U.S. Army Medical Research Institute of Chemical Defense (USAMRICD)

MEDICAL MANAGEMENT OF CHEMICAL CASUALTIES HANDBOOK

Chemical Casualty Care Division
USAMRICD
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USAMRICD
Chemical Casualty Care Division's
MEDICAL MANAGEMENT OF CHEMICAL CASUALTIES HANDBOOK

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EDITORS

COL (Ret) Gary Hurst, MC, USA
LTC Shirley Tuorinsky, AN, USA
COL James Madsen, MC, USA
COL Jonathan Newmark, MC, USA
LTC Benjamin Hill, MC, USA
Lt Col Charles Boardman, USAF
SFC Jeffrey Dawson, USA

ACKNOWLEDGEMENTS

Ms. Patricia Hurst
Ms. Leslie Key
Mr. Laukton Rimpel
Mr. Daniel Boehm
Ms. Crystal Chadwick

Disclaimer

The purpose of this Handbook is to provide concise, supplemental reading material for attendees of the Medical Management of Chemical Casualties Course. It is to be used as a guide in the chemical arena and not to replace official doctrine.

Every effort has been made to make the information contained in this Handbook consistent with official policy and doctrine.

This Handbook, however, is not an official Department of the Army publication, nor is it official doctrine. It should not be construed as such unless it is supported by other documents.

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INTRODUCTION

PURPOSE

Poisoning, that is, exposure to toxic chemicals, a process also called intoxication, has been an important medical issue for centuries. A particularly frightening type of poisoning is the generation of military mass casualties on the battlefield by the use either of chemicals developed specifically for that purpose, or of chemicals produced for industry and co-opted for battlefield use. In addition, these agents can produce civilian casualties during warfare and can lead to both military and civilian casualties from terrorist use in settings remote from a defined battlefield.

Military healthcare providers have a special responsibility to recognize and manage chemical casualties whatever the setting. First, military healthcare providers will likely be the first medical personnel to receive exposed warfighters, sailors, or airmen. Secondly, because of the preeminence of the U.S. military medical establishment in research, response, and training concerning chemical warfare agents, military medical personnel must be able to respond to chemical exposures at U.S. stockpile sites of chemical warfare agents and to provide expert consultation to their civilian

counterparts in the event of a terrorist attack involving these agents. Knowledge of the medical aspects of the prevention, preparedness, response, and recovery phases of any military or civilian event involving chemical exposure is expected of every military medical care provider.

This handbook has been produced to help address the need for training military medical officers. Its primary use is as an adjunct, together with a companion biological agents handbook, to the Medical Management of Chemical and Biological Casualties (MCBC) Course. It is not designed to replace the hands-on training afforded by this course, but it has been designed to help MCBC students both during the course and after its completion.

Medicine is a changing art and science, and the treatment protocols and drug dosages described in this handbook are not to be construed as replacements for sound clinical judgment or to be inflexible rules incapable of change as new information becomes available. It is the responsibility of the users to avail themselves of the latest available resources. One such resource is the Internet site of the Chemical Casualty Care Division of the U.S. Army Medical Research Institute of Chemical Defense (USAMRICD): http://ccc.apgea.army.mil. Registration at this

site is strongly encouraged so that access to the user-restricted portions of the site in an emergency will not be delayed. The user-restricted area of this site includes the text of the latest published edition of this handbook as well as other important references for chemical casualty care.

CHEMICAL AGENTS

GENERAL CONCEPTS AND TERMINOLOGY

To paraphrase Paracelsus, any substance delivered in excess can act as a poison; it is the dose that makes the poison. Nevertheless, the term *poison* traditionally refers to chemicals that are sufficiently potent to induce poisoning in relatively low doses. The terms *toxic chemical*, *chemical agent*, and *chemical warfare agent* are often defined in different ways, and their use in this handbook needs to be clarified at the outset along with the use of related terms.

The Chemical Warfare Convention defines a *toxic chemical* as “any chemical which through its chemical action on life processes can cause death, temporary incapacitation or permanent harm to humans or animals;" specifies that this definition includes “all such chemicals, regardless of their origin of their method of production;” and specifically includes toxins such as ricin and saxitoxin.

The U.S. Army defines *chemical warfare (CW) agents* (often called simply *chemical agents*) as toxic substances developed for military use to produce death, serious injury, or incapacitation through their toxicological effects

in exposed humans or animals and officially excludes from this definition three broad categories of chemicals: (1) riot-control agents, (2) herbicides, and (3) smoke and flame materials

Toxic industrial materials, or TIMs, are industry-associated materials with harmful effects on humans; they can be subdivided *into toxic industrial biologicals*, or TIBs; *toxic industrial chemicals*, or TICs; and *toxic industrial radiologicals*, or TIRs. The North Atlantic Treaty Organization (NATO) has a more restricted definition for *toxic industrial chemical* as a chemical that (1) is more toxic than ammonia (that is, has an LCt_{50} of 100,000 mg-min/m^3 or less [see later in this section] and (2) is produced in quantities greater than 30 tons per year at a given production facility. However, TICs are also commonly defined as any industrially produced chemicals that could be used to produce mass casualties.

Toxicant is a general synonym for *poison* and can be used interchangeably with the latter term. *Toxin* is also commonly used as a synonym for *poison* but in discussions of chemical and biological agents is best defined in its more restricted sense as a toxic chemical synthesized by a living organism. Toxins have characteristics of both chemical and biological agents and are increasingly being separately

classified as *mid-spectrum agents*, which include not only toxins but also bioregulators (small molecules with regulatory functions in the body in physiological doses but with toxic effects in larger doses), synthetic viruses, and genocidal weapons.

Chemical, biological, and mid-spectrum agents are often referred to as *weapons of mass destruction*, or *WMD*, and the official military definition of WMD in fact includes these three kinds of agents. However, in the strict sense of the word, weapons of mass destruction are weapons capable of causing extensive damage to physical structures, such as buildings, and include high explosives and nuclear and thermonuclear bombs. Chemical agents, biological agents, toxins, and point sources of radiation may cause mass casualties while leaving structures intact; and a better term for these kinds of weapons is *mass-casualty weapons*, or *MCW*, a term that is gradually gaining currency and that this handbook recommends when referring to chemical agents. *Unconventional weapons* is a term used to refer to chemical agents, biological agents, toxins, nuclear and thermonuclear bombs, radiological dispersal devices (also called RDDS, or “dirty bombs”), and point sources of radiation used as weapons. It does not, however, include conventional high explosives.

The list of chemical warfare agents officially designated as such by the U.S. military includes those chemicals that are intended to cause death or serious injury and also those intended to cause incapacitation, that is, temporary inability to perform one's military duties. The former are called *toxic agents* and include (1) lung-damaging agents (also called pulmonary or choking agents), (2) "blood" agents (specifically, cyanide compounds), (3) vesicants (blistering agents), and (4) nerve agents; those designed to produce only temporary incapacitation are referred to as *incapacitating agents*. This handbook will address each of these groupings of "official" chemical warfare agents as well as riot-control agents, which are technically not chemical warfare agents according to the U.S. military definition, but they are widely used in law enforcement for mass incapacitation.

Chemical agents may have chemical names and may also have common names. Every chemical agent developed for military use in the U.S. also has a NATO code, a one- to three-letter designation assigned after World War II (WWII) by the NATO to provide a standard shorthand recognizable in any of the languages used by NATO countries. Thus, the chemical compound O-isopropyl methylphosphonofluoridate has the common name sarin and the NATO code GB. This handbook will make extensive use of NATO

codes as well as common names for chemical agents.

PHYSICAL FORMS OF CHEMICAL AGENTS

Chemical agents, like all other substances, may exist as solids, liquids, or gases, depending on temperature and pressure. Except for riot-control agents and the incapacitating agent BZ, which are solids at usually encountered temperatures and pressures, chemical agents in munitions are liquids. Following detonation of the munitions container, the agent is primarily dispersed as liquid or as an aerosol, defined as a collection of very small solid particles or liquid droplets suspended in a gas (in this case, in the explosive gases and the atmosphere). Thus, "tear gas," a riot-control agent, is not really a gas at all, but rather an aerosolized solid. Likewise, mustard "gas" and nerve "gas" do not become true gases even at the boiling point of water ($212^{o}F/100^{o}C$ at sea level).

Certain chemical agents, such as hydrogen cyanide, chlorine, and phosgene, may be gases when encountered during warm months of the year at sea level. The nerve agents and mustard are liquids under these conditions, but they are to a certain extent volatile; that is, they volatilize or evaporate, just as water or gasoline does, to form an often invisible vapor. A vapor is the gaseous form of a substance that is normally a

liquid at usual environmental temperatures. Another way of conceptualizing a vapor is to regard it as the gaseous form of a substance at a temperature lower than the boiling point of that substance at a given pressure. Liquid water, for example, becomes a gas (steam) when heated to its boiling point at a given pressure, but below that temperature, it slowly evaporates to form water vapor, which is invisible. Visible water clouds (including “clouds of steam”) are composed neither of water vapor nor water gas (steam) but rather are suspensions of minute water droplets, i.e., an aerosol.

The tendency of a chemical agent to evaporate depends not only on its chemical composition and on the temperature and air pressure, but also on such variables as wind velocity and the nature of the underlying surface with which the agent is in contact. Just as water evaporates less quickly than gasoline but more quickly than motor oil at a given temperature, pure mustard is less volatile than the nerve agent GB but is more volatile than the nerve agent VX. However, all of these agents evaporate more readily when the temperature rises, when a strong wind is blowing, or when they are resting on glass rather than on, for example, porous fabric.

Volatility is thus inversely related to persistence, because the more volatile a

substance is, the more quickly it evaporates and the less it tends to stay or persist as a liquid and contaminate terrain and materiel. The liquid hazard of a persistent agent is often more significant than the danger created by the relatively small amounts of vapor that it may generate. The converse is true of nonpersistent agents, which may pose a serious vapor hazard but which also evaporate quickly enough not to create a liquid hazard for an extended time. The arbitrary but generally accepted divisions between persistent and nonpersistent agents is 24 hours, meaning that a persistent agent will by definition constitute a liquid hazard and contaminate surfaces for 24 hours or longer. Such agents (mustard and VX) are thus suitable for contaminating and denying terrain and materiel to the enemy. Nonpersistent agents, such as GB and cyanide, find tactical employment in the direct line of assault into enemy territory since they will have evaporated within a day and will no longer contaminate surfaces. These generalizations are obviously subject to the modifying factors of temperature, environmental factors such as wind, and surface characteristics.

EXPOSURE, ABSORPTION, AND TOXICITY OF CHEMICAL AGENTS

Toxicologically speaking, exposure means contact of a poison with an epithelial surface

(such as the skin, respiratory epithelium, eyes, or gastrointestinal mucosa) and is quantified as external dose. Penetration of an epithelial barrier is called absorption and results in an absorbed dose, or internal dose. Furthermore, an absorbed agent may exert local effects at or near the site of exposure and absorption or systemic effects following distribution in the circulation to sites remote from the exposure site. Biological effects occur following exposure to chemical agents dispersed as solids, liquids, gases, aerosols, or vapors. Eye or skin injury may follow direct exposure to the suspended solid particles of aerosolized riot-control agents, and inhalation of these agents brings the aerosolized solid in contact with the epithelium of the respiratory tree. Nevertheless, systemic effects from exposure to riot-control agents are rare. Contact of the eyes, or more likely the skin, with liquid nerve or vesicant agents may produce local effects or lead to absorption and systemic effects.

Liquid exposure is the most important hazard associated with persistent agents and necessitates the proper wearing of chemical protective clothing. At low temperatures, hydrogen cyanide (AC), cyanogen chloride (CK), and phosgene (CG) exist as liquids. However, because of their high volatility (low persistence), they seldom present a significant liquid hazard unless the area of exposure is large or

evaporation is impeded by trapping of liquid agent in saturated, porous clothing. Penetration of shrapnel or clothing contaminated with liquid chemical agent of any type may also lead to intramuscular or intravenous exposure and subsequent systemic effects.

Chemical agents in the form of aerosolized liquid droplets, vapor, or gas may directly contact the eyes, skin, or (through inhalation) the respiratory tree. Local damage is possible at any of these sites, but systemic absorption through dry, intact skin is usually less important than with the other routes. Vapor or gas exposure to the eyes, and especially the respiratory tree, is the most important hazard associated with nonpersistent agents and necessitates the proper wearing of a mask that provides both ocular and inhalation protection.

Specialized terms refer to the amount of chemical agent encountered during an exposure. The ED_{50} and the ID_{50} denote the quantities (usually measured as the weight in μg, mg, or g) of liquid agent that will predictably cause effects (E) or incapacitation (I) in 50% of a given group. Similarly, the LD_{50} is the lethal dose or quantity (weight) of liquid agent that will kill 50% of a group. Note that the lower the LD_{50}, the less agent is required and thus the more potent is the agent. Because of differences in absorption, the ED_{50} and LD_{50} values for a given

agent are site-specific; i.e., the LD_{50} for mustard absorbed through dry, unabraded skin is much higher than the LD_{50} for mustard absorbed through the eye.

Comparison of the amounts of chemical agent encountered as aerosol, vapor, or gas requires use of the concentration-time product or Ct, which refers to the agent concentration (usually in mg/m^3) multiplied by the time (usually in minutes) of exposure. For example, exposure to a concentration of 4 mg/m^3 of soman (GD) vapor for 10 minutes results in a Ct of 40 $mg \cdot min/m^3$. Exposure to 8 mg/m^3 for 5 minutes results in the same Ct (40 $mg \cdot min/m^3$). For almost any given agent (with the notable exception of cyanide, which will be discussed in a separate chapter), the Ct associated with a biological effect is relatively constant even though the concentration and time components may vary within certain limits (Haber's Law). Therefore, a 10-minute exposure to 4 mg/m^3 of soman causes the same effects as a 5-minute exposure to 8 mg/m^3 of the agent or to a one-minute exposure to 40 mg/m^3. The ECt_{50}, ICt_{50}., and $LCt_{50,}$ then correspond for vapor or gas exposures to the ED_{50}, ID_{50}, and LD_{50}, respectively, for liquid exposure and are likewise site-specific. However, the concentration-time product does not take into account variables such as respiratory rate and depth and is

therefore not an exact measure of inhalation exposure.

GENERAL PRINCIPLES OF CHEMICAL CASUALTY CARE

Chemical casualties must be *recognized* and appropriately *managed*. Management includes *triage* (i.e., sorting for medical treatment), *medical treatment*, *decontamination* (if liquid contamination is present), and *disposition*, which may include evacuation and, hopefully, eventual return to duty.

Casualty Recognition. Recognition of a chemical casualty is heavily dependent upon recognition and differential diagnosis of *toxidromes*, i.e., constellations of symptoms and signs that characterize exposure to a specific agent or kind of agent. This handbook will present distinct toxidromes characteristic of exposure to central-compartment lung-damaging agents, peripheral-compartment lung-damaging agents, cyanide compounds, sulfur mustard, Lewisite, phosgene oxime, nerve agents, anticholinergic incapacitating agents, "traditional" riot-control agents, and vomiting agents (a subset of riot-control agents). It is important to recognize these toxidromes and also to recognize other conditions that could mimic them.

Chemical agent casualties may be exposed to more than one agent and may also have other diagnoses, such as concomitant exposure to other kinds of mass-casualty agents, pre-existing medical or surgical conditions, or trauma. It is just as important to view the casualty, and the situation, as a whole as it is to identify chemical exposure. The best way to accomplish this is to use a toxicological perspective, i.e., to consider the triad of *agent*, *environment*, and *host*. Specifically, the astute clinician will think of (1) the agent; (2) the form(s) or state(s) of the agent in the environment; (3) the passage of agent from the environment to the host, i.e., the route or routes of entry; (4) whether exposure and absorption of the host are producing only local effects (restricting to the site of exposure) or are also generating systemic effects (effects remote from the site of exposure); (5) the severity of effects and of exposure; (6) the time course of effects; (7) other possible diagnoses (*instead of* the originally considered diagnosis, i.e., differential diagnoses; and *in addition to* the originally considered agent, i.e., concomitant diagnoses); and (8) any possible interaction or synergism among co-existing diagnoses. Without a consideration of these factors, treating healthcare providers have a tendency to focus on the chemical exposure, sometimes to the neglect of other life-threatening conditions.

Casualty Management. Once a chemical casualty has been recognized, appropriate management must begin. *Triage* is the process of sorting casualties for medical treatment when not all casualties can receive care simultaneously. It is in some ways both an assessment step and a management step and can act as a bridge between initial assessment and management. The triage categories used by the U.S. military are (1) *immediate* (those who are in danger of losing their lives unless action is taken within a few minutes), (2) *delayed* (those who require significant medical intervention but who can tolerate a somewhat longer delay in treatment), (3) *minimal* (casualties who have minor injuries, illnesses, or intoxications, often amenable to self or buddy care), and (4) *expectant* (those who are gravely ill and who cannot be treated with available resources without causing the death, by misallocation of resources, of other casualties). In this handbook, triage for specific kinds of chemical agents will be addressed within the chapter devoted to that agent type.

Initial management for severely affected chemical casualties (especially those who are triaged as immediate) can conveniently be summarized as an application of the **ABCDD**s, i.e., A) **A**irway, B) **B**reathing, C) **C**irculation, D_1) immediate **D**econtamination, and D_2) **D**rugs, i.e., specific antidotes. These interventions will be

described in specific agent chapters and also in the sections on decontamination and casualty management in a contaminated area. The latter section will also address the issues of patient *disposition*, including evacuation and return to duty.

Eight appendices (Appendices A through H) at the end of this handbook summarize and illustrate major concepts from the preceding sections of this publication, and following the index, review cards for each type of agent will help the reader review the material presented previously.

This handbook is meant to be as user-friendly as possible. New editions build upon new information and also are heavily dependent upon reader feedback. Please consider helping with the next edition by sending your comments and suggestions to the Chemical Casualty Care Division at ccc@apg.amedd.army.mil.

LUNG-DAMAGING AGENTS: Toxic Industrial Chemicals

CG, Cl

SUMMARY

Signs and Symptoms: Central effects: Eye and airway irritation, dyspnea. Peripheral effects: Chest tightness, and **delayed** pulmonary edema.

Field Detection: The Chemical Agent Detector Kit and the M93A1 FOX RECONNAISSANCE System will detect small concentrations of CG; however, they will not detect Cl.

Decontamination: **Vapor** - fresh air; **Liquid** - copious water irrigation.

Management: Termination of exposure, ABCs of resuscitation, enforced rest and observation, oxygen with or without positive airway pressure for signs of respiratory distress, other supportive therapy as needed.

