distress, pneumonitis) and gastrointestinal (vomiting, diarrhea, abdominal pain) abnormalities.

## ASBESTOS

H-83. Asbestos is still used in insulation and concrete and is only a concern when buildings are damaged due to explosions or compressive forces. Exposure is mainly due to inhalation with initial clinical signs of coughing and bronchitis.

## SOAPS AND DETERGENTS

H-84. Soaps are only mildly irritating to gastrointestinal and mucous membranes. More alkaline detergents cause ocular, dermal, and gastrointestinal irritation and potentially corrosive burns and ulcerations to those systems as well.

H-85. Decontamination for soaps and detergents is large amounts of water.

## ACIDS AND ALKALIS

H-86. Acids and alkalis are common in many cleaning products. Exposure can occur via oral, ocular, inhalation (acid aerosols, mists, or vapors), and dermal routes. Clinical signs include mild to moderately severe burns for oral, ocular, and dermal exposure and dyspnea and pulmonary edema for inhalation exposure.

H-87. Decontamination for acids and alkalis is large amounts of water.

## ETHYLENE GLYCOL AND PROPYLENE GLYCOL

H-88. Ethylene glycol and propylene glycol are found in various household and industrial compounds. Exposure is mainly oral with CNS clinical signs of ataxia, weakness, and depression that can progress to renal failure, seizures, and liver damage.

H-89. Ethylene glycol treatment is as follows:

- Within 1 to 2 hours of ingestion—induce vomiting or gastric lavage (or both) followed by administration of activated charcoal.
- After 1 to 2 hours—administer 4-Methylpyrazole (5 percent solution [50 mg/ml]) 20 mg/kilogram IV, initially; followed by 15 mg/kilogram IV at 12 and 24 hours after ingestion, and 5 mg/kilogram IV at 36 hours after ingestion.
- Supportive treatment—correction of fluid, electrolyte, and acid-base disorders.

***Note.*** Temporary dialysis to clear the toxin from the system is the preferred therapy for human patients and has been used with great success in veterinary medicine. Access to this level of intensive care is often limited, especially in field environments. However, this is the recommended ideal course of therapy if equipment and veterinary expertise is available.

H-90. Propylene glycol treatment—supportive treatment.

## PHENOLS

H-91. Phenols may be present in large quantities in industrial settings. At low concentrations (<4.5 percent), phenols are irritants; and at concentrations >5 percent, phenols are caustic. Exposure can occur via oral and dermal routes. Clinical signs include oral and esophageal burns, panting, profuse vomiting and diarrhea, salivation, muscle tremors, convulsions, coma, and death and can lead to live and kidney damage.

H-92. Decontamination for phenols is initially large amounts of water under pressure until the phenol odor is gone. Rapid skin decontamination is critical. Flush eyes with copious amounts of water or saline. After the smell of phenol is gone, decontaminate with large amounts of water and soap.

H-93. In cases of ingestion, do not induce emesis. Administer activated charcoal.

## ALCOHOLS

H-94. Unless ingested in large quantities, alcohols do not generally cause severe problems. Exposure occurs via oral, inhalation, and dermal routes. Clinical signs include ataxia, CNS depression, dyspnea, and gastritis. With significant exposures, aspiration is highly likely.

## GASES

H-95. Common harmful gases are bromine, chlorine, fluorine, hydrogen sulfide, and carbon monoxide.