LUNG-DAMAGING AGENTS: TOXIC INDUSTRIAL CHEMICALS

OVERVIEW

Over 1,800 toxic industrial chemicals (TICs) are used in industry, stored at industrial sites, and transported on the world's road and rail systems. Some of these chemicals were deployed as chemical warfare agents during World War I (WWI), killing and injuring thousands, and can have the same deadly consequences today if released during an accident or through terrorist sabotage. Death from exposure to TICs is more frequent when they are inhaled. Inhaling TICs in the form of a gas, vapor (gas coming from a liquid), or aerosol (liquid or solid particles suspended in a gas) can cause a sudden closure of the larynx (laryngospasm) causing the victim to become unconscious and collapse. TICs can also cause damage to the tissues of the upper airways, resulting in swelling, scarring, and airway narrowing, which can restrict breathing. TICs can damage lung tissues, allowing body plasma and other fluids to leak into the lung air sacs (alveoli), causing pulmonary edema and death from asphyxiation.

Warfighters are frequently exposed to TICs and military medical personnel must know about these agents so that they can help reduce troop exposure and prevent serious injury from them.

Understanding the Respiratory System. The respiratory system can be divided into two parts. Understanding these parts can greatly simplify the treatment problem-solving process.

Central Airway. This includes the nasopharynx (nose), oropharynx (mouth), larynx (vocal cords), trachea and bronchi (airway from the throat into the lungs). Tissues in this area are very moist and thin and can be damaged by TICs.

Peripheral Airway. This includes the lung sacs (alveoli) distributed throughout the lung tissue. During normal respiration, inhaled gasses fill the alveoli and then move slowly through their walls. The gasses then move through the thin walls of the blood vessels, capillaries, surrounding the alveoli and into the blood. TICs can damage the walls of alveoli and the capillaries surrounding them, allowing blood plasma and cells to leak into the air space of the alveoli.

PHYSIOCHEMICAL CHARACTERISTICS

Lung-damaging TICs are typically heavier than air and hang close to the ground when released. They tend to evaporate and disperse very quickly, depending on temperature and wind conditions. If the TIC is in liquid form at room temperature, then it will tend to give off a vapor. Vapors can become trapped in clothing fibers and “off-gas” to affect those who are nearby and have no respiratory protection. Although skin decontamination after vapor exposure is not a high priority, clothing should be removed and the underlying skin decontaminated with soap and water.

	Molecular Weight	Boiling Point	Freezing Point	Characteristic Odor
Phosgene (CG)	99	7.6°C	-128°C	Newly mown hay
Hydrogen Chloride (HCl)	36.46	-85°C	-114°C	Pungent
Ammonia	17.03	-33.35 °C	-77.7°C	Sharp, intensely irritating
PFIB	Polymer	214 - 217°C	257- 263°C	None
Oxides of Nitrogen (NOx)	28.1	-195.8 °C	-210°C	Irritating
White Phosphorus Smoke (P_4)	123.895	280°C	44.1°C	Garlic-like

DETECTION

Chlorine and ammonia have their own distinctive odors. Phosgene smells like newly cut grass, newly mown hay, or green corn. It is important to remember that odor is not a reliable detection method, and this does not protect against toxic inhalation effects. There are no specific field detection devices for these compounds; however, the ACADA can detect battlefield agent vapors when deployed as an area surveillance tool.

PROTECTION

The military mask, if fitted with a C2A1 filter canister, will protect against chlorine, phosgene, PFIB, NOx, and HC smoke in the open battlefield. Specific filters, or the use of a self-contained breathing apparatus with its own air supply, are mandated for other TICs such as ammonia. Masks will not be effective in environments where the TIC displaces oxygen, such as occurs with carbon dioxide.

TOXICITY

The LCt_{50} of phosgene is approximately 3200 mg-min/m^3, which is half the LCt_{50} (6,000 mg-min/m^3) of chlorine. Since only half as much phosgene is required to kill half of an exposed

group, phosgene is thus twice as potent as chlorine. Perfluoroisobutylene (PFIB) is 10 times more toxic than phosgene. Listed below are the OSHA standards for exposure limits in parts per million.

AGENT	CG	Cl	Ammonia	PFIB	NOx	White Phosphorus Smoke
Concen. Exposure Limits (ppm)	0.1	5	50	0.01	25	1

Lung-Damaging Agents

TICs are numerous. Those that pose a frequent threat to the warfighter in the field are listed here. Though the list is not complete, casualties from other lung-damaging agents are managed the same way as these examples. In low doses highly reactive TICs have a greater effect on the central airway; other TICs act on both airways; and still others that are not as reactive in the central airway travel deeper in the respiratory tract and destroy the tissues of the alveoli in the peripheral airways. Any TIC inhaled in large doses will cause damage to both central and peripheral airways.

CENTRALLY-ACTING TICs

Ammonia. This highly caustic and reactive gas is used for industrial refrigeration, for cleaning, for the processing of some illicit drugs, and for numerous legitimate industrial processes. It is a good example of a TIC that, in low doses, is primarily centrally acting. It rapidly forms a strong base (alkali) when it contacts the moist tissues of the central airway. The alkali burns and destroys the tissues it contacts. The victim may suddenly go into laryngospasm and collapse. The tissues of the airway will also become swollen. Scar tissue may form along the airway. Frequently, damaged tissue in the airway will die, slough off, and obstruct it.

Sulfur Mustard (HD) is an example of a chemical agent produced solely for warfare that acts on the central airway if inhaled. HD will cause tissue to slough off in large sheets, known as pseudomembranes, which block the airway. The various degrees of airway restriction cause casualties with central airway restriction/obstruction to be unable to breathe, or to sneeze and cough, to have hoarseness when they talk, and to make wheezing noises when they breathe.

PERIPHERALLY-ACTING TICs

Phosgene. Today phosgene is a major industrial chemical used in many manufacturing processes. More importantly, it is released from heating or burning many common chemicals or solvents. Carbon tetrachloride, perchloroethylene (a degreasing compound), methylene chloride (used in paint removal), and many other compounds break down to phosgene with flame or heat. Also, common substances such as foam plastics release phosgene when they burn. A warfighter presenting with shortness of breath in the absence of a chemical attack or other obvious cause should be questioned very carefully about whether he/she has been near any burning substances or chemical vapors that were near

flame or other hot materials (e.g., a heater with open coils).

Perfluoroisobutylene (PFIB) is given off when Teflon® burns at high temperatures, such as in a vehicle fire. Teflon® is used to line the interior of many military vehicles, particularly armored vehicles and aircraft. Closed-space fires in these vehicles release PFIB. Survivors of vehicle fires who are short of breath should be questioned carefully regarding their exposure to the smoke.

Oxides of Nitrogen, or NOx, are components of photochemical smog that can be produced by the burning of gunpowder or the burning of industrial waste. These substances can build up to high concentrations where artillery is fired and there is inadequate ventilation. Warfighters who become short of breath after heavy firing should be suspected of exposure to this lung-damaging agent.

HC Smoke is a mixture of equal amounts of hexachloroethane, zinc oxide, and approximately 7% grained aluminum or aluminum powder used in the military for obscuration. The zinc oxide can cause lung damage if inhaled in toxic amounts. Appropriate precautions, such as the wearing of protective masks, must be taken when HC smoke is used.

TICs THAT ACT BOTH CENTRALLY AND PERIPHERALLY

Chlorine: This is a good example of a combination agent, one that acts on both airway compartments in low doses. It is widely used in industry for the manufacture of plastics, lubricants, and to purify water. It was the first chemical agent used effectively on the WWI battlefield against unprotected military troops. Its effectiveness as a weapon was greatly reduced once protective masks were widely available for wear on the battlefield. Chlorine turns to hydrochloric acid when it contacts the moisture of the airway; it then causes chemical burns to the tissue. It produces signs and symptoms seen with exposure to both central and peripherally- acting agents. Its action is a reminder that even though central damage may seem like the primary concern in some patients (e.g., they are coughing and wheezing), the medic must always treat the casualty as if they could develop peripheral symptoms and take seriously any patient complaints about feeling chest tightness or having breathing difficulty.

Site of Action

Agent	Central	Peripheral
CG	Large dose	Yes
NOx	Large dose	Yes
HC Smoke	Large dose	Yes
Ammonia	Yes	Large dose
Cl	Yes	Yes
PFIB	Large dose	Yes

TOXICODYNAMICS
MECHANISM OF ACTION

Central Airway. Centrally-acting TICs like ammonia and HD, will form strong acids or bases (alkali) with the water in the tissues of the central airway and then destroy these tissues. Damaged tissues will swell and can slough into the airway and restrict breathing.

Peripheral Airway. Phosgene is the most studied peripheral agent. It causes pulmonary edema, which is life threatening. Less is known about the other compounds; however, it is believed that they are very similar.

Phosgene causes effects in the lung by inhalation only. It does not cause lung effects when absorbed through the skin, injected, or orally ingested.

When inhaled, phosgene travels to the very end of the smallest airways, the bronchioles, and causes damage to these airways. Additionally, it causes damage to the thin membrane that separates the smallest blood vessels (the capillaries) and the air sacs (the alveoli). Phosgene reacts with the proteins and enzymes in these alveolar-capillary membranes to cause damage to the membranes. These membranes usually function to separate the blood in the capillaries from the air in the alveoli, but when the membranes are damaged, they cannot do this. Blood, or at least the liquid part of the blood, the plasma, can leak through the damaged membrane into the alveoli. When the plasma leaks into the alveoli, the air sacs become full of fluid, and air cannot enter them. Therefore, exchange of oxygen from the air into the blood is hindered, and the casualty suffers oxygen deprivation. The extent of the lack of oxygen depends on the extent of the phosgene exposure and the number of alveoli filled with plasma. This is similar to what happens with drowning, in that the alveoli fill up with fluid; however, in this instance, it is fluid from the blood, not from an external source. For this

reason, phosgene poisoning is sometimes referred to as “dry land drowning.”

CLINICAL EFFECTS

Centrally-acting agents: Immediately or shortly after exposure to these gasses or vapors, the individual can develop laryngospasm, though this is not true in all exposures. As the airways are irritated and damaged the individual will sneeze and have pain in the nose (nasopharynx inflammation); can develop painful swallowing (oropharynx inflammation); hoarseness, a feeling of choking and noise with exhalation (larynx inflammation); pain in the chest, coughing, and wheezing during breathing (trachea and bronchi inflammation). This can progress to peripheral effects if the exposure is great enough where the TIC has reached the peripheral airway. Scarring of the central airway can create permanent airway narrowing depending on the agent involved and the dose received.

Peripherally-acting agents: Very shortly after exposure to phosgene or other agents affecting the peripheral airway, the casualty may typically have an asymptomatic period of 30 minutes to 72 hours, but most significant exposures have a latent period less than 24 hours. The duration and concentration of the exposure will determine the time to symptom

onset. The casualty may notice irritation of the eyes, nose, and throat, but more commonly, there may be no effects during or immediately after exposure. The **major effects** from phosgene exposure and other peripherally-acting agents **do not occur until hours later. HD signs are also delayed, but the damage is more in the central compartment.**

The casualty with peripheral damage who is developing pulmonary edema will notice shortness of breath between 2 and 24 hours after exposure. Initially, this may be mild, and the eventual severity of the shortness of breath (dyspnea) will depend on the amount of exposure. As the damage progresses, the dyspnea will become more severe, and soon a cough will develop. If the damage is severe, the casualty will start coughing up clear, foamy sputum, the plasma from his blood that has leaked into his alveoli.

A casualty with a very mild exposure to phosgene (or another of these compounds) will develop dyspnea 6 to 24 hours after exposure. He will notice it first after heavy exertion; however, later he will become short of breath after any activity. With proper care, he will do well and recover completely.

A casualty with a severe exposure to phosgene (or another of these compounds) will

notice shortness of breath within **4 to 6 hours** after exposure. Increased difficulty to breathe, even at rest, will occur, and even with intensive pulmonary care, the casualty may not survive.

The average casualty from a lung-damaging agent will be in between these two extreme cases. When the onset of dyspnea is greater than 6 hours after exposure, there may be progression to dyspnea at rest. However, with good pulmonary care beginning early after the onset of effects, the individual should recover completely.

DIFFERENTIAL DIAGNOSIS

Many TICs can be distinguished by their odor; they generally irritate the mucous membranes and can lead to dyspnea, and *pulmonary edema of delayed onset*.

Riot-control agents produce a burning sensation predominantly in the eyes and upper airways. This irritation is typically more intense than that caused by TICs and is unaccompanied by the distinctive odor.

Nerve agents induce the production of watery secretions as well as respiratory distress; however, their other characteristic effects distinguish nerve agent toxicity from TIC inhalation injury.

The respiratory toxicity associated with vesicants is usually delayed but predominantly affects the central, rather than the peripheral, airways. Vesicant inhalation severe enough to cause dyspnea typically causes signs of airway necrosis, often with pseudomembrane formation and partial or complete upper airway obstruction. Finally, pulmonary parenchymal damage following vesicant exposure usually manifests itself as hemorrhage rather than pulmonary edema.

LABORATORY FINDINGS

No commonly available laboratory tests exist for the specific identification or quantification of exposure to lung-damaging agents. However, an increase in the hematocrit may reflect the hemoconcentration induced by transudation of fluid into the pulmonary parenchyma from the peripherally-acting agents. Arterial blood gases may show a low PaO_2 or $PaCO_2$, which is an early, nonspecific warning of increased interstitial fluid in the lung.

Peak expiratory flow rate may decrease early after a massive exposure to peripherally-acting agents. This nonspecific test helps to assess the degree of airway damage and the effect of bronchodilator therapy. Decreased lung compliance and carbon monoxide diffusing

capacity are particularly sensitive indicators of interstitial fluid volume in the lung but are complex tests for hospital use only.

With peripherally-acting agents, early findings on chest x-ray are hyperinflation, followed later by pulmonary edema without cardiovascular changes of redistribution or cardiomegaly. Ventilation profusion ratio (V/Q) scanning is very sensitive but is nonspecific and for hospital use only.

MEDICAL MANAGEMENT

Terminate exposure as a vital first measure. This may be accomplished by physically removing the casualty from the contaminated environment or by isolating him from surrounding contamination by supplying a properly fitting mask. Decontamination of any liquid agent on skin, and removal of clothing if vapors are trapped there, fully terminates exposure from that source.

Execute the ABCs of resuscitation as required. Establishing an airway is especially crucial in a patient exhibiting hoarseness or stridor; such individuals may face impending laryngeal spasm and require intubation. Establishing a clear airway also aids in interpretation of auscultatory findings. Steps to minimize the work of breathing must be taken.

Because of the always present danger of hypotension induced by pulmonary edema or positive airway pressure, accurate determination of the casualty's circulatory status is vital not just initially, but also at regularly repeated intervals and whenever indicated by the clinical situation.

Enforce rest. Even minimal physical exertion may shorten the clinical latent period and increase the severity of respiratory symptoms and signs in a lung-damaging agent casualty. Physical activity in a symptomatic patient may precipitate acute clinical deterioration and even death. Strict limitation of activity (i.e., forced bed rest) and litter evacuation are mandatory for patients suspected of having inhaled any of the edematogenic agents. This is true whether or not the patient has respiratory symptoms and whether or not objective evidence of pulmonary edema is present.

Prepare to manage airway secretions and prevent/treat bronchospasm. Unless superinfection is present, secretions present in the airways of lung- damaging agent casualties are usually copious and watery. They may serve as an index to the degree of pulmonary edema and do not require specific therapy apart from suctioning and drainage. Antibiotics should be reserved for those patients with an infectious process documented by sputum gram staining

and culture. Bronchospasm may occur in individuals with reactive airways, and these patients should receive theophylline or beta-adrenergic bronchodilators. Steroid therapy is also indicated for bronchospasm as long as parenteral administration is chosen over topical therapy, which may result in inadequate distribution to damaged airways. Methylprednisolone, 700-1000 mg or its equivalent, may be given intravenously in divided doses during the first day and then tapered during the duration of the clinical illness. The increased susceptibility to bacterial infection during steroid therapy mandates careful surveillance of the patient. There is some support in the literature for steroid use in those exposed to HC smoke (zinc/zinc oxide) and oxides of nitrogen, as it is theorized that these can reduce autoimmune reactions that can foster scar development and subsequent bronchiolitis obliterans. The literature does not give strong support for the use of steroids in the treatment of other toxic inhalants. Thus, steroids are not recommended in individuals without evidence of overt or latent reactive airway disease.

Prevent/treat pulmonary edema. Positive airway pressure provides some control over the clinical complications of pulmonary edema. Early use of a positive pressure mask may be beneficial. Positive airway pressure may exacerbate hypotension by decreasing thoracic venous return, necessitating intravenous fluid administration and perhaps judicious use of the pneumatic anti-shock garment.

Prevent/treat hypoxia. Oxygen therapy is definitely indicated and may require supplemental positive airway pressure administered via one of several available devices for generating intermittent or continuous positive pressure. Intubation, with or without ventilatory assistance, may be required, and positive pressure may need to be applied during at least the end-expiratory phase of the ventilator cycle.

Prevent/treat hypotension. Sequestration of plasma-derived fluid in the lungs may cause hypotension that may be exacerbated by positive airway pressure. Urgent intravenous administration of either crystalloid or colloid (which in this situation appears equally effective) may need to be supplemented by the judicious application of the pneumatic anti-shock garment. The use of vasopressors is a temporary measure until fluids can be replaced.

TRIAGE

Patients seen within 12 hours of exposure. A patient with pulmonary edema only is classified ***immediate*** if intensive pulmonary care is immediately available. In general, a shorter latent period portends a more serious illness. A ***delayed*** patient is dyspneic without objective signs and should be observed closely and retriaged hourly. An asymptomatic patient with known exposure should be classified *minimal* and observed and retriaged every two hours. If this patient remains asymptomatic 24 hours after exposure, discharge the patient. If exposure is doubtful and the patient remains asymptomatic 12 hours following putative exposure, consider discharge. An ***expectant*** patient presents with pulmonary edema, cyanosis, *and* hypotension. A casualty who presents with these signs within six hours of exposure generally will not survive; a casualty with the onset of these signs six hours or longer after exposure may survive with immediate, intensive medical care. If ventilatory support is not available, but adequate evacuation assets are available, these patients should have priority for urgent evacuation to a facility where adequate ventilatory support is available.

Patients seen more than 12 hours after exposure. A patient with pulmonary edema is classified ***immediate*** provided he will receive

intensive care within several hours. If cyanosis and hypotension are also present, triage the patient as ***expectant***. A ***delayed*** patient is dyspneic and should be observed closely and retriaged every two hours. If the patient is recovering, discharge him 24 hours after exposure. An asymptomatic patient or patient with resolving dyspnea is classified ***minimal*****.** If the patient is asymptomatic 24 hours after exposure, discharge him. A patient with persistent hypotension despite intensive medical care is ***expectant*****.**

RETURN TO DUTY

If the patient has only eye or upper airway irritation and is asymptomatic with normal physical examination 12 hours later, he may be returned to duty. If the patient's original complaint was dyspnea only, yet physical examination, chest x-ray, and arterial blood gases are all normal at 24 hours, he may be returned to duty. If the patient presented initially with symptoms and an abnormal physical examination, chest x-ray, or arterial blood gas, he requires close supervision but can be returned to duty at 48 hours if physical examination, chest x-ray, and arterial blood gases are all normal at that time.

CYANIDE

AC, CK

SUMMARY

Signs and Symptoms: few. After exposure to high concentrations, seizures, respiratory and cardiac arrest.

Field Detection: The M256A1 Sampler-detector, M18A2, and M90 Chemical agent detectors detect Hydrogen Cyanide (AC) as vapor or gas in the air, and the M272 chemical water testing kit detects AC in water.

Decontamination: Skin decontamination is usually not necessary because the agents are highly volatile. Wet, contaminated clothing should be removed and the underlying skin decontaminated with water or other standard decontaminants to prevent off-gassing as a hazard.

Management: Antidote: intravenous (IV) sodium nitrite and sodium thiosulfate. ***Supportive:*** oxygen, correct acidosis.

OVERVIEW

Cyanide is a rapidly acting, lethal agent that is limited in its military usefulness by its high LCt_{50} and high volatility. Death occurs around eight minutes after inhalation of a high Ct. Sodium nitrite and sodium thiosulfate are effective antidotes.

HISTORY/MILITARY RELEVANCE

The French used cyanide in WWI without notable military success, possibly because of the insufficient amounts delivered and the nature of the chemical. After WWI, the Nazis in Germany used cyanide in the extermination of death camp prisoners. Zyklon B was used in enclosed chambers. Nazi leaders themselves chose to commit suicide using cyanide The U.S. maintained a small number of cyanide munitions during WWII. Japan allegedly used cyanide against China before and during WWII, and Iraq may have used cyanide against the Kurds in the 1980s. In 1978 cyanide contamination of Tylenol led to the production of tamper-proof bottle caps and caplets. The Jim Jones cult staged a final mass suicide in Guyana in 1982, where over 900 followers drank a cyanide-laced beverage. In 1995, the Aum Shinrikyo used cyanide in train station restrooms with poor success. Today there are rebel cults like the Tamils of Sri Lanka

who wear cyanide capsules to take in the event they are captured.

Terms. The term cyanide refers to the anion CN^-, or to its acidic form, hydrocyanic acid (HCN). Cyanogen (C_2N_2) is formed by the oxidation of cyanide ions; however, the term cyanogen has also come to refer to a substance that forms cyanide upon metabolism and produces the biological effects of free cyanide. Simple **cyanide** (HCN, NaCN) is a compound that dissociates to the cyanide anion (CN^-) and a cation (H, Na^+). A **nitrile** is an organic compound that contains cyanide. A **cyanogen** usually refers to a nitrile that liberates the cyanide anion during metabolism and produces the biological effects of the cyanide anion. Cyanogens may be simple (cyanogen chloride) or complex (sodium nitroprusside).

Cyanides are also called "blood agents," an antiquated term still used by many in the military. At the time of the introduction of cyanide in WWI, the other chemical agents in use caused mainly local effect. In contrast, inhaled cyanide produces systemic effects and was thought to be carried in the blood, hence, the term "blood agent." The widespread distribution of absorbed nerve agents and vesicants via the blood invalidates this term as a specific designator for cyanide. Also, the use of "blood agent" carries the connotation that the main site

of action of cyanide is in the blood, whereas cyanide actually acts primarily outside the bloodstream.

Materials of interest as chemical agents are the cyanide **hydrogen cyanide (hydrocyanic acid, AC)** and the simple cyanogen, **cyanogen chloride (CK)**. Cyanogen bromide was used briefly in WWI but is of no present interest.

Sources other than military. The cyanide ion is ubiquitous in nearly all living organisms that tolerate and even require the ion in low concentrations. The fruits and seeds (especially pits) of many plants of the Roseacae family, such as cherries and peaches, as well as almonds and lima beans contain cyanogens capable of releasing free cyanide following enzymatic degradation. The edible portion (the roots) of the cassava plant (widely used as a food staple in many parts of the world) contains the cyanogenic glucoside linamarin. The combustion of any material containing carbon and nitrogen has the potential to form cyanide; some plastics (particularly acrylonitriles) predictably release clinically significant amounts when burned. Industrial concerns in the U.S. manufacture over 300,000 tons of hydrogen cyanide annually. Cyanides find widespread use in chemical syntheses, electroplating, mineral extraction, dyeing, printing, photography,

agriculture, and in the manufacture of paper, textiles, and plastics.

PHYSIOCHEMICAL CHARACTERISTICS

The cyanides exist as liquids in munitions but rapidly vaporize upon detonation of the munitions. The major threat is from the vapor. The liquid toxicity is approximately that of mustard (see Toxicity, below).

The preferred way to deliver cyanide on the battlefield is by large munitions (bombs, large shells), because smaller weapons will not provide the concentrations needed for effects. In reality, the low efficiency of cyanide on the tactical battlefield over time has led to its disuse in combat operations.

DETECTION AND PROTECTION

The immediately-dangerous-to-life-and-health (IDLH) concentration of hydrogen cyanide (AC) is 50.0 parts per million (ppm); that for cyanogen chloride (CK) is 0.6 mg/m^3. The military detectors are capable of detecting AC and CK at the threshold limits given.

DETECTOR	AC (Hydrocyanic Acid)	CK (Cyanogen Chloride)
M256A1	7.0 mg/m^3	
M272 (in water)	20.0 mg/m^3	
M18A2	8.0 mg/m^3	
M90	30 mg/m^3	
M93A1 Fox		46 mg/m^3

Because the odor of cyanide may be faint or lost after accommodation, olfactory detection of the odor of bitter almonds is not a reliable indicator of cyanide exposure, even for those who possess the gene required to smell cyanide. The activated charcoal in the canister of the chemical protective mask adsorbs cyanide, and the mask affords full protection from this gas in an open field environment.

MECHANISM OF TOXICITY

Cyanide salts in solid form or in solution are readily absorbed from the gastrointestinal (GI) tract when ingested. Moreover, the lower the pH in the stomach, the more hydrogen cyanide is released as gas from ingested salts. Liquid cyanide and cyanide in solution can be absorbed even through intact skin, but this route

of entry is usually not clinically significant. Parenteral absorption of liquid cyanide can also occur from wounds. Cyanide is readily absorbed through the eyes, but the most important route of entry in a battlefield or terrorist scenario would likely be by inhalation. Following absorption, cyanide is quickly and widely distributed to all organs and tissues of the body. Ingestion leads to particularly high levels in the liver when compared with inhalation exposure, but both routes lead to high concentrations in plasma and erythrocytes and in the heart, lungs, and brain.

An example of the ability of cyanide to react with metals in the body is its reaction with the cobalt in hydroxycobalamin (vitamin B_{12a}) to form cyanocobalamin (vitamin B_{12}). The reactions of cyanide with metals are reversible and exhibit concentration-dependent equilibria, but the reactions of cyanide with sulfur-containing compounds are catalyzed by the enzyme rhodanese and are essentially one-way and irreversible. The rate-limiting factor in the rhodanese-mediated reactions is usually the availability of sulfur donors in the body. These reactions can be accelerated therapeutically by providing a sulfane such as sodium thiosulfate. The reaction products, thiocyanates and sulfites, are significantly less toxic than cyanide itself and are eliminated in the urine. Cyanide also reacts with carbonyl and sulfhydryl groups (directly or via 3-MPST and other enzymes). However, the

two most important kinds of reactions from the perspective of understanding the classical mechanism of action of cyanide and its response to specific antidotal therapy are the reactions with metals and the enzyme-catalyzed reactions with sulfur-containing compounds.

Cyanide is eliminated unchanged from the body in breath, sweat, and urine--as sodium thiocyanate in the urine, and as iminothiocarboxylic acid (ITCA) from reaction with sulfhydryl groups. High concentrations of cyanide in the body will also lead to measurable increases in urinary elimination of cyanocobalamin (vitamin B_{12}).

TOXICITY

The **LCt_{50}s** of **AC** and **CK** by inhalation have been estimated to be **2500-5000 mg-min/m^3** for **AC** and about **11,000 mg-min/m^3** for **CK. LD_{50}s** for hydrogen cyanide have been estimated to be 1.1 mg/kg for IV administration and 100 mg/kg after skin exposure. The **oral LD_{50}s** for sodium and potassium cyanide are about 100 and 200 mg/kg, respectively.

Cyanide is unique among military chemical agents because it is detoxified at a rate that is of practical importance, about 17 µg/kg·min. As a result, the LCt_{50} is greater for a long exposure

(e.g., 60 minutes) than for a short exposure (e.g., 2 minutes).

MECHANISM OF ACTION

Cyanide has a high affinity for certain sulfur and for certain metallic complexes, particularly those containing cobalt and the trivalent form of iron (Fe^{3+}). The cyanide ion can rapidly combine with iron in cytochrome a_3 (a component of the cytochrome aa_3 or cytochrome oxidase complex in mitochondria) to inhibit this enzyme, thus preventing intracellular oxygen utilization. The cell then utilizes anaerobic metabolism, creating excess lactic acid and a metabolic acidosis. Cyanide also has a high affinity for the ferric iron in methemoglobin, and one therapeutic stratagem induces the formation of methemoglobin to which cyanide preferentially binds.

The small quantity of cyanide always present in human tissues is metabolized at the approximate rate of 17 µg/kg·min, primarily by the hepatic enzyme rhodanese, which catalyzes the irreversible reaction of cyanide and a sulfane to produce thiocyanate, a relatively nontoxic compound excreted in the urine. (An elevated concentration of thiocyanate in either blood or urine is evidence of cyanide exposure.) The limiting factor under normal conditions is the availability of a sulfane as a substrate for

rhodanese, and sulfur is administered therapeutically as sodium thiosulfate to accelerate this reaction. The lethal dose of cyanide is time dependent because of the ability of the body to detoxify small amounts of cyanide via the rhodanese-catalyzed reaction with sulfane. A given amount of cyanide absorbed slowly may cause no biological effects even though the same amount administered over a very short period of time may be lethal. In contrast, the LCt_{50} of each of the other chemical agents, which are not metabolized to the same extent as is cyanide, is relatively constant over time. A lethal amount causes death whether administered within minutes or over several hours.

CLINICAL EFFECTS

The organs most susceptible to cyanide are the central nervous system (CNS) and the heart. Most clinical effects are of CNS origin and are nonspecific.

Approximately 15 seconds after inhalation of a high concentration of cyanide, there is a transient hyperpnea, followed within 15 to 30 seconds by the onset of convulsions. Respiratory activity stops two to three minutes later, and cardiac activity ceases several minutes later still, or approximately six to eight minutes after exposure.

The onset and progression of signs and symptoms after ingestion of cyanide or after inhalation of a lower concentration of vapor are slower. The first effects may not occur until several minutes after exposure, and the time course of these effects depends on the amount absorbed and the rate of absorption. The initial transient hyperpnea may be followed by feelings of anxiety or apprehension, agitation, vertigo, a feeling of weakness, nausea with or without vomiting, and muscular trembling. Later, consciousness is lost, respiration decreases in rate and depth, and convulsions, apnea, and cardiac dysrhythmias and standstill follow. Because this cascade of events is prolonged, diagnosis and successful treatment are possible.

The effects of cyanogen chloride include those described for hydrogen cyanide. Cyanogen chloride is also similar to the riot-control agents in causing irritation to the eyes, nose, and airways, as well as marked lacrimation, rhinorrhea, and bronchosecretions.

Physical Findings. Physical findings are few and nonspecific. The two that are said to be characteristic are in fact not always observed. The first is severe respiratory distress in an acyanotic individual. The natural complexion of the casualty may mask any “cherry-red” skin. When seen, "cherry-red" skin suggests either

circulating carboxyhemoglobin from carbon monoxide poisoning or high venous oxygen content from failure of extraction of oxygen by tissues poisoned by cyanide or hydrogen sulfide. However, cyanide victims may have normal appearing skin and may even be cyanotic, although cyanosis is not classically associated with cyanide poisoning.

Table. Cyanide (AC and CK): Effects from Vapor Exposure

Moderate, from low concen-tration	Transient increase in rate and depth of breathing, dizziness, nausea, vomiting, headache.	These may progress to severe effects if exposure continues.	The time of onset of these effects depends on the concentrati on but is often within minutes after onset of exposure.
Severe, from high concen-tration	Transient increase in rate and depth of breathing - 15 seconds. Convulsions - 30 seconds. Cessation of respiration- 2 to 4 minutes. Cessation of heartbeat - 4 to 8 minutes.		

In addition to the preceding effects, CK causes intense irritation of the eyes, nose, and airways.

The second classic sign of cyanide poisoning is the odor of bitter almonds; however, approximately 50% of the population is genetically unable to detect the odor of cyanide.

The casualty may be diaphoretic with normal sized or large pupils. A declining blood pressure and tachycardia follow an initial hypertension and compensatory bradycardia. Terminal hypotension is accompanied by bradyrhythmias before asystole.

TIME COURSE OF EFFECTS

Effects begin 15 seconds following inhalation of a lethal Ct; death ensues in about 6 to 8 minutes. The onset of effects following inhalation of lower Cts may be as early as minutes after the onset of exposure. After exposure is terminated by evacuation to fresh air or by masking, there is little danger of delayed onset of effects.

The time course for ingested cyanide is longer with the victim initially complaining of stomach upset, due to the alkaline nature of potassium cyanide. This is followed after a period of approximately 7 minutes by hyperpnoea, a feeling of anxiety in the patient, and within 15 minutes, the patient feels weakness and experiences a loss of

consciousness. Convulsions follow. Within 25 minutes apnea occurs, and soon the heart stops and death occurs. Typically, death occurs within 30 minutes after cyanide ingestion, depending on the dose ingested and the physiological make-up of the victim.

DIFFERENTIAL DIAGNOSIS

Inhalation exposure to either cyanide or a nerve agent may precipitate the sudden onset of loss of consciousness, followed by convulsions and apnea. The cyanide casualty has normal sized or dilated pupils, few secretions, and muscular twitching, but no fasciculations. In contrast, the nerve agent casualty has miosis (until shortly before death), copious oral and nasal secretions, and muscular fasciculations. In addition, the nerve agent casualty may be cyanotic, whereas the cyanide casualty usually is not.

LABORATORY FINDINGS

There are several laboratory tests that can be requested, but antidotal treatment must be provided immediately if signs and symptoms are clearly indicative of cyanide poisoning since the rapid timeline will not allow the provider to wait for confirmatory lab results without compromising the patient.

1. An elevated blood cyanide concentration. Mild effects may be apparent at concentrations of 0.5 to 1.0 µg/ml, and concentrations of 2.5 µg/ml and higher are associated with coma, convulsions, and death and are used primarily for forensic confirmation.

2. Acidosis. Metabolic acidosis with a high concentration of lactic acid (lactic acidosis) or a metabolic acidosis with an unusually high anion gap (if the means to measure lactic acid are not available) may be present. Because oxygen cannot be utilized, anaerobic metabolism with the production of lactic acid replaces aerobic metabolism. Lactic acidosis, however, may reflect other disease states and is not specific for cyanide poisoning. Test results are fairly rapid and valuable as an early confirmatory lab result.

3. Oxygen content of venous blood greater than normal. This also is a result of poisoning of the cellular respiratory chain and the resulting failure of cells to extract oxygen from arterial blood. This finding is also not specific for cyanide poisoning. It is helpful in confirming a diagnosis and for evaluating methemoglobin levels for later adjusting the levels of methemoglobin-forming antidotes.

MEDICAL MANAGEMENT

Management of cyanide poisoning begins with self protection, then removal of the casualty to fresh air. Dermal decontamination is unnecessary if exposure has been only to vapor. With liquid exposure, wet clothing should be removed, and the underlying skin should be washed either with soap and water or water alone if liquid on the skin is a possibility. A casualty that has ingested cyanide does not require decontamination.

Attention to the basics of intensive cardio-respiratory supportive care is critical and includes mechanical ventilation as needed, circulatory support with crystalloids and vasopressors, correction of metabolic acidosis with IV sodium bicarbonate, and seizure control with benzodiazepine administration. The fact that cyanide inhibits cellular utilization of oxygen would lead to the expectation that supplemental oxygen would not be of use in cyanide poisoning. However, in fact, administration of 100% oxygen has been found empirically to exert a beneficial effect and should be a part of general supportive care for the cyanide casualty.

Gavage and administer activated charcoal if the cyanide was ingested. All vomitous should be collected so that vapors from it do not sicken the staff.

Symptomatic patients, especially those with severe manifestations, may further benefit from specific antidotal therapy. This is provided in a two-step process. First, a methemoglobin-forming agent such as amyl nitrite (available in civilian antidote kits, but not in military kits, as crushable ampoules for inhalation) or sodium nitrite (for IV use) is administered, since the ferric ion (Fe^{3+}) in methemoglobin has an even higher affinity for cyanide than does cytochrome a_3. The equilibrium of this reaction causes dissociation of bound cyanide from cytochrome a_3 and frees the enzyme to help produce adenosine triphosphate (ATP) again. Hypotension, produced by nitrite administration, should be monitored. Further, a prudent concern for overproduction of methemoglobin must be realized as this may compromise oxygen-carrying capacity. Thus, nitrite is relatively contraindicated in, for example, smoke-inhalation victims. The initial adult dose, equivalent to 1 of the 2 sodium nitrite vials in the standard Cyanide Antidote Kit, is 10 ml. Pediatric nitrite dosing (in the case of a military response to a civilian terrorist incident) is dependent on body weight and hemoglobin concentration. The recommended pediatric dose, assuming a hemoglobin concentration of 12 g/dl, is 0.33 ml/kg of the standard 3% solution given slowly, IV, over 5 to 10 minutes.