Bromine gas is used in agriculture and sanitation and petrochemical industries and causes coughing, nosebleeds, and pulmonary edema. Chlorine gas is used in many industries and for sewage treatment and causes ocular and respiratory irritation and may cause pulmonary edema. Fluorine gas is used in aluminum and petrochemical industries as well as dyes, agricultural chemicals, and ceramics and causes severe ocular and respiratory irritation. Hydrogen sulfide is produced by various industries such as paper mills, food processing plants, and petroleum refineries. Hydrogen sulfide smells like rotten eggs and because it is heavier than air, it accumulates in low-lying areas. Clinical signs from hydrogen sulfide are conjunctivitis and pulmonary edema, but large exposures can rapidly induce unconsciousness and seizures. Carbon monoxide usually only poses a risk when entering poorly ventilated, confined spaces containing motor vehicle obscurant or exhaust. Mild clinical signs of CO exposure are vomiting and lethargy; moderate clinical signs are dyspnea, weakness, ataxia, tachypnea, and tachycardia; and severe clinical signs are disorientation, severe lethargy, hypotension, syncope, seizures, pulmonary edema and coma.

# Glossary

This glossary lists acronyms and terms with Army or joint definitions. Where Army and joint definitions differ, (Army) precedes the definition. Terms for which ATP 4-02.85/MCRP 4-11.1A/NTRP 4-02.22/AFTTP(I) 3-2.69 is the proponent are marked with an asterisk (*). The proponent publication for other terms is listed in parentheses after the definition.

## SECTION I – ACRONYMS AND ABBREVIATIONS

| | |
|---|---|
| **AFI** | Air Force instruction |
| **AFTTP(I)** | Air Force tactics, techniques, and procedures (instruction) |
| **AMedP** | allied medical publication |
| **AR** | Army regulation |
| **ATNAA** | Antidote Treatment Nerve Agent, Autoinjector |
| **ATP** | Army techniques publication |
| **attn** | attention |
| **C** | Celsius |
| **CANA** | convulsant antidote for nerve agent |
| **CBRN** | chemical, biological, radiological, and nuclear |
| **CNS** | central nervous system |
| **CO** | carbon monoxide |
| **CPS** | collective protective shelter |
| **CW** | chemical warfare |
| **CWC** | Chemical Weapons Convention |
| **DA PAM** | Department of the Army pamphlet |
| **DOD** | Department of Defense |
| **DODD** | Department of Defense directive |
| **DODI** | Department of Defense instruction |
| **EUA** | emergency use authorization |
| **F** | Fahrenheit |
| **FDA** | Food and Drug Administration |
| **FHP** | force health protection |
| **FM** | field manual |
| **HSS** | health service support |
| **HRCoE** | Health Readiness Center of Excellence |
| **IM** | intramascular |
| **IND** | investigational new drug |
| **IV** | intravenous |
| **JP** | joint publication |
| **MCRP** | Marine Corps reference publication |
| **MCWP** | Marine Corps warfighting publication |
| **MOPP** | mission-oriented protective posture |

| | |
|---|---|
| **MTF** | medical treatment facility |
| **MWD** | military working dog |
| **NATO** | North Atlantic Treaty Organization |
| **NAVSUP P** | Navy supplement publication |
| **NBC** | nuclear, biological, and chemical |
| **NTRP** | Navy technical reference publication |
| **NTTP** | Navy tactics, techniques, and procedures |
| **OPNAV M** | Office of the Chief of Naval Operations manual |
| **OSHA** | Occupational Safety and Health Administration |
| **PB** | pyridostigmine bromide |
| **PPW** | patient protective wrap |
| **RSDL** | reactive skin decontamination lotion |
| **SB** | supply bulletin |
| **SNAPP** | Soman Nerve Agent Pretreatment Pyridostigmine |
| **STANAG** | standardization agreement |
| **TB MED** | technical bulletin (medical) |
| **TIC** | toxic industrial chemical |
| **U.S.** | United States |
| **USMC** | United States Marine Corps |
| **USAMEDDC&S** | United States Army Medical Department Center and School |
| **WP** | white phosphorus |

## SECTION II – TERMS

**blister agent**

A chemical agent that injures the eyes and lungs, and burns or blisters the skin. Also called vesicant agent. (JP 3-11).

**blood agent**

A chemical compound, including the cyanide group, that affects bodily functions by preventing the normal utilization of oxygen by body tissues. (JP 3-11).