The second step for treatment is the infusion of a sulfur donor, typically sodium thiosulfate, which is utilized as a substrate by rhodanese for its conversion of cyanide to thiocyanate. Sodium thiosulfate itself is efficacious, relatively benign, and also synergistic with oxygen administration and thus may be used without nitrites empirically in situations such as smoke inhalation with high carboxyhemoglobin levels. In the cyanide antidote kit, the antidotes are already prepared in premeasured vials. The initial adult dose, equivalent to 1 of the 2 large bottles in the Cyanide Antidote Kit, is 50 ml; the initial thiosulfate dose for pediatric patients is 1.65 ml/kg of the standard 25% solution, IV. Second treatments with each of the two antidotes may be given at up to half the original dose if needed. Directions are also found on the inside of the kit lid.

It is important to realize that, although the combination of sodium nitrite and sodium thiosulfate may save victims exposed to 10 to 20 lethal doses of cyanide and are effective even after breathing has stopped, many patients will recover even without specific antidotal treatment if vigorous general supportive care is emphasized. Lack of availability of antidotes is therefore not a reason to consider even apneic cyanide casualties expectant. It is also important to realize that administration of antidotes, especially if not given slowly enough or if given

in extremely large doses, is also associated with morbidity, and even mortality. Antidotes should not be withheld in a patient with suspected cyanide poisoning, but infusion rates should be slow, and the drugs should be titrated to effect. Overdosage should be avoided.

Several alternative therapies and experimental antidotes are used in other NATO countries. Germany uses dimethylaminophenol (DMAP), a rapid methemoglobin former developed for intramuscular (IM) and IV use. However, muscle necrosis at the site of injection occurs, and only the IV route of administration is recommended.

Certain cobalt compounds directly chelate cyanide to reduce its toxicity. Because cobalt compounds do not depend upon the formation of methemoglobin, they may exert their antidotal activity more quickly than do methemoglobin-formers. Great Britain and France use cobalt edetate (Kelocyanor), but clear superiority to the methemoglobin formers has not been demonstrated, and cobalt toxicity is occasionally seen, particularly if the patient has only a mild exposure. The other cobalt compound sometimes used in France is hydroxycobalamin (vitamin B_{12a}), which complexes with cyanide on a molar basis. Clinical trials of this compound are underway in the U.S. All of these have been

found to be most effective when combined with the administration of thiosulfate.

Ongoing research is examining whether slow methemoglobin formers can be used as pretreatment to induce clinically asymptomatic methemoglobinemia in troops at high risk for cyanide exposure.

TRIAGE

An **immediate** casualty is one who presents within minutes of inhalation exposure with convulsions or the recent onset of apnea, but with circulation intact. Immediate antidote administration will be lifesaving.

A **minimal** casualty is one who has inhaled less than a lethal amount and has mild effects. The antidotes may reduce his symptoms but are not lifesaving.

The **delayed** casualty is one recovering from mild effects or successful therapy. Generally, it will be hours before full recovery. Evacuation is not necessary but might be considered until full recovery takes place.

An **expectant** casualty is apneic with circulatory failure (more likely to triage as expectant here if there are limited resources and the casualty has a co-exposure to other

toxicants or serious trauma resulting in anoxic encephalopathy).

Generally, a casualty who has had inhalation exposure and survives long enough to reach medical care will need little treatment. A severely compromised logistical capability to provide adequate respiratory support might be a basis for re-triage to a different category.

RETURN TO DUTY

Full recovery is usually relatively fast after cyanide intoxication. Those with mild to moderate effects from the agent can usually return to duty within hours, and those successfully treated after severe effects can return within a day as long as there is no neurological damage caused by significant hypoxia.

VESICANTS
HD, H, L, CX

OVERVIEW

Sulfur mustard has posed a military threat since its introduction on the battlefield in WWI. Most of this chapter concerns this agent. Unless otherwise noted, the term "mustard" refers to sulfur mustard.

The nitrogen mustards (HN1, HN2, and HN3) were synthesized in the 1930s, but were not produced in large amounts for warfare. Mechlorethamine (HN2, Mustargen) became the prototypical cancer chemotherapeutic compound and remained the standard compound for this purpose for many years.

Lewisite (L) was synthesized during the late stages of WWI but probably has not been used on a battlefield. The Lewisite antidote, British Anti-Lewisite (BAL), finds medicinal use today as a heavy-metal chelator.

Although classified as a vesicant, phosgene oxime (CX) is a corrosive urticant that also has not seen battlefield use.

Lewisite and phosgene oxime pose only minor potential military threats and will be discussed briefly at the end of this chapter.

MUSTARD
HD, H

SUMMARY

Signs and Symptoms: Asymptomatic latent period (hours). Erythema and blisters on the **skin**; irritation, conjunctivitis, corneal opacity, and damage in the **eyes**; mild upper respiratory signs to marked **airway** damage; also gastrointestinal (GI) effects and bone marrow stem cell suppression.

Field Detection: M256A1 Kit, M18A2 chemical agents detector kits; individual Chemical Agents Alarm (ICAM), M90 chemical agents detector, M8 and M9 chemical agent detector papers, M21 Remote Sensing Chemical Agent Alarm (RSCAAL), M93A1 FOX NBC RECONNAISSANCE System, M272 chemical water testing kit, M22 automatic chemical agent (ACADA) detection alarm

Decontamination: 0.5% hypochlorite, M291 kit, and water in large amounts.

Management: Decontamination immediately after exposure is the only way to prevent damage. Supportive care of patients; there is no specific therapy.

OVERVIEW

Vesicant agents, specifically sulfur mustard (H, HD), have been major military threat agents since their introduction in WWI. They constitute both a vapor and a liquid threat to all exposed skin and mucous membranes. Mustard's effects are delayed, appearing hours after exposure. The organs most commonly affected are the skin (with erythema and vesicles), eyes (with mild conjunctivitis to severe eye damage), and airways (with mild irritation of the upper respiratory tract, to severe bronchiolar damage leading to necrosis and hemorrhage of the airway mucosa and musculature). Following exposure to large quantities of mustard, precursor cells of the bone marrow are damaged, leading to pancytopenia and increased susceptibility to infection. The GI tract may be damaged, and there are sometimes CNS signs. There is no specific antidote, and management is symptomatic therapy. Immediate decontamination is the only established way to reduce damage.

HISTORY/MILITARY RELEVANCE

Sulfur mustard was first synthesized in the early 1800s and was first used on the battlefield during WWI by Germany in July 1917. Despite its introduction late in that conflict, mustard produced the most chemical casualties,

although fewer than 5% of the casualties who reached medical treatment facilities died. Italy allegedly used mustard in the 1930s against Abyssinia. Egypt apparently employed mustard in the 1960s against Yemen, and Iraq used mustard in the 1980s against Iran and the Kurds. Most recently, 2005, the Burmese military has allegedly used a substance against the Karenni people of Burma causing many of the clinical symptoms seen in mustard victims. Accidental exposure from old ordinance also occurs frequently with recent events in China in 2003 and Delaware in 2004. Mustard is still considered a major threat agent.

The U.S. manufactured mustard during WWI and WWII. Most of its stockpile has been or is being destroyed.

Nomenclature. Sulfur mustard manufactured by the Levinstein process contains up to 30% impurities (mostly sulfur) and is known as H. Mustard made by a distillation procedure is almost pure and is known as HD (distilled mustard). An early term for the German agent was HS (probably derived from the WWI slang term Hun Stoffe).

PHYSIOCHEMICAL CHARACTERISTICS

Mustard is an oily liquid with a color ranging from light yellow to brown. Its odor is that of

garlic, onion, or mustard (hence its name), but because of accommodation of the sense of smell, odor should not be relied on for detection. Under temperate conditions, mustard evaporates slowly and is primarily a liquid hazard, but its vapor hazard increases with increasing temperature. At 100°F/37.7°C or above, it is a definite vapor hazard. Mustard freezes at 57°F/13.9 °C, and since a solid is difficult to disperse, mustard is often mixed with substances with a lower freezing point, e.g., Lewisite (the mixture is HL), so that the mixture will remain liquid at lower temperatures. The mixture HT also refers to mustard that has been thickened with small quantities of newer thickening agents to make it even more persistent.

DETECTION AND PROTECTION

The immediately-dangerous-to-life-and-health (IDLH) concentration of sulfur mustard (H) is 0.003 mg/m3. The M8A1 automatic chemical agent detector alarm is incapable of detecting mustard. However, liquid mustard turns M8 paper a ketchup red, and M9 paper will turn pink, red, reddish-brown, or purple when exposed to liquid nerve agents or vesicants, but does not specifically identify either the class of agent or the specific agent. The detectors in the following chart have the capacity to detect sulfur mustard (H) at the threshold limits given.

DETECTOR	H
M256A1	3.0 mg/min^3
M272 (in water)	2.0 mg/min^3
M18A2	0.5 mg/min^3
M21	150 mg/min^3
M90	0.2 mg/min^3
M93A1 Fox	0.01 - 1 mcg/l
CAM	0.1 mg/min^3

Because the odor of sulfur mustard may be faint or lost after accommodation, olfactory detection of the odor of mustard, garlic, onions, or horseradish is not a reliable indicator of mustard exposure. The activated charcoal in the canister of the chemical protective mask adsorbs mustard, as does the charcoal in the chemical protective overgarment. The butyl rubber in the chemical protective gloves and boots is impermeable to mustard. Proper wear of the chemical protective mask and the chemical protective ensemble affords full protection against sulfur mustard.

MECHANISM OF TOXICITY

Mustard vapor and liquid readily penetrate thin layers of most fabrics (but not the chemical protective ensemble) to reach underlying skin.

Although mustard dissolves relatively slowly in aqueous solutions such as sweat, the lipophilicity of mustard guarantees effective absorption through even intact skin. Penetration is rapid (1 to 4 mcg/cm^2-min) and is enhanced by moisture, heat, and thin skin. This explains the otherwise baffling observation that WWI mustard burns involved the scrotum in 42% of cases, but the presumably more readily exposed hands in only 4% of cases. Ocular and respiratory routes of entry are also important, as is parenteral absorption in casualties with conventional wounds. Ingestion (enteral absorption) was an important route of entry for mustard in the sailors who jumped into mustard floating on the sea from the exploding, mustard carrying ship, the SS John Harvey, docked at Bari Harbor, Italy during WWII.

Approximately 10% of the amount of mustard that begins to penetrate the skin will bind to the skin as "fixed" (reacted) mustard; the remaining 90% of the dose reaches the circulation and is systemically distributed as "free" (unreacted and hydrolyzed) mustard. Distribution is to almost all organs and tissues including the kidneys, liver, intestines, and lungs; although, because of dilutional effects and reactions of mustard in the bloodstream, clinical effects from systemic distribution are seen only at high doses. After IV administration, mustard disappears from the blood within

seconds to minutes. Because of the rapid fixation of mustard to tissue, the fluid inside the blisters that eventually develop at the sites of skin contact contains no free mustard and does not pose a contamination hazard to health care providers. Mustard participates in a variety of biotransformative (metabolic) reactions in the body. Some of these reactions are catalyzed by enzymes, but most absorbed mustard reacts directly by forming covalent bonds (via alkylation) with DNA, RNA, proteins, components of cell membranes, and other macromolecules in the body. Mustard is eliminated primarily in the urine as by-products of alkylation.

TOXICITY

The LCt_{50} of sulfur mustard dispersed as a vapor is 1500 mg-min/m^3 in an unprotected group and 10,000 mg-min/m^3 in a group with respiratory protection. This demonstrates not only the importance of respiratory protection, but also the fact that sufficient concentrations of vapor and sufficient exposure times render mustard vapor lethal, even in masked individuals. The LD_{50} of liquid mustard on the skin is 100 mg/kg. Thus, administration of 7 g (about a teaspoon) of liquid mustard to each member of a group of individuals weighing 70 kg would be expected to cause the death of half of those exposed. Although 7 g of a liquid applied

evenly to the surface of the skin may cover approximately 20 to 25% of the total body surface area (BSA), the correlation between BSA involvement and deaths from mustard in the field is poor. One plausible reason for this discrepancy is that using BSA figures by themselves ignores the inhalational component of mustard exposure. Another conceivable explanation is that measurement solely of affected BSA neglects factors such as the thickness of coverage, subsequent spread, contact time, and continued exposure. A 10 mcg droplet of sulfur mustard can produce a small vesicle on exposed skin.

TOXICODYNAMICS (MECHANISM OF ACTION)

Absorbed mustard must first dissolve in aqueous solution such as sweat or extracellular fluid. Although mustard molecules dissolve slowly in such solutions, once they dissolve they rapidly (within seconds to a minute or two) rearrange to form extremely reactive cyclic ethylene sulfonium ions that immediately bind to intracellular and extracellular enzymes, proteins, and other cellular components. Mustard has many biological actions, but the exact mechanism by which it produces tissue injury is not known. According to one prominent hypothesis, biological damage from mustard results from DNA alkylation and crosslinking in

rapidly dividing cells, such as basal keratinocytes, mucosal epithelium, and bone marrow precursor cells. This leads to cellular death and inflammatory reaction, and in the skin, protease digestion of anchoring filaments at the epidermal-dermal junction and the formation of blisters.

Mustard also possesses mild cholinergic activity, which may be responsible for effects such as early GI symptoms and miosis.

It should be re-emphasized that mustard reacts with tissue within minutes of entering the body and that blood, tissue, and blister fluid do not contain free mustard, nor do they represent a contamination risk for medical personnel.

CLINICAL EFFECTS

Topical effects of mustard occur in the eye, airways, and skin. Systemically absorbed mustard may produce effects in the bone marrow, GI tract, and CNS. Direct injury to the GI tract may also occur following ingestion of the compound.

Combined data from U.S. forces in WWI and Iranians in the Iraq-Iran conflict suggest equal incidence of eye, airway, and skin involvement (between 80 and 90% for each). However, there were higher incidences of eye and lung damage

in Iranian casualties than in WWI casualties, probably because of the larger amount of evaporation of the agent in the hot climate.

Skin. Erythema is the mildest and earliest form of skin injury after exposure to mustard. It resembles sunburn and is associated with pruritus or burning, stinging pain. Erythema begins to appear in 2 to 48 hours after vapor exposure with time of onset dependent on Ct, ambient temperature and humidity, and skin site exposed. The skin sites most sensitive are the warm, moist locations with thinner skin such as the perineum, external genitalia, axillae, antecubital fossae, and neck.

Within the erythematous areas, small vesicles can develop which may later coalesce to form bullae. The typical bulla, or blister, is large, dome-shaped, thin-walled, translucent, yellowish, and surrounded by erythema. The blister fluid is clear, at first thin and straw-colored, but later yellowish and tending to coagulate. The fluid does not contain mustard and is not a vesicant.

At extremely high doses such as those from liquid exposure, lesions may develop a central zone of coagulation necrosis with blister formation at the periphery. These lesions take longer to heal and are more prone to secondary

infection than the uncomplicated lesions seen at lower exposure levels.

Pulmonary. The primary airway lesion from mustard is necrosis of the mucosa with later damage to the musculature of the airways if the amount of agent is large. The damage begins in the upper airways and descends to the lower airways in a dose-dependent manner. Usually the terminal airways and alveoli are affected only as a terminal event. Pulmonary edema is not usually present unless the damage is very severe, and then it usually is hemorrhagic.

The earliest effects from mustard, perhaps the only effects from a low Ct, involve the nose, sinuses, and pharynx. There may be irritation or burning of the nares, epistaxis, sinus pain or irritation, and irritation or soreness of the pharynx. As the Ct increases, other effects occur, such as laryngitis with voice changes and a nonproductive cough, and damage to the trachea and upper bronchi leads to a cough productive of sputum. Lower airway involvement causes dyspnea and an increasingly severe cough with increased quantities of sputum. Terminally, there may be necrosis of the smaller airways with hemorrhagic edema into surrounding alveoli. This hemorrhagic pulmonary edema is rarely a feature.

Necrosis of the airway mucosa with resulting inflammation can cause pseudomembrane formation. Pseudomembranes may occur from the most proximal parts of the airways to the most distal portions. These membranes may cause local airway obstruction at the sites of formation, and detachment may lead to obstruction of lower airways.

The cause of death in mustard poisoning is commonly respiratory failure. Mechanical obstruction by pseudomembranes and agent-induced laryngospasm are important causes of death in the first 24 hours after exposure. Deaths occurring from the third to the sixth day after exposure result from secondary bacterial pneumonia caused by bacterial invasion of denuded respiratory mucosa and necrotic debris. Agent-induced bone marrow suppression is a contributory factor in later, septic deaths from pneumonia.

Eyes. The eyes are the organs most sensitive to mustard vapor injury. The latent period is shorter for eye injury than for skin injury and is also Ct dependent.

After low-dose vapor exposure, irritation evidenced by reddening of the eyes may be the only effect. As the dose increases, the spectrum of injury includes progressively more severe

conjunctivitis, photophobia, blepharospasm, pain, and corneal damage.

Blisters do not normally form in the eyes. Instead, swelling and loosening of corneal epithelial cells lead to corneal edema and clouding with leukocytes (which affects vision). Corneal vascularization with secondary edema may last for weeks. Scarring between the iris and lens may follow severe effects; this scarring may restrict pupillary movements and may predispose victims to glaucoma.

The most severe damage is caused by liquid mustard from airborne droplets or by self-contamination. After extensive eye exposure, severe corneal damage with possible perforation of the cornea and loss of the eye can occur. Eye loss also results from panophthalmitis if appropriate therapy is not instituted.

During WWI, mild conjunctivitis accounted for 75% of eye injuries, with recovery in 1 to 2 weeks. Moderate conjunctivitis with minimal corneal involvement, blepharospasm, edema of the lids and conjunctivae, and orange-peel roughening of the cornea accounted for 15% of the cases, with recovery in 4 to 6 weeks. Severe corneal involvement accounted for 10% of the cases. Those with permanent corneal damage accounted for less than 1% of cases. About

0.1% of these severe casualties would meet the criteria for legal blindness today.

Miosis noted after mustard exposure in both humans and experimental animals is probably from the cholinomimetic activity of mustard.

Gastrointestinal (GI) tract. The mucosa of the GI tract is very susceptible to mustard damage, either from systemic absorption or ingestion of the agent. However, reports of severe GI effects from mustard poisoning are relatively infrequent.

Mustard exposure, even exposure to a small amount, will often cause nausea, with or without vomiting, lasting 24 hours or less. The nausea and vomiting appear not to be a direct effect of the agent on the GI tract, but rather they are from a stress reaction, a nonspecific reaction to the odor, or cholinergic stimulation by mustard. Further GI symptoms are usually minimal unless the exposure was severe (even then, GI signs are not common) or exposure resulted from ingestion of contaminated food or drink. Diarrhea has been reported; constipation is equally common. Diarrhea (rarely bloody) and vomiting beginning days after a high-dose exposure imply a poor prognosis.

Central nervous system (CNS). The CNS effects of mustard remain poorly defined. Animal

work demonstrated that mustards (particularly the nitrogen mustards) are convulsants, and there are several human case reports describing victims who were exposed to very large amounts and had neurological effects within several hours after exposure just prior to death. Reports from WWI, and again from Iran, described people exposed to small amounts of mustard that appeared sluggish, apathetic, and lethargic. These reports suggest that minor psychological problems could linger for a year or longer.

TIME COURSE OF EFFECTS

Mustard binds irreversibly to tissue within several minutes after contact. If decontamination is not done immediately after exposure, there is no way to prevent injury, although later decontamination might prevent a more severe lesion.

The clinical effects of mustard are delayed. Signs and symptoms may appear as early as 2 hours after a high-dose exposure, whereas following a low-dose vapor exposure, the latent or asymptomatic period may extend to 48 hours. There are several reports of individuals exposed to very large amounts who died within hours; this type of occurrence is extremely rare. The typical onset time is between four and eight hours. The concentration (C) of the mustard vapor, time (t) of exposure, ambient weather,

and body site exposed are factors in the onset time.

It must be emphasized that **mustard causes tissue damage within several minutes after contact without causing any concomitant clinical effects**, e.g., burning or erythema. Because of the lack of immediate effects, the contaminated person is often unaware of the exposure and does not decontaminate. **To prevent injury, decontamination must be done immediately after contact**. Later decontamination may prevent further damage, absorption, or spread of the agent.

Table: Effects of Mustard Vapor

ORGAN	SEVERITY	EFFECTS	ONSET OF FIRST EFFECT
Eye	Mild	Tearing, itchy, burning, gritty feeling	4-12 hours
	Moderate	Above, plus reddening, swelling of lids, moderate pain	3-6 hours
	Severe	Marked swelling of lids, possible cornea damage, severe pain	1-2 hours
Airways	Mild	Runny nose, sneezing, nosebleed, hoarseness, hacking cough	12-24 hours
	Severe	Above, plus severe productive cough, shortness of breath	2-4 hours
Skin	Mild to Severe	Erythema (redness), blisters	2-24 hours

DIFFERENTIAL DIAGNOSIS

Of the three vesicant agents, mustard is the only one that does not cause immediate pain. The casualty is asymptomatic until the lesion becomes apparent hours later.

Lewisite and phosgene oxime, in contrast, cause immediate pain or irritation to the eye, skin, or respiratory tract. This causes sufficient stimulus to decontaminate immediately or to mask.

Isolated small blisters or a small group of blisters suggest possible exposure to mustard, to plants such as poison ivy or poison oak, drugs, or other substances. The physical characteristics of the lesion are not distinctive; therefore, the history of exposure is invaluable.

Although the blisters of mustard and Lewisite are slightly different (there is less erythema around the Lewisite blister), this information is of little value in individual cases.

LABORATORY FINDINGS

Leukocytosis occurs during the first day, and the magnitude of increase in leukocytes during the subsequent days correlates roughly with the amount of tissue injury, primarily to skin or pulmonary tissue. If systemic absorption is large,

leukocytes in the peripheral blood will decrease beginning on day three to day five; this decrease indicates damage to precursor cells in the blood-forming organs. The fall may be precipitate, e.g., a decrease of 5000 to 10,000 cells/day. If the marrow damage is severe, erythrocytes and thrombocytes may later decrease, but the casualty usually recovers or dies before this is apparent. A leukocyte count of 500 or fewer is a sign of an unfavorable prognosis.

Signs of a chemical pneumonitis may appear within the first 2 to 3 days after inhalation exposure. Leukocytosis, fever, and sputum production suggest a bacterial process, but within this time period sputum cultures are usually negative for pathogens. Organisms commonly invade the damaged airway tissue at days 3 to 5. A change in the fever pattern, an increase in leukocytosis, and a change in the character of the sputum in this time period suggest a bacterial process. Sputum Gram Stain and culture should be done for identification of the specific organism.

Damaged skin should be cultured routinely, particularly if there is an increase in the exudate or in the inflammatory reaction.

Although GI bleeding is unusual, declining hematocrit values should prompt serial analyses of stool for occult blood.

Thiodiglycol, a urinary metabolite of sulfur mustard, can be measured by the Army Medical Laboratory (AML) that will be deployed. There is no clinical laboratory test for mustard in blood or tissue, nor is one expected, since mustard is biotransformed and bound to tissues within minutes after absorption. However, ways to measure blood and tissue adducts produced in the body after reaction with sulfur mustard are being studied.

MEDICAL MANAGEMENT

The management of a patient exposed to mustard may be simple, as in the provision of symptomatic care for a sunburn-like erythema, or extremely complex, as in providing total management for a severely ill patient with burns, immunosuppression, and multi-system involvement. Suggested therapeutic measures for each organ system are provided below. Guidelines for general patient care are not intended to take the place of sound clinical judgment, especially in the management of complicated cases.

Skin. Erythema should be treated with calamine or other soothing lotion or cream (e.g., 0.25% camphor and menthol, calamine) to reduce burning and itching. Small blisters (under 1-2 cm) should be left intact, but because larger

ones will eventually break (the blister fluid does not contain mustard), they should be carefully unroofed. Denuded areas should be irrigated three to four times daily with saline, another sterile solution, or soapy water and then liberally covered with a topical antibiotic such as silver sulfadiazine or mafenide acetate to a thickness of 1-2 mm. If an antibiotic cream is not available, sterile petrolatum will be useful. Modified Dakins solution (sodium hypochlorite) was used in WWI and in Iranian casualties for irrigation and as an antiseptic.

Multiple or large areas of vesication suggest the need for hospitalization and whirlpool bath irrigation.

Systemic analgesics should be used liberally, particularly before manipulation of the patient or irrigation of the burn areas. Systemic antipruritics such as trimeprazine should be tried if needed. Monitoring of fluids and electrolytes is important in any sick patient, but it must be recognized that fluid loss is not of the magnitude seen with thermal burns. Clinicians accustomed to treating patients with thermal burns must resist the temptation to over-hydrate a mustard casualty with a similar amount of burned body surface.

Eyes. Conjunctival irritation from a low Ct will respond to any of a number of available

ophthalmic solutions after the eyes are thoroughly irrigated. Regular application of homatropine (or other anticholinergic drug) ophthalmic ointment will reduce or prevent future synechiae formation. A topical antibiotic applied several times a day will reduce the incidence and severity of infection. Vaseline or a similar substance should be applied to the edges of the lids regularly to prevent them from sticking together. This prevents adhesions and later scarring during healing and also permits drainage of any underlying infection or pus. Topical analgesics may be useful initially if blepharospasm is too severe to permit an adequate examination, but topical analgesics should otherwise be avoided, and systemic analgesics should be given for eye pain. Topical steroids are not of proven value, but their use during the first day or two might reduce inflammation. Further use should be relegated to an ophthalmologist. Sunglasses may reduce discomfort from photophobia.

The patient should be constantly reassured that complete healing and restoration of vision will be the outcome.

Pulmonary. Upper airway symptoms (sore throat, nonproductive cough, and hoarseness) may respond to steam inhalation and cough suppressants. Although a productive cough and dyspnea accompanied by fever and leukocytosis

occurring 12 to 24 hours after exposure may suggest a bacterial process to the clinician, he must resist the urge to use antibiotics for this process, which in fact is a sterile bronchitis or pneumonitis. Infection often occurs on about the third day. Its presence is signaled by an increased fever, an increase in the pulmonary infiltrate by x-ray, and an increase in sputum production and a change in sputum character to purulent. Appropriate antibiotic therapy should await confirmation of the clinical impression by positive sputum studies (Gram stain and culture).

Intubation should be performed early, before laryngeal spasm or edema makes it difficult or impossible. Intubation permits better ventilation and facilitates suction of the necrotic and inflammatory debris. Oxygen may be needed, and early use of PEEP or CPAP may be of benefit. If there is a suggestion of pseudomembrane formation, bronchoscopy should be done to permit suctioning of the necrotic debris by direct vision.

Bronchodilators may be of benefit for bronchospasm. If they fail, steroids may be tried. There is little evidence that the routine use of steroids is beneficial. The need for continuous use of assisted or controlled ventilation suggests a poor prognosis.

Death often occurs between the fifth and tenth day after exposure because of pulmonary insufficiency and infection complicated by a compromised immune response from agent-induced bone marrow damage.

Gastrointestinal. Atropine (0.4-0.6 mg, IM or IV), another anticholinergic drug or antiemetic should control the early nausea and vomiting. Prolonged vomiting or voluminous diarrhea beginning days after exposure suggests direct involvement of the GI tract by severe systemic poisoning, a poor prognostic sign.

Bone marrow. Sterilization of the gut by nonabsorbable antibiotics should be considered to reduce the possibility of sepsis from enteric organisms. Cellular replacement (bone marrow transplants or transfusions) may be successful, as intact mustard does not persist beyond the few minutes following absorption and would not damage the new cells.

General. A patient severely ill from mustard poisoning requires the general supportive care provided for any severely ill patient, as well as the specific care given to a burn patient. Liberal use of systemic analgesics and antipruritics, as needed, maintenance of fluid and electrolyte balance, and other supportive measures are necessary. Parenteral food supplements including vitamins may also be helpful.

Other. Sulfur donors such as sodium thiosulfate decreased systemic effects and elevated the LD_{50} when given before exposure or within 20 minutes after exposure in experimental animals. Activated charcoal given orally to casualties was of no value. Hemodialysis was not only ineffective, but actually harmful in several casualties. The rapid biotransformation of the mustard molecule suggests that none of these measures would be beneficial hours or days after exposure.

TRIAGE

Most mustard casualties will be triaged as **delayed**. Those with skin lesions covering several percent to 50% of the BSA will require further medical care but do not need immediate lifesaving assistance. (In contrast, patients with thermal burns covering 20 to 70% of their BSA are considered immediate because of their fluid requirements.) Those with mild to moderate pulmonary effects will also eventually require further care but are not in the immediate category for triage. Eye injuries from other causes require immediate care, but by the time the mustard eye lesion develops, there is no possibility of reducing the injury. These casualties are also in the delayed category.

Patients with skin lesions covering a small percentage of BSA (under 5%) when the lesions are not in vital areas (a burn on the face might prevent mask donning) are triaged as **minimal**. Clinical judgment should dictate whether these patients should be evacuated for care or whether they can return to duty. The tactical situation will also be a factor in the decision. Patients with minor eye injuries to include irritation and reddening can be treated and returned to duty. Those with slight upper respiratory complaints of a hacking cough and an irritated throat that developed 12 hours or longer after exposure might be given symptomatic therapy and returned to duty.

The only mustard casualties that might be triaged as **immediate** are those with moderately severe to severe pulmonary signs and symptoms. Two factors should temper this decision. First, casualties who develop severe pulmonary effects within four to six hours of exposure will probably not survive despite maximal medical care, and it might be better to expend limited medical resources elsewhere. Second, if evacuation to a maximal medical care facility is required, the casualty may survive the lengthy trip, but during the delay, his lesion may progress to an irreversible stage.

A mustard casualty who has severe pulmonary effects that developed within four to

six hours of exposure should be triaged as **expectant**. A casualty who has over 50% BSA burns from mustard liquid might also be categorized as expectant, but this decision would depend on available medical resources at the far rear echelons of medical care. (The LD_{50} for liquid mustard is about 7 grams, or between 1 and 1½ teaspoons of liquid. This amount will cover about 25% BSA, so an individual with a 50% BSA burn could possibly have 2 LD_{50}s on his skin. This person might be saved, but at great expenditure of medical resources.)

RETURN TO DUTY

Casualties with minor skin, eye, or pulmonary injuries might be returned to duty as soon as they are given symptomatic therapy at a medical facility. The range of return to duty times for those with more severe but treatable injuries is from one week to a year or longer.

Those with eye injuries should recover in one to three weeks, except for the low percentage of casualties with severe injuries or complications. Casualties with mild to moderate pulmonary injuries should return to duty in a week to a month. Healing of mild skin lesions will enable the casualty to return within several weeks, but patients with large skin lesions will require hospitalization for many months.

LONG-TERM EFFECTS

Repeated symptomatic exposures to mustard over a period of years (as in manufacturing workers) seem to be well established as a causal factor in an increased incidence of upper airway cancer. However, the association between a single exposure to mustard and airway cancer is not well established. A single, severe exposure to mustard may have contributed to other airway problems, such as chronic bronchitis, based on WWI data. A new complication seen in Iranian casualties from the Iran-Iraq War in the 1980s was late-onset tracheobronchial stenosis, which presumably would have been seen in WWI casualties had antibiotic therapy been available to allow those who died from secondary bacterial pneumonia to survive.

Several eye diseases, such as chronic conjunctivitis and delayed keratitis, may follow a single, severe exposure of the eye to mustard. Skin scarring and pigment changes may follow a severe skin lesion from mustard; cancer sometimes develops in scarred skin.

Mustard is classed as a mutagen and carcinogen, based on laboratory studies. However, there are no data to implicate mustard as a reproductive toxin in man, and there is no

evidence that mustard is a causative factor in nonairway, non-skin cancer in man.

Supplemental Considerations for Treatment and Disposition Written for Iraqi Freedom War Effort

In addition to the treatment modalities listed in the references above, there has been ongoing animal research dealing with the management of mustard injuries and observation of accidents that have occurred with sulfur mustard in the U.S. and abroad. (We do not have major human experience dealing with mustard casualties other than WWI and the Iran/Iraq conflict in the 1980s. It is unethical to perform human mustard injury research.)

A. Eye.

Research at the U.S. Army Medical Research Institute of Chemical Defense (USAMRICD) with rabbits exposed to sulfur mustard showed remarkable results using steroids and antibiotic eye combinations. Eyes that would have been all but destroyed appeared almost normal when these combinations were applied early and frequently. In the study, they were given both by injection and topically in the form of solutions and ointments. These results were so remarkable that it was recommended this past year that

commercially available ophthalmologic steroid/antibiotic solutions or ointments be added to the field medical sets. Recommended use would be as soon as possible in connection with even the mildest mustard eye injury. The frequency of use would be every one to two hours until the full extent of the developing mustard injury became known. The treatment would then be modified accordingly, with consultation and examination by an ophthalmologist at the earliest time possible. This initial treatment would be applied only in the absence of a penetrating injury to the eye or in the case of obvious, secondary bacterial infection. Eye pain could be severe enough to require narcotic analgesia.

Exposure to sulfur mustard can lead to a chronic eye inflammation with associated pain, erosions, and even frank ulceration. This keratitis has been seen to develop as early as 8 months and as late as 20 years after initial exposure. It does not seem to be associated with severity of exposure, although one would suspect a higher incidence with more severe exposures. Mustard-induced chronic keratitis was either infrequent or undetected in the years following WWI.

B. Lung.

No specific antidotes for mustard injury to the lung exist; however, a tremendous amount of supportive care is available for all pulmonary injuries.

Mustard lung injuries in the trachea and bronchi have a high rate of secondary bacterial infection starting as early as three days and developing as late as two to three weeks after exposure. The late development is especially frequent with doses leading to significant bone marrow depression. Prophylactic administration of antibiotics is contraindicated and will lead to the selection of resistant bacterial infections. Vigilant watch for the earliest signs and symptoms of infection with Gram stain and cultures to help choose the most appropriate antibiotic are the watch words.

The sloughing of the necrotic bronchial mucosa, pseudomembranes, either as amorphous debris or as pseudomembranes, can be severe enough to cause mechanical blockage and suffocation. Treatment is rigorous percussion and postural drainage and provision of humidified air with supplemental moisturized air/oxygen. At times, fiberoptic bronchoscopy may be needed to remove the blockage. Warfighters in WWI died from these blockages.

A complication not reported from WWI, but seen in casualties from the Iran/Iraq War in the 1980s, was severe tracheobronchial stenosis, which may be irreversible. Bronchospasm with asthma-like symptoms can be a frequent complication of the mustard lung injury. The medicines used for the bronchospasm are the same as in asthma: beta- adrenergic dilators, steroids, and theophylline-type drugs.

Steroidal anti-inflammatory agents have never been shown scientifically to be beneficial in cases of mustard lung injury. However, if beta adrenergic bronchodilators are not providing complete relief, many physicians would be quick to add steroids to aid in breaking the bronchospasm. Again, caution is warranted because of the likelihood of secondary bacterial infection in cases of exposure to sulfur mustard.

With significant irritation to the larynx, acute closure caused by laryngospasm is possible and could result in death if a patent airway is not maintained.

Pulmonary edema is not a normal feature of the mustard lung injury, except in the case of very large exposures where hemorrhagic pulmonary edema may be seen.

Mounting circumstantial evidence suggests the possibility of chronic bronchial disease developing after significant pulmonary exposure.

Mustard is a proven carcinogen, but no cases of cancer have been documented with acute exposures. However, some factory workers chronically exposed to low doses of sulfur mustard in WWI developed cancers of the respiratory tract (nasopharynx, larynx, and lung).

A small amount of laboratory data in rats and mice exists pointing to reproductive abnormalities. Anecdotal stories are now coming out of Iran and Iraq that will take years to substantiate if good epidemiological studies are performed.

The possibility of a causal link between mustard exposure and late onset or chronic health effects should always be investigated in patients with a documented or suspected history of exposure.

C. Skin.

In general, mustard skin burns are more superficial than thermal burns, but the services of an intensive care unit or surgical burn unit can easily be a necessity. Judicious IV fluid and electrolyte therapy are required with significant mustard skin burns, but fluid requirements are

less than with corresponding thermal burns. Fluids and electrolytes should be closely monitored, as fluids may be lost to edematous areas, with resultant dehydration. Medical personnel are cautioned not to over-hydrate the patient, as hypervolemia and pulmonary edema can be iatrogenically induced in mustard casualties. The exact fluid replacement requirements for cutaneous mustard injuries should be based on patient status and considered on a case-by-case basis.

Vesication may take several days to complete. Mustard blister fluid does NOT contain active sulfur mustard. Once a patient has been adequately decontaminated, medical personnel do not have to fear contamination.

Multiple techniques exist for caring for the mustard skin burns/blisters/wounds: (1) leaving the blisters intact; (2) removing/debriding the roof of large blisters; (3) leaving the blister roof intact with sterile needle aspiration of the fluid; and (4) removing the blister roof with temporary placement of artificial or pig skin. A universal measure is the use of a topical antibiotic cream or ointment whether the blister is intact or not. The topical antibiotic depends on individual experience and preference, starting with traditional surgical preparations and working down to whatever is available. A moist wound-healing environment should be maintained

during the re-epithelialization process for optimal outcome. Initially, the attachment of the neoepidermis to the underlying dermis may be weak, and protective dressings may be needed to avoid or minimize damage as a result of friction with clothing or bedding.

Mustard skin wounds can easily develop a secondary bacterial cellulitis requiring the use of appropriate systemic antibiotics. We have been impressed by the tremendous inflammation caused by sulfur mustard in human skin. To the uneducated eye, this can easily be confused with bacterial cellulitis. Infection surveillance and specialty consultation may be necessary.

Mustard casualties with skin injury may require narcotics for analgesia.

It has long been recognized that mustard skin wounds are slow to heal, taking sometimes twice the time that would be expected with a conventional wound or a thermal burn. The hypothesis explaining these observations is that abnormal compounds of DNA (DNA adducts) are produced, delaying the healing time. Also, the skin histologically very often looks more like scar tissue than normal skin. Recent studies in the U.S. (USAMRICD) and England (Porton Down) have shown that appropriate debridement of the deeper mustard burns leads to more normal healing times and return to

regular skin architecture. Good results were obtained with both laser debridement and traditional mechanical techniques. Accurate depth assessment is important, because it dictates how aggressive treatment needs to be to minimize or prevent cosmetic and functional deficits (e.g., deep injuries will need to be excised and grafted). Microcutaneous blood flow is a good prognostic indicator and should be monitored using laser Doppler perfusion imaging or indocyanine green fluorescence imaging, if available.

Surgeons are encouraged to contact the USAMRICD or the U.S. Army Institute of Surgical Research (ISR).

D. Bone Marrow.

Sulfur mustard, like nitrogen mustard and certain chemotherapeutic compounds, is an alkylating agent. Systemic absorption of sulfur mustard usually above what would be 25% of a lethal dosage can lead to significant bone marrow depression. This is why the systemic effects of sulfur mustard sometimes have been described as radiomimetic. The earliest indicator that a patient may have received a significant systemic exposure is nausea and vomiting persisting longer than the first hour or two after exposure. Nausea and vomiting 24 hours later is definitely a warning sign. The next most

sensitive indicator is a fall in the lymphocyte count; this lymphopenia may occur as early as the first 24 hours. The polymorphonuclear cell count may actually rise in the first 24 hours. Other cellular components of blood may show a significant decline as early as three days after exposure, and patients can be in profound marrow suppression by one to three weeks following exposure. The usual life- threatening complication is sepsis and septic pneumonia. Transfusions, isolation techniques, hormonal stimulation of the marrow and appropriate antibiotics may all be utilized.

Studies in the non-human primates conducted by the Navy using nitrogen mustard and by the Army with sulfur mustard showed an improved bone marrow recovery time using granulocyte colony stimulating factor (GCSF). GCSF is a commercially available product for use in more standard cases of marrow suppression. *Physicians are encouraged to contact the USAMRICD should the need arise.*

E. Gastrointestinal.

Severe hemorrhagic diarrhea may be caused either by direct ingestion of sulfur mustard or by systemic absorption following exposure by other routes. High doses of sulfur mustard can induce a necrosis and sloughing of the gastrointestinal mucosa. The most important

aspect of treatment is IV fluids and electrolytes. Anticholinergics to control bowel spasm and possibly narcotic analgesia are indicated as long as an acute surgical abdomen is not a complication. Hemorrhage could be severe enough to require transfusion.

F. Central Nervous System.

In the first few hours of exposure to sulfur mustard, patients can experience mood swings ranging from depression to euphoria. The mechanism for these mood changes is not understood. Supportive care is indicated.

A few individuals in WWI who received massive exposures to sulfur mustard experienced seizures and died rapidly. This same phenomenon has been observed in animals.

I. Guidelines for Return to Duty

Due to the slow healing properties of sulfur mustard injuries, any casualties with significant injury to the eyes, respiratory tract, skin, GI tract, or CNS will not be returning to duty for weeks to months.

A. Eye.

Only the mildest eye irritations to sulfur mustard, those requiring perhaps only soothing eye drops, will be able to return to duty. The mildest form of conjunctivitis causes a functional blindness from pain, photophobia, and spasm of the eyelid muscles; this conjunctivitis takes an average of two weeks to resolve. As the severity of the injury increases, so does the time for healing. A moderate conjunctivitis may require a full two months before return to duty is possible. In a few rare instances, blindness may result from severe exposures.

B. Lung.

Only those individuals experiencing an irritation without significant tissue injury will be able to return to duty. Determining who has received only an irritation or the mildest of injuries will require observation from three to seven days. Anyone with documented mustard lung injury producing a bronchial pneumonia or pseudomembrane formation will not be able to return to duty for several months. Severe cases may never return to duty.

C. Skin.

Only small percentage surface areas (less than 5%) in non-critical areas will be able to return to duty following treatment with topical antibiotic, dressings, and oral analgesics. Burns to the hands, feet, face, axillae, and groin are all potentially disabling*. (A recent accident victim required hospitalization in a burn center for burns on the left arm and left leg amounting to 6% body surface area. The disability caused by this injury for such a relatively small surface area was striking.)*

Return to duty will require weeks to months in all but the mildest of injuries.

Burns by liquid on the skin and in the eye cause the most severe injury. It is possible in some instances to receive a nearly total body burn with mustard vapor with effects no more severe than those from a second-degree sunburn. A vapor burn of this milder level of severity would take 48 hours or more to develop. However, a vapor burn developing in only a few hours could be as severe as a liquid burn. Severity of a mustard burn is dependent upon the total absorbed dose of vapor and liquid.

II. Guidelines for Medical Evacuation to Either Landstuhl, Germany or CONUS.

A casualty that requires hospital care for longer than two weeks, specialty care not available in theater, or intensive care or burn center-level treatment should be medically evacuated as soon as feasible.

III. Physical Examination, Laboratory, and Procedures at Medical Center.

These recommendations pertain ONLY to patients requiring evacuation to the MEDCEN level of care.

There should be a full, appropriate internal medicine, dermatology, ophthalmology, or burn surgical examination on admission and documentation. Serial evaluations should focus on any abnormalities until these abnormalities have resolved with time and appropriate treatment. Patients with injury involving specific organ systems (for example the eyes, respiratory tract, GI tract, blood, or CNS) should at a minimum receive consultative care by the appropriate specialists.

Return to duty should be delayed until after full recovery. Temporary duties during convalescence should be appropriate to the

patient's condition while awaiting full return to duty or medical retirement.

After full recovery, the patient should have follow-up evaluations every six months with appropriate studies for specific injuries. If problems are found at follow-up visits, appropriate care should be given with return visits as frequently as necessary. Once a patient has had two six-month follow-up visits without problems arising, he or she should start annual follow-up visits for five years. Any associated medical problems will extend the period of close follow-up until complete resolution or maximal medical improvement. Once a patient goes five years without related medical problems, he or she may be discharged to an as-indicated, follow-up status.

All patients should have access to military or veterans' health care for life for effects of exposure to sulfur mustard.

Patients with a mustard eye injury should have ophthalmology evaluations every five years (and more frequently as necessary) for the rest of their lives.

Patients with mustard lung injury to the respiratory tract (including any of the nasopharynx, larynx, trachea, and lung) should

have a pulmonary evaluation as indicated, but at least every five years, for life.

Patients who have recovered from pancytopenia caused by sulfur mustard should have a hematology evaluation as indicated, but at least every five years, for life.

LEWISITE

L

SUMMARY

Signs and Symptoms: Lewisite causes immediate pain or irritation of skin and mucous membranes. Erythema and blisters on the skin and eye and airway damage similar to those seen after mustard exposures develop later.

Field Detection: M256A1 kit, M18A2 chemical agent detector kits; Individual Chemical Agent Alarm (ICAM), M90 chemical agents detector, M8 and M9 chemical agent detector paper, M21 Remote Sensing Chemical Agents Alarm (RSCAAL), M93A1 FOX NBC RECONNAISSANCE System, M272 Chemical water testing kit, M22 Automatic Chemical Agent (ACADA) Detection Alarm.

Decontamination: M291, soap and water, 0.5% bleach solution.

Management: Immediate decontamination; symptomatic management of lesions the same as for mustard lesions; a specific antidote (BAL) will decrease systemic effects.

OVERVIEW

Lewisite is a vesicant that damages the eyes, skin, and airways by direct contact. After absorption, it causes an increase in capillary permeability to produce hypovolemia, shock, and organ damage. Exposure to Lewisite causes immediate pain or irritation, although lesions require hours to become full-blown. Management of a Lewisite casualty is similar to management of a mustard casualty, although a specific antidote, British Anti-Lewisite (BAL, dimercaprol), will alleviate some effects.

HISTORY/MILITARY RELEVANCE

Dr. Wilford Lee Lewis first synthesized Lewisite in 1918, but production was too late for its use in WWI. It has not been used in warfare, although some countries may stockpile it. Lewisite is sometimes mixed with mustard to achieve a lower freezing point of the mixture for ground dispersal and aerial spraying.

PHYSIOCHEMICAL CHARACTERISTICS

Lewisite is an oily, colorless liquid with the odor of geraniums. It is more volatile than mustard.

DETECTION AND PROTECTION

The immediately-dangerous-to-life-and-health (IDLH) concentration of Lewisite (L) is 0.003 mg/m^3. The M8A1 automatic chemical-agent detector alarm is incapable of detecting Lewisite. However, liquid Lewisite turns M8 paper a ketchup red, and M9 paper will turn pink, red, reddish-brown, or purple when exposed to liquid nerve agents or vesicants, but does not specifically identify either the class of agent or the specific agent. The detectors in the chart below can detect Lewisite (L) at the threshold limits given.

DETECTOR	L
M256A1	14.0 mg/min^3
M272 (in water)	2.0 mg/min^3
M18A2 Kit	10.0 mg/min^3
M21 (RSCAAL)	150.0 mg/min^3
M90	0.2 mg/min^3
M93A1 Fox	10 - 100 mcg/l
ICAM	2.0 mg/min^3

Because the odor of Lewisite may be faint or lost after accommodation, olfactory detection of the odor of geraniums is not a reliable indicator of Lewisite exposure. The activated charcoal in the canister of the chemical protective mask adsorbs Lewisite, as does the charcoal in the chemical protective overgarment. Lewisite attacks the butyl rubber in the chemical protective gloves and boots, which nevertheless are expected to protect against field concentrations of Lewisite until they can be exchanged for fresh gloves and boots. Proper wear of the chemical protective mask and chemical protective ensemble affords full protection against Lewisite.

MECHANISM OF TOXICITY

Lewisite is readily absorbed from the skin, eyes, and respiratory tract, as well as by ingestion and via wounds. It is systemically distributed to almost all organs and tissues of the body where it participates in a variety of chemical reactions. It is eventually eliminated primarily as reaction products in the urine.

TOXICITY

Lewisite causes nasal irritation at a Ct of about 8 mg-min/m^3, and its odor is noted at a Ct of about 20 mg-min/m^3. Lewisite causes vesication and death from inhalation at the same

Ct as mustard. Liquid Lewisite causes vesication at about 14 mcg, and the LD_{50} of liquid Lewisite applied to the skin is about 2.8 grams.

TOXICODYNAMICS (MECHANISM OF ACTION)

Although Lewisite contains trivalent arsenic and combines with thiol groups in many enzymes, its exact mechanism of biological activity is unknown.

CLINICAL EFFECTS

Organ Systems. Unlike mustard, Lewisite vapor or liquid causes immediate pain or irritation. A person with a droplet of Lewisite on his skin will note the burning and will immediately take steps to try and remove it. The vapor is so irritating that a person will seek to mask or leave the contaminated area if possible. Because this warning causes the person exposed to take immediate steps to decontaminate, the Lewisite lesion will probably not be as severe as the lesion from mustard, since exposure to mustard is often undetected and decontamination is not done.

There are almost no data on humans exposed to Lewisite. The following information is based on animal investigations.

Skin. Within about five minutes after contact, liquid Lewisite will produce a grayish area of dead epithelium. Erythema and blister formation follow more rapidly than in a similar lesion from mustard, although the full lesion does not develop for 12 to 18 hours. The lesion has more tissue necrosis and tissue sloughing than does a mustard lesion.

Eye. Lewisite causes pain and blepharospasm on contact. Edema of the conjunctiva and lids follows, and the eyes may be swollen shut within an hour. Iritis and corneal damage may follow if the dose is high. Liquid Lewisite causes severe eye damage within minutes of contact.

Respiratory. The extreme irritancy of Lewisite to the nasal area and upper airways causes the person to mask or exit the area. Scanty data indicate that Lewisite causes the same airway signs and symptoms as mustard. The airway mucosa is the primary target, and damage progresses down the airways in a dose-dependent manner. Pseudomembrane formation is prominent. Pulmonary edema, which occurs rarely and usually only to a minimal degree after mustard exposure, may complicate exposure to Lewisite.

Other. Available data suggest that Lewisite causes an increase in permeability of systemic

capillaries with resulting intravascular fluid loss, hypovolemia, shock, and organ congestion. This may lead to hepatic or renal necrosis with more prominent GI effects (including vomiting and diarrhea) than after mustard.

Physical Findings. The findings are similar to those caused by mustard. As noted, the tissue damage at the site of the skin lesion may be more severe.

TIME COURSE OF EFFECTS

Pain and irritation from either liquid or vapor Lewisite are immediate. Early tissue destruction is more obvious than after mustard, but the lesion is not full-blown for 12 hours or longer.

DIFFERENTIAL DIAGNOSIS

Although differences have been reported between the skin lesions from mustard and Lewisite (less surrounding erythema and more tissue destruction characterize Lewisite blisters), these are of little diagnostic assistance in a single patient. The history of immediate pain on contact is absent after mustard exposure and present after Lewisite or phosgene oxime exposures.

Other substances cause erythema and blisters, and often the history of exposure is the most helpful tool in diagnosis.

LABORATORY FINDINGS

There is no specific diagnostic test for Lewisite. Leukocytosis, fever, and other signs of tissue destruction will occur.

MEDICAL MANAGEMENT

Early decontamination is the only way of preventing or lessening Lewisite damage. Since this must be accomplished within minutes after exposure, this is self-aid rather than medical management.

The guidelines for the management of a mustard casualty will be useful. Lewisite does not cause damage to hematopoietic organs as mustard does; however, fluid loss from the capillaries necessitates careful attention to fluid balance.

BAL was developed as an antidote for Lewisite and is used in medicine as a chelating agent for heavy metals. There is evidence that BAL in oil, given intramuscularly, will reduce the systemic effects of Lewisite. However, BAL itself causes some toxicity, and the user should read

the package insert carefully. BAL skin and ophthalmic ointment decreases the severity of skin and eye lesions when applied immediately after early decontamination; however, neither is currently manufactured.

TRIAGE

Casualties should be triaged using the guidelines for triage of mustard patients.

RETURN TO DUTY

Casualties with minor skin lesions who receive symptomatic therapy can be returned to duty quickly. Because Lewisite generally causes more tissue damage than mustard, casualties with eye and larger skin lesions should be triaged as **delayed** and **evacuated**. Whether to triage those with pulmonary injury as immediate, delayed, or expectant depends on the severity of the injury and how quickly after exposure it occurred.

PHOSGENE OXIME

CX

SUMMARY

Signs and Symptoms: Immediate burning and irritation followed by wheal-like skin lesions and eye and airway damage.

Field Detection: M256A1 and M18A2 chemical agent detector kits; M90 chemical agent detector, M93A1 FOX NBC RECONNAISSANCE SYSTEM.

Decontamination: M291, soap and water, 0.5% bleach solution.

Management: Immediate decontamination, symptomatic management of lesions.

OVERVIEW

Phosgene oxime (CX) is an urticant or nettle agent that causes a corrosive type of skin and tissue lesion. It is not a true vesicant since it does not cause blisters. The vapor is extremely irritating, and both the vapor and liquid cause almost immediate tissue damage upon contact. There is very scanty information available on CX.

MILITARY SIGNIFICANCE

There is no current assessment of the potential of CX as a military threat agent.

PHYSIOCHEMICAL CHARACTERISTICS

Phosgene oxime is a solid at temperatures below 95^{o}F/35^{o}C, but the vapor pressure of the solid is high enough to produce symptoms. Traces of many metals cause it to decompose; however, it corrodes most metals.

DETECTION AND PROTECTION

The IDLH concentration of CX has not been defined. The M272 water testing kit, MINICAMS, ICAD, M21 remote sensing alarm, CAM, ACAMS, DAAMS, and M8A1 automatic chemical agent detector alarm are incapable of detecting CX. Likewise, M8 and M9 paper

should not be depended upon to detect this agent. The M256A1 detector ticket reacts to the presence of CX, but the detection threshold is not known with certainty. The detectors in the following chart are capable of detecting CX at the threshold limits given.

DETECTOR	CX
M18A2	0.5 mg/min^3
M90	0.15 mg/min^3
M93A1 Fox	10 - 100 mcg/l

Because the odor of phosgene may be faint or lost after accommodation, olfactory detection of a pepperish or pungent odor is not a reliable indicator of the presence of CX. The activated charcoal in the canister of the chemical protective mask adsorbs CX, as does the charcoal in the chemical protective overgarment. Phosgene oxime may attack the butyl rubber in the chemical protective gloves and boots, which nevertheless are expected to protect against field concentrations of CX until they can be exchanged for fresh gloves and boots. Proper wear of the chemical protective mask and chemical protective ensemble affords full protection against CX.

MECHANISM OF TOXICITY

The toxicokinetics of CX are not known in detail. Penetration of exposed surfaces is rapid, and systemic distribution to most organs and tissues, including the GI tract, is probably important.

TOXICITY

The estimated LCt_{50} by inhalation is 1500-2000 mg·min/m^3. The LD_{50} for skin exposure has been estimated as 25 mg/kg.

TOXICODYNAMICS (MECHANISM OF ACTION)

The mechanism by which CX causes biological effects is unknown.

CLINICAL EFFECTS

Skin. Phosgene oxime liquid or vapor causes pain on contact, which is followed in turn by blanching with an erythematous ring in 30 seconds, a wheal in 30 minutes, and necrosis later. The extreme pain may persist for days.

Eye. Phosgene oxime is extremely painful to the eyes. The damage is probably similar to that caused by Lewisite.

Pulmonary. Phosgene oxime is very irritating to the upper airways. This agent causes pulmonary edema after inhalation and after skin application.

Other. Some animal data suggest that CX may cause hemorrhagic inflammatory changes in the GI tract.

TIME COURSE OF EFFECTS

Phosgene oxime causes immediate pain and irritation to all exposed skin and mucous membranes. The time course of damage to other tissue probably parallels that of damage to the skin.

DIFFERENTIAL DIAGNOSIS

Other causes of urticaria and skin necrosis must be considered. Common urticants do not cause the extreme pain that CX does.

LABORATORY FINDINGS

There are no distinctive laboratory findings.

MEDICAL MANAGEMENT

Management is supportive. The skin lesion should be managed in the same way that a necrotic ulcerated lesion from another cause would be managed.

TRIAGE

Because of the continuing pain, most casualties should be placed in the **delayed** category and evacuated.

RETURN TO DUTY

The decision to return a CX casualty to duty should be based on healing of the lesion(s) and the casualty's freedom from discomfort.

NERVE AGENTS

GA, GB, GD, GF, VX

SUMMARY

Signs and Symptoms:

Vapor:

Small exposure -- miosis, rhinorrhea, mild difficulty breathing.

Large exposure -- sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions, miosis.

Liquid on skin:

Small to moderate exposure -- localized sweating, nausea, vomiting, feeling of weakness.

Large exposure -- sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions.

Field Detection: M256A1 chemical agent detector kit, M8 chemical agent detector paper, M9 chemical agent detector paper; Improved Chemical Agent Alarm (ICAM), M93A1 FOX NBC RECONNAISSANCE System, M18A2 chemical agent detector kit, M21 Remote Sensing Chemical Agent Alarm (RSCAAL), M90 chemical agent detector, M22 Automatic Chemical Agents Detection Alarm (ACADA).

Decontamination: M291 SDK, soap and water, 0.5% Hypochlorate solution.

Immediate management: Administration of MARK I Kits (atropine and pralidoxime chloride); diazepam in addition if casualty is severe; ventilation and suction of airways for respiratory distress.

OVERVIEW

Nerve agents are the most toxic of the known chemical agents. They are hazards in their liquid and vapor states and can cause death within minutes after exposure. Nerve agents inhibit acetylcholinesterase in tissue, and their effects are caused by the resulting excess acetylcholine.

HISTORY/MILITARY RELEVANCE

Nerve agents were developed in pre-WWII Germany. Germany had stockpiles of nerve agent munitions during WWII but did not use them for reasons that are still unclear. In the closing days of the war, the U.S. and its allies discovered these stockpiles, developed the agents, and manufactured nerve agent munitions. The U.S. chemical agent stockpile, which is in the process of being destroyed, contains the nerve agents sarin (GB) and VX.

Nerve agents are considered major military threat agents. The only known battlefield use of nerve agents was in the Iraq-Iran conflict. Intelligence analysts indicate that many countries have the technology to manufacture nerve agent munitions.

PHYSICAL CHARACTERISTICS

Nerve agents are liquids under temperate conditions. When dispersed, the more volatile ones constitute both a vapor and a liquid hazard. Others are less volatile and represent primarily a liquid hazard. The "G-agents" are more volatile than VX. Sarin (GB) is the most volatile, but it evaporates less readily than water. GF is the least volatile of the G-agents.

Nerve agents can be dispersed from missiles, rockets, bombs, howitzer shells, spray tanks, land mines, and other large munitions.

DETECTION AND PROTECTION

The IDLH concentrations of nerve agents are 0.0001 mg/m^3 for tabun (GA), 0.0001 mg/m^3 for sarin (GB), 0.0003 mg/m^3 for soman (GD), 0.0001 mg/m^3 for GF, and 0.0001 mg/m^3 for VX. Liquid G agents turn M8 paper a "gold" yellow, and VX turns M8 paper a "verdana" or "olive" green. M9 paper will turn pink, red, reddish brown, or purple when exposed to liquid nerve agents or vesicants but does not specifically identify either the class of agent or the specific agent.

Because the odor of nerve agents may be faint or lost after accommodation, olfactory detection of the odor of fruit or fish is not a

reliable indicator of mustard exposure. The activated charcoal in the canister of the chemical protective mask adsorbs nerve agents present as vapor or gas, as does the charcoal in the chemical protective overgarment; and the butyl rubber in the chemical protective gloves and boots is impermeable to nerve agents. Proper wear of the protective mask and the chemical protective ensemble affords full protection against nerve agents.

Table: The following detectors have the capacity to detect nerve agents at the threshold limits given.

Detector	GA (Tabun)	GB (Sarin)	GD (Soman)	GF	VX
M256A1	Unknown	0.05 mg/m^3	Unknown	Unknown	0.02 mg/m^3
M272 (in water)	0.02 mg/m^3	0.02 mg/m^3	0.02 mg/m^3		0.02 mg/m^3
M18A2		0.02 mg/m^3			0.1 mg/m^3
M21 RSCAAL	3.0 mg/m^3	3.0 mg/m^3	3.0 mg/m^3		
M90	0.04 mg/m^3	0.04 mg/m^3	0.04 mg/m^3	0.04 mg/m^3	0.04 mg/m^3
M93A1 Fox		62 mg/m^3	0.1-1 mcg/l		1-10 mcg/l
CAM	0.03 mg/m^3	0.03 mg/m^3	0.03 mg/m^3	0.03 mg/m^3	0.01 mg/m^3
M22 ACADA	0.001 PPM	0.002 PPM	0.002 PPM		0.0009 PPM

MECHANISM OF TOXICITY

Nerve agents are organophosphorus cholinesterase inhibitors. They inhibit the butyrylcholinesterase in the plasma, acetylcholinesterase on the red cell, and acetylcholinesterase at cholinergic receptor sites in tissue. The three enzymes are not the same; even the two acetylcholinesterases have slightly different properties, although both have a high affinity for acetylcholine. The blood enzymes provide an estimate of the tissue enzyme activity. After acute exposure to a nerve agent, the erythrocyte enzyme activity most closely reflects the activity of the tissue enzyme, but during recovery the plasma enzyme activity more closely parallels tissue enzyme activity.

After a nerve agent inhibits the tissue enzyme, the enzyme cannot hydrolyze acetylcholine, the neurotransmitter, at cholinergic receptor sites. Acetylcholine accumulates and continues to stimulate the affected organ. The clinical effects from nerve agent exposure are caused by excess acetylcholine.

The attachment of the agent to the enzyme is permanent (unless removed by therapy). Erythrocyte enzyme activity returns at the rate of erythrocyte turnover, about 1% per day. Tissue and plasma enzyme activities return with

synthesis of new enzymes. The rate of return of the tissue and plasma enzymes is not the same, nor is the rate the same for all tissue enzymes. However, the agent can be removed from the enzyme and the enzyme "reactivated" by several types of compounds, the most useful of which are the oximes. If the agent-enzyme complex has not "aged," oximes are useful therapeutically. Aging is a biochemical process by which the agent-enzyme complex becomes refractory to oxime reactivation of the enzyme. For most nerve agents, the aging time is longer than the time within which acute casualties will be seen. However, the aging time of the GD-enzyme complex is about two minutes, and the usefulness of oximes in GD poisoning is greatly decreased after this period.

Organs with cholinergic receptor sites include the smooth muscles, skeletal muscles, CNS, and most exocrine glands. In addition, cranial efferents and ganglionic afferents are cholinergic nerves.

Muscarine will stimulate some of the cholinergic sites, and these are known as muscarinic sites. Organs with these sites include the smooth muscles and glands. Nicotine will stimulate other cholinergic sites, known as nicotinic sites, which are those in skeletal muscle and ganglia. The CNS contains both types of receptors, but the pharmacology in the

CNS is more complex and less well understood. Atropine and similar compounds block the effects of excess acetylcholine more effectively at muscarinic sites than at nicotinic sites.

Some commonly used pesticides (for example, the organophosphate [OP] Malathion and the carbamate Sevin) and some common therapeutic drugs (the carbamates pyridostigmine [Mestinon] and physostigmine [Antilirium]) also inhibit acetylcholinesterase and can be considered "nerve agents." However, while the OP pesticides cause the same biological effects as nerve agents, there are some important differences in the duration of biological activity and response to therapy.

CLINICAL EFFECTS

The initial effects of exposure to a nerve agent depend on the dose and route of exposure. The initial effects from a sublethal amount of agent by vapor exposure are different than the initial effects from a similar amount of liquid agent on the skin.

Toxicities. The estimated amounts to cause certain effects in man are shown in Tables I and II. In Table I, L indicates lethal, I indicates incapacitating (severe), and M indicates miosis. The large amounts of GA and GB required to produce effects after skin

application reflect the volatility of these agents. They evaporate rather than penetrate the skin. However, if these agents are occluded and prevented from evaporating, they penetrate the skin very well.

Table I.
Vapor Toxicity (mg-min/$m^{3)}$

Agent	LCt_{50}	ICt_{50}	MCt_{50}
GA	400	300	2-3
GB	100	75	3
GD	70	Unknown	<1
GF	Unknown	Unknown	<1
VX	50	35	0.04

Table II.
LD_{50} on Skin

Agent	Amount
GA	1000 mg
GB	1700 mg
GD	50 mg
GF	30 mg
VX	10 mg

Sarin (GB), the agent studied most thoroughly in man, will cause miosis, rhinorrhea, and a feeling of tightness in the throat or chest at a Ct of 3 to 5 mg·min/m^3.

Effect. Exposure to a small amount of nerve agent vapor causes effects in the eyes, nose, and airways. These effects are from local contact of the vapor with the organ and do not indicate systemic absorption of the agent. In this circumstance, the erythrocyte-ChE may be normal or depressed. A small amount of liquid agent on the skin causes systemic effects initially in the (GI) tract. Lethal amounts of vapor or liquid cause a rapid cascade of events culminating within a minute or two with loss of consciousness and convulsive activity, followed by apnea and muscular flaccidity within several more minutes.

Eye. Miosis is a characteristic sign of exposure to nerve agent vapor. It occurs as a result of direct contact of vapor with the eye. Liquid agent on the skin will not cause miosis if the amount of liquid is small. A moderate amount of liquid may or may not cause miosis. A lethal or near-lethal amount of agent usually causes miosis. A droplet of liquid in or near the eye will also cause miosis. Miosis will begin within seconds or minutes after the onset of exposure to agent vapor, but it may not be complete for many minutes if the vapor

concentration is low. Miosis is bilateral in an unprotected individual, but occasionally may be unilateral in a masked person with a leak in his mask eyepiece.

Miosis is often accompanied by complaints of pain, dim vision, blurred vision, conjunctival injection, nausea, and occasionally, vomiting. The pain may be sharp or dull, in or around the eyeball, but more often is a dull ache in the frontal part of the head. Dim vision is due in part to the small pupil, and cholinergic mechanisms in the visual pathways also contribute. The complaint of blurred vision is less easily explained, as objective testing usually indicates an improvement in visual acuity because of the "pin-hole" effect. Conjunctival injection may be mild or severe, and occasionally subconjunctival hemorrhage is present. Nausea (and sometimes vomiting) is part of a generalized complaint of not feeling well. Topical homatropine or atropine in the eye can relieve miosis, pain, dim vision, and nausea.

Nose. Rhinorrhea may be the first indication of nerve agent vapor exposure. Its severity is dose dependent.

Airway. Nerve agent vapor causes bronchoconstriction and increased secretions of the glands in the airways in a dose-related manner. The exposed person may feel a slight

tightness in his chest after a small amount of agent and may be in severe distress after a large amount of agent. Cessation of respiration occurs within minutes after the onset of effects from exposure to a large amount of nerve agent. This apnea is probably mediated through the CNS, although peripheral factors (skeletal muscle weakness, e.g., the intercostal muscles, and bronchoconstriction) may contribute.

GI tract. After they are absorbed, nerve agents cause an increase in the motility of the GI tract and an increase in secretions by the glands in the wall of the GI tract. Nausea and vomiting are early signs of liquid exposure on the skin. Diarrhea may occur with large amounts of agent.

Glands. Nerve agent vapor causes increases in secretions from the glands it contacts, such as the lacrimal, nasal, salivary, and bronchial glands. Localized sweating around the site of liquid agent on the skin is common, and generalized sweating after a large liquid or vapor exposure is common. Increased secretions of the glands of the GI tract occur after systemic absorption of the agent by either route.

Skeletal muscle. The first effect of nerve agents on skeletal muscle is stimulation producing muscular fasciculations and twitching.

After a large amount of agent, fatigue and weakness of muscles are rapidly followed by muscular flaccidity.

Fasciculations are sometimes seen early at the site of a droplet of liquid agent on the skin, and generalized fasciculations are common after a large exposure. These may remain long after most of the other acute signs decrease.

Central nervous system (CNS). The acute CNS signs of exposure to a large amount of nerve agent are loss of consciousness, seizure activity, and apnea. These begin within a minute after exposure to a large amount of agent vapor and may be preceded by an asymptomatic period of 1 to 30 minutes after contact of liquid with the skin.

After exposure to smaller amounts of nerve agents, CNS effects vary and are nonspecific. They may include forgetfulness, an inability to concentrate fully, insomnia, bad dreams, irritability, impaired judgment, and depression. They do not include frank confusion and misperceptions (i.e., hallucinations). These may occur in the absence of physical signs or other symptoms of exposure. After a severe exposure, these symptoms occur upon recovery from the acute severe effects. In either case, they may persist for as long as four to six weeks.

Cardiovascular. The heart rate may be decreased because of stimulation by the vagus nerve, but it is often increased because of other factors such as fright, hypoxia, and the influence of adrenergic stimulation secondary to ganglionic stimulation. Thus, the heart rate may be high, low, or in the normal range. Bradyarrhythmias such as first-, second-, or third-degree heart block may occur. The blood pressure may be elevated from adrenergic factors, but is generally normal until the terminal decline.

PHYSICAL FINDINGS

Physical findings depend on the amount and route of exposure. After exposure to small to moderate amounts of vapor, there are usually miosis and conjunctival injection, rhinorrhea, and pulmonary signs, although the latter may be absent even in the face of mild to moderate pulmonary complaints. In addition to these signs, an exposure to a high Ct may precipitate copious secretions from the nose and mouth, generalized muscular fasciculations, twitching or seizure activity, loss of consciousness, and apnea. Cyanosis, hypotension, and bradycardia may be present just before death.

Exposure to a small droplet of liquid on the skin may produce few physical findings. Sweating, blanching, and occasionally,

fasciculations, at the site may be present soon after exposure, but may no longer be present at the onset of GI effects. After a large exposure, the signs are the same as after vapor exposure.

Miosis is a useful sign of exposure to vapor but does not occur after a liquid exposure unless the amount of exposure is large or the exposure is in or close to the eye.

TIME COURSE OF EFFECTS

Effects from nerve agent vapor begin within seconds to several minutes after exposure. Loss of consciousness and onset of seizure activity have occurred within a minute of exposure to a high Ct. After exposure to a very low Ct, miosis and other effects may not begin for several minutes, and miosis may not be complete for 15 to 30 minutes after removal from the vapor. There is no latent period or delay in onset from vapor exposure. Effects may continue to progress for a period of time, but maximal effects usually occur within minutes after exposure stops.

A large amount of liquid on the skin causes effects within minutes. Commonly there is an asymptomatic period of 1 to 30 minutes, and then the sudden onset of an overwhelming cascade of events, including loss of consciousness, seizure activity, apnea, and

muscular flaccidity. After small amounts of liquid agent on the skin, the onset of effects has been delayed for as long as 18 hours after contact. These effects are initially gastrointestinal and are usually not life-threatening. Generally, the longer the interval, the less severe are the effects.

DIFFERENTIAL DIAGNOSIS

The effects caused by a mild vapor exposure, namely, rhinorrhea and tightness in the chest, may easily be confused with an upper respiratory malady or an allergy. Miosis, if present, will help to distinguish these, but the eyes must be examined in very dim light to detect this. Similarly, GI symptoms from another illness may be confused with those from nerve agent effects, and in this instance there will be no useful physical signs. History of possible exposure will be helpful, and laboratory evidence (decreased RBC-ChE activity), if available, will be useful to distinguish the two.

The diagnosis is easier in the severely intoxicated patient. The combination of miosis, copious secretions, and generalized muscular fasciculations in a gasping, cyanotic, and convulsing patient is characteristic.

LABORATORY FINDINGS

Nerve agents inhibit the cholinesterase activity of the blood components, and estimation of this activity is useful in detecting exposure to these agents. The erythrocyte enzyme activity is more sensitive to acute nerve agent exposure than is the plasma enzyme activity.

The amount of inhibition of this enzyme activity does not correlate well with the severity of local effects from mild to moderate vapor exposure. The enzyme activity may be from 0 to 100% of the individual's normal activity in the face of miosis, rhinorrhea, and/or airway symptoms. Normal or nearly normal erythrocyte acetylcholinesterase activity may be present with moderate effects in these organs. At the other extreme, the enzyme may be inhibited by 60 to 70% when miosis or rhinorrhea is the only sign of exposure. Severe systemic effects generally indicate inhibition of the erythrocyte acetylcholinesterase by 70 to 80% or greater.

Other laboratory findings will relate to complications. For example, acidosis may occur after prolonged hypoxia.

MEDICAL MANAGEMENT

Management of a casualty with nerve agent intoxication consists of decontamination, ventilation, administration of the antidotes, and supportive therapy. The condition of the patient dictates the need for each of these and the order in which they are done.

Decontamination is described elsewhere in this manual. Skin decontamination is not necessary after exposure to vapor alone, but clothing should be removed because it may contain "trapped" vapor.

The need for **ventilation** will be obvious, and the means of ventilation will depend on available equipment. Airway resistance is high (50 to 70 cm of water) because of bronchoconstriction and secretions, and initial ventilation is difficult. The resistance decreases after atropine administration, after which ventilation will be easier. The copious secretions that may be thickened by atropine also impede ventilatory efforts and require frequent suctioning. In reported cases of severe nerve agent exposure, ventilation has been required from 0.5 to 3 hours.

Three drugs are used to treat nerve agent exposure, and another is used as pretreatment for potential nerve agent exposure. The three

therapeutic drugs are atropine, pralidoxime chloride, and diazepam. The use of the pretreatment drug pyridostigmine bromide is discussed later in this chapter.

Atropine is a cholinergic blocking or anticholinergic compound. It is extremely effective in blocking the effects of excess acetylcholine at peripheral muscarinic sites. When small amounts (2 mg) are given to normal individuals without nerve agent intoxication, atropine causes mydriasis, a decrease in secretions (including a decrease in sweating), mild sedation, a decrease in GI motility, and tachycardia. The amount in three MARK I Kits may cause adverse effects on military performance in a normal person. In people not exposed to nerve agents, amounts of 10 mg or higher may cause delirium. Potentially, the most hazardous effect of inadvertent use of atropine (2 mg, IM) in a young person not exposed to a cholinesterase-inhibiting compound in a warm or hot atmosphere is inhibition of sweating, which may lead to heat injury. In the military, atropine is packaged in autoinjectors, each containing 2 mg.

Pralidoxime chloride (Protopam chloride, 2-PAMCl) is an oxime. Oximes attach to the nerve agent that is inhibiting the cholinesterase and break the agent-enzyme bond to restore the normal activity of the enzyme. Clinically, this is

noticeable in those organs with nicotinic receptors. Abnormal activity in skeletal muscle decreases and normal strength returns. The effects of an oxime are not apparent in organs with muscarinic receptors; oximes do not cause a decrease in secretions, for example. They also are less useful after aging occurs, but with the exception of GD (soman) intoxicated individuals, casualties will be treated before significant aging occurs. Pralidoxime chloride (600 mg) is in an autoinjector for self-use along with the atropine injector. These atropine and pralidoxime chloride autoinjectors are packaged together in a MARK I Kit. Each warfighter is issued three MARK I Kits.

Diazepam is an anticonvulsant drug used to decrease convulsive activity and reduce the brain damage caused by prolonged seizure activity. Without the use of pyridostigmine pretreatment, experimental animals died quickly after superlethal doses of nerve agents despite conventional therapy. With pyridostigmine pretreatment (followed by conventional therapy), animals survived superlethal doses of soman but had prolonged periods of seizure activity before recovery. They later had performance decrements and anatomic lesions in their brains. The administration of diazepam with other standard therapy to soman-poisoned animals pretreated with pyridostigmine reduced the seizure activity and its sequelae. Current military doctrine is to administer diazepam with other

therapy (three MARK I Kits) at the onset of severe effects from a nerve agent, whether or not seizure activity is among those effects. Each warfighter carries one autoinjector containing 10 mg of diazepam for his buddy to administer to him (if he could self-administer it, he would not need it). **Diazepam should be administered with the three MARK I Kits when the casualty's condition warrants the use of three kits at the same time.** Medical personnel can administer more diazepam to a casualty if necessary. The medical corpsman carries extra diazepam injectors and is authorized to administer two additional injectors at ten-minute intervals to a convulsing casualty.

The doctrine for **self-aid** for nerve agent intoxication states that if an individual has effects from the agent, he/she should self-administer one MARK I Kit. If there is no improvement in 10 minutes, he/she should seek out a buddy to assist in the evaluation of his/her condition before further MARK I Kits are given. If a buddy finds an individual severely intoxicated (e.g., gasping respirations, twitching, etc.) so that the individual cannot self-administer a MARK I Kit, the buddy should administer three MARK I Kits and diazepam immediately. The discussion below is advice for medical assistance.

The appropriate number of MARK I Kits to administer initially to a casualty from nerve agent vapor depends on the severity of the effects. Systemic atropine will not reverse miosis (unless administered in very large amounts), and miosis alone is not an indication for a MARK I Kit. If the eye or head pain and nausea associated with the miosis are severe, topical application of atropine (or homatropine) in the eye will bring relief. Topical atropine should not be used without good reason (severe pain), because it causes blurred vision for a day or longer. A casualty with miosis and rhinorrhea should be given one MARK I Kit only if the rhinorrhea is severe and troublesome (he cannot keep his mask on because of fluid). A casualty with mild to moderate dyspnea should be given one or two MARK I Kits, depending on the severity of his distress and the time between exposure and therapy. Some of the respiratory distress from a mild exposure will spontaneously decrease within 15 to 30 minutes after termination of exposure, so if the casualty is not severely uncomfortable, only one MARK I Kit should be used initially. Atropine is quite effective, and care should be taken not to give too much in a casualty who does not need it.

A severe casualty from nerve agent vapor has miosis, copious secretions from the nose and mouth, severe difficulty breathing or apnea, possibly some degree of cyanosis, muscular fasciculations, and twitching or convulsive activity, and is unconscious. He should be given three MARK I Kits and diazepam immediately. Ventilation will be needed and should be done via an endotracheal airway if possible. Suctioning of the excessive airway secretions will be necessary to enhance air exchange and will make ventilatory efforts easier. Atropine, 2 mg, should be repeated at three- to five-minute intervals and should be titrated to a reduction of secretions and to reduction of ventilatory resistance. When the IV preparation is available, the preferred route of atropine administration is via the IV route, but this route should be avoided until hypoxia is corrected, because intravenously administered atropine in hypoxic animals has produced ventricular fibrillation. In a hypotensive patient or a patient with poor veins, atropine might be given intratracheally, either via the endotracheal tube or directly into the trachea, for more rapid absorption via the peribronchial vessels.

The medical care provider might err in giving too much atropine to a mild to moderate casualty. More importantly, the care provider might err by giving too little atropine to a severe casualty. In a severe casualty, atropine should

be pushed at frequent intervals until secretions are dry (or nearly dry) and until ventilation can be accomplished with ease. In reported cases this has required 10 to 20 mg of atropine within the first several hours. A conscious, less severely exposed casualty should receive atropine until he is breathing comfortably, and he will be able to communicate this. Dry secretions need not be an endpoint in mild to moderate casualties.

The casualty with skin exposure to liquid is more difficult to evaluate and manage than is a vapor exposure casualty. Agent on the surface of the skin can be decontaminated, but agent absorbed into the skin cannot be removed. The initial effects from absorbed liquid agent can start two to three hours after thorough decontamination of agent droplets on the skin. A casualty from liquid exposure on the skin may continue to worsen because of continued absorption of the agent from the skin depot.

The first effects of a liquid droplet on the skin are sweating with or without blanching, and occasionally, muscular fasciculations at the site. Gastrointestinal effects (nausea, vomiting, and sometimes diarrhea) are the first systemic effects, and these may start from 0.5 to 18 hours after contact with the agent. If these effects occur within the first several hours after exposure, they may portend more severe

effects, and initial therapy should be two MARK I Kits. If effects begin later, initial therapy should be one MARK I Kit.

A large amount of liquid agent on the skin will cause effects 1 to 30 minutes after contact, whether or not decontamination was done. Nevertheless, early decontamination may lessen the magnitude of the effects. After a 1- to 30-minute latent or asymptomatic period, the casualty will suddenly lose consciousness and begin seizure activity. The condition of the casualty and management are the same as described for a severe casualty from vapor exposure.

Further care of the severe casualty consists of atropine administration to minimize secretions and ventilation until spontaneous respiration resumes. Oxime administration should be repeated at hourly intervals for two or three additional doses. The preferred method of administration of the oxime is by IV drip of 1 gram over 20 to 30 minutes (more rapid administration will cause hypertension), but 3 additional oxime autoinjectors (total dose of 1.8 grams) may be given if the IV route cannot be used. The need for ventilation may continue for 0.5 to 3 hours. Unless prolonged hypoxia or other complications have occurred, the casualty will eventually begin having spontaneous muscular activity and make sporadic attempts to

breathe. Muscles will become stronger and breathing more regular, and the casualty will have intermittent episodes of conscious behavior. Within an hour or two, he will be breathing, moving, and conscious, although he will be weak and intermittently obtunded.

Table III.
Nerve Agent Effects: Vapor Exposure

Mild

- Eyes: miosis, dim vision, headache
- Nose: rhinorrhea
- Mouth: salivation
- Lungs: dyspnea ("tightness in the chest")
- Time of onset: seconds to minutes after exposure
- **Self-aid**: one MARK I Kit
- **Buddy-aid**: stand by

Severe

- All of the above, plus
- Severe breathing difficulty or cessation of respiration
- Generalized muscular twitching, weakness, or paralysis
- Convulsions
- Loss of consciousness
- Loss of bladder, bowel control
- Time of onset: seconds to minutes after exposure
- **Self-aid**: None - warfighter will be unable to help self
- **Buddy-aid**: 3 MARK I Kits and diazepam immediately

Table IV.
Nerve Agent Effects: Liquid on Skin

Mild/moderate

- Muscle twitching at site of exposure
- Sweating at site of exposure
- Nausea, vomiting
- Feeling of weakness
- Time of onset: 10 minutes to 18 hours after exposure
- **Self-aid**: 1 to 2 MARK I Kits, depending on severity of symptoms
- **Buddy-aid**: stand-by

Severe

All of the above, plus

- Severe breathing difficulty or cessation of breathing
- Generalized muscular twitching, weakness, or paralysis
- Convulsions
- Loss of consciousness
- Loss of bladder and bowel control
- Time of onset: minutes to an hour after exposure
- **Self-aid**: none - warfighter will be unable to help himself
- **Buddy-aid**: 3 MARK I Kits and diazepam immediately

PRETREATMENT

In late 1990, the U.S. military fielded pyridostigmine bromide as a pretreatment for nerve agent exposure. Each individual received a blister pack containing twenty-one 30-mg tablets. The dose regimen is one 30-mg tablet every 8 hours. When to start and stop dosing is a division or corps' command decision and is made with the advice of the intelligence, chemical, and medical staffs. To use or to stop the pretreatment is not a local decision, nor is it an individual decision. Thus, pyridostigmine is, in a sense, not a medical treatment, but a defensive weapons system.

Pyridostigmine bromide (Mestinon) is the drug of choice for myasthenia gravis and has been approved for the treatment of this disease since 1951. In 2003, the additional on-label use of pyridostigmine bromide for pretreatment against soman was approved by the Food and Drug Administration. Consequently, commanders have the authority to order its use in this situation without warfighter consent, exactly as they may order an approved vaccine.

When given before soman exposure and when that exposure is followed by the standard MARK I therapy, the use of pretreatment will increase the LD_{50} several fold over the LD_{50} obtained without the use of the pretreatment.

Functionally, this means that a warfighter can survive what would have been otherwise a lethal dose; pyridostigmine converts what would have been a dead warfighter to a very sick patient who can be saved only if the antidotes are properly and promptly administered. When soman is the nerve agent, the use of pyridostigmine increases survival. When the agent is GB or VX, survival after standard MARK I therapy is essentially the same whether or not pyridostigmine pretreatment is used, i.e., pyridostigmine use provides no benefit in GB or VX poisoning. Current data are not adequate to evaluate the effectiveness of pyridostigmine pretreatment for GA or GF exposure.

Pyridostigmine is not an antidote, and it should not be taken after soman exposure. Its use will not decrease the effects of soman. It is ineffective unless standard MARK I therapy is also used in the appropriate manner.

One consequence of the greater survival from the use of pyridostigmine is prolonged seizure activity and subsequent possible brain damage in the survivors. The early administration of diazepam will decrease these effects.

About 50 years ago, it was noted that carbamates bind to the active site of cholinesterase in a similar manner to the binding

of organophosphonate cholinesterase inhibitors to cholinesterase. Additionally, while the carbamate was attached to the active site, an organophosphorus compound could not attach to the enzyme. The carbamate-enzyme binding, or carbamylation, lasts only for hours, rather than for the lifetime of the enzyme as the organophosphorus compound attachment does, and is therefore spontaneously reversible. While the enzyme is carbamylated, the active site is protected from attack by other compounds such as organophosphorus cholinesterase inhibitors, including nerve agents. After several hours, the carbamate leaves the enzyme (i.e., decarbamylation occurs), and the enzyme becomes completely functional again. Thus, the carbamate provides temporary protection for the enzyme against nerve agent attack. Luckily, people have far more AChE than they need, so the use of pyridostigmine to carbamylate a small proportion of AChE converts that proportion into a reserve that will be available to save the patient if soman attack inactivates all of the rest.

Over the past several decades, many carbamates have been investigated for their effectiveness in animals and their safety in man. Pyridostigmine was chosen and underwent extensive testing in humans. It already had a long safety record, having been in use for over 40 years; there are over 16,000 myasthenic patients in the U.S. who use it on a daily basis.

Investigations indicated that it did not interfere with the performance of military tasks and caused no adverse physiological disturbances. The incidence of side effects from the drug during these studies was reported as fewer than 5%.

Tens of thousands of U.S. troops took pyridostigmine during the 1990-91 Gulf War. The incidence of side effects (primarily gastrointestinal and urinary) was over 50%, but only a few percent of the troops sought medical help because of the severity of these effects. The drug was discontinued by the warfighters' medical officers in less than 1% of cases.

TRIAGE

A severe nerve agent casualty who is unconscious, convulsing or post-ictal, breathing with difficulty or apneic, and possibly flaccid will survive with appropriate, immediate therapy, including ventilation, if he still has an intact circulation. He should be triaged as **immediate** if that therapy can be provided. If a blood pressure cannot be obtained, he may be considered **expectant**.

The casualty with severe symptoms who is spontaneously breathing, has not lost consciousness, and has not seized has an excellent chance of survival with a minimal

amount of therapeutic effort. He should be categorized as **immediate** and given three MARK I Kits and diazepam. He may worsen if his exposure was to liquid, and atropine administration should be repeated at frequent intervals. If he loses consciousness, seizes, and becomes apneic, he will be retriaged, and his further care will depend on available resources.

Casualties who are walking and talking and are no longer being exposed to agent will usually be triaged as **minimal**. If a casualty can walk and talk, he is breathing and his circulation is intact. He would not appear to need immediate, life-saving care. This does not preclude self-administration or medic-administration of further antidotes for symptoms, and these should be given as necessary.

A casualty recovering from a severe exposure that has received large amounts of antidotes and has been ventilated will be triaged as **delayed**, because he is in need of further medical observation or care. A casualty who suffered liquid exposure and has been both treated and decontaminated may also be triaged as **delayed.**

RETURN TO DUTY

Return to duty depends on the status of the casualty, his military assignment, and the tactical situation.

Studies indicate that animals with decreased erythrocyte AChE activity from a nerve agent exposure have a decreased LD_{50} for another nerve agent exposure (they are more susceptible to the agent) until that cholinesterase activity returns to at least 75% of its baseline, or pre-exposure activity. Nerve agent-exposed workers in a depot or research facility are prevented from returning to work with agents until this recovery occurs. In a battlefield situation, this conservative management should be balanced against the need for the person and his risk of being exposed to a large amount of agent.

In a military field situation, the capability to analyze blood for erythrocyte cholinesterase activity is usually not available, and the "normal" or baseline activity of each individual is not known. The erythrocyte cholinesterase activity in a casualty with severe systemic effects will be inhibited by 70% or greater (30% or less of his pre-exposure activity), and 45 days or longer will be required for cholinesterase activity to return to 75% of pre-exposure activity. The enzyme activity of a casualty with mild or moderate

effects from agent vapor might be nearly normal or might be markedly inhibited. A prediction of erythrocyte cholinesterase recovery time is unreliable.

Most individuals triaged as minimal could return to duty within several hours if the tactical situation required all available manpower. The lingering ocular and CNS effects may be limiting factors in these cases. These individuals might be able to fire a rifle, but their performance on a tracking screen might be severely decremented because of both visual problems and difficulty in concentrating. These prolonged effects must be thoroughly evaluated before the casualties are returned to duty.

A casualty who had severe effects might be walking and talking after 6 to 24 hours but will still be unfit for most duties. Ideally, he should be kept under medical observation for a week or longer and not returned to duty until recovery of cholinesterase activity. However, the tactical situation may lead to modification of these guidelines.

LONG-TERM EFFECTS

Minor electroencephalographic changes were noted more than a year after nerve agent exposure when averaged EEGs in a group of people who had been exposed to a nerve agent

were compared to a control group. Changes could not be identified in individuals.

Neuropsychiatric changes have been noted in individuals for weeks to months after exposure to insecticides. Both in the Tokyo subway attack and in the Iran-Iraq war, reports of long-term neuropsychiatric changes after exposure to nerve agent have surfaced. Little is known about the pathophysiology of these syndromes, as they are clearly not dose-related and overlap with post-traumatic stress disorder.

Polyneuropathy, reported after OP insecticide poisoning, has not been reported in humans exposed to nerve agents and has been produced in animals only at doses of nerve agents so high that survival would be unlikely. The intermediate syndrome has not been reported in humans after nerve agent exposure, nor has it been produced in animals by nerve agent administration. Muscular necrosis has been produced in animals after high dose nerve agent exposure but reverses within weeks; it has not been reported in humans.

INCAPACITATING AGENTS
BZ, Fentanyl Derivatives

SUMMARY

Signs and Symptoms: **BZ and other glycolates:** mydriasis; dry mouth; dry skin; increased DTRs; decreased level of consciousness; confusion; disorientation; disturbances in perception and interpretation (illusions and/or hallucinations); denial of illness; short attention span; impaired memory. **Fentanyl derivatives (carfentanil):** dizziness, sleepiness, ataxia, miosis (if there is no hypoxia, with hypoxia there is pupil dilation), rapid unconsciousness, vomiting, decreased respirations, central apnea, coma.

Field Detection: No field detector is available for either BZ or fentanyl derivatives.

Decontamination: BZ: Gentle, but thorough flushing of skin and hair with water or soap and water is all that is required. Remove clothing. Fentanyl derivatives (carfentanil): No decontamination required.

Management: **BZ: Antidote:** physostigmine. **Supportive:** monitoring of vital signs, especially core temperature. Fentanyl derivatives (carfentanil): **Antidote:** opioid antagonist naloxone/naltrexone. Supportive: monitoring of vital signs. Proper positioning of patient to maintain airway is critical until effects of central respiratory depression diminish.

OVERVIEW

BZ and other glycolates. BZ is a glycolate anticholinergic compound dispersed as an aerosolized solid (primarily for inhalation) or as agent dissolved in one or more solvents for ingestion or percutaneous absorption (physical properties). Acting as a competitive inhibitor of acetylcholine at postsynaptic and postjunctional muscarinic receptor sites BZ causes peripheral nervous system (PNS) effects that in general are the opposite of those seen in nerve agent poisoning. CNS effects include stupor, confusion, and confabulation with concrete and panoramic illusions and hallucinations, and with regression to automatic "phantom" behaviors such as plucking and disrobing. The U.S. weaponized BZ, but demilitarization began in 1988 and is complete. Refugees fleeing Yugoslavia in 1993 were probably exposed to a glycolate like substance based on anecdotal reports of their behaviors. The combination of anticholinergic PNS and CNS effects aids in the diagnosis of patients exposed to these agents. Physostigmine, which increases the concentration of acetylcholine in synapses and in neuromuscular and neuroglandular junctions, is a specific antidote.

Fentanyl derivatives (carfentanil). Fentanyl, an opioid, has more than a dozen derivatives. While the exact fentanyl derivative

used by the Russians in 2002 is not known at this time, it is believed that it is related to the most potent