**chemical agent**

A chemical substance that is intended for use in military operations to kill, seriously injure, or incapacitate mainly through its physiological effects. (JP 3-11).

**chemical hazard**

Any chemical manufactured, used, transported, or stored that can cause death or other harm through toxic properties of those materials, including chemical agents and chemical weapons prohibited under the Chemical Weapons Convention as well as toxic industrial chemicals. (JP 3-11).

**chemical warfare**

All aspects of military operations involving the employment of lethal and incapacitating munitions/agents and the warning and protective measures associated with such offensive operations. Also called CW. (JP 3-11).

**chemical weapon**

Together or separately, (a) a toxic chemical and its precursors, except when intended for a purpose not prohibited under the Chemical Weapons Convention; (b) a munition or device, specifically designed to cause death or other harm through toxic properties of those chemicals specified in (a), above, which would be released as a result of the employment of such munition or device; (c) any equipment

specifically designed for use directly in connection with the employment of munitions or devices specified in (b), above. (JP 3-11).

***choking agent**

A chemical warfare agent which produces irritation to the eyes and upper respiratory tract and damage to the lungs, primarily causing pulmonary edema. Also known as lung-damaging agent.

**contamination**

1. The deposit, absorption, or adsorption of radioactive material, or of biological or chemical agents on or by structures, areas, personnel, or objects. Also called fallout radiation. 2. Food and/or water made unfit for consumption by humans or animals because of the presence of environmental chemicals, radioactive elements, bacteria or organisms, the by-product of the growth of bacteria or organisms, the decomposing material or waste in the food or water. (JP 3-11).

**definitive identification**

The employment of multiple state-of-the-art, independent, established protocols and technologies by scientific experts in a nationally recognized laboratory to determine the unambiguous identity of a chemical, biological, radiological, and/or nuclear hazard with the highest level of confidence and degree of certainty necessary to support strategic-level decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

**field confirmatory identification**

The employment of technologies with increased specificity and sensitivity by technical forces in a field environment to identify chemical, biological, radiological, and/or nuclear hazards with a moderate level of confidence and the degree of certainty necessary to support follow-on tactical decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

**incapacitating agent**

A chemical agent, which produces temporary disabling conditions that can be physical or mental and persist for hours or days after exposure to the agent has ceased. (JP 3-11).

**nerve agent**

A potentially lethal chemical agent that interferes with the transmission of nerve impulses. (JP 3-11).

**nonpersistent agent**

A chemical agent that when released dissipates and/or loses its ability to cause casualties after 10 to 15 minutes. (JP 3-11).

**persistent agent**

A chemical agent that, when released, remains able to cause casualties for more than 24 hours to several days or weeks. (JP 3-11).

**presumptive identification**

The employment of technologies with limited specificity and sensitivity by general-purpose forces in a field environment to determine the presence of a chemical, biological, radiological, and/or nuclear hazard with a low level of confidence and the degree of certainty necessary to support immediate tactical decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

**riot control agent**

Any chemical, not listed in a schedule of the Convention on the Prohibition of the Development, Production, Stockpiling and Use of Chemical Weapons and on Their Destruction that can produce rapidly in humans sensory irritation or disabling physical effects that disappear within a short time following termination of exposure. Also called RCA. (JP 3-11).

**theater validation identification**

The employment of multiple independent, established protocols and technologies by scientific experts in the controlled environment of a fixed or mobile/transportable laboratory to characterize a chemical, biological, radiological, and/or nuclear hazard with a high level of confidence and the degree of certainty necessary to support operational-level decisions (ATP 3-11.37/MCWP 3-37.4/NTTP 3-11.29/AFTTP 3-2.44).

**toxic industrial chemical**

A chemical developed or manufactured for use in industrial operations or research by industry, government, or academia that poses a hazard. Also called TIC. (JP 3-11).

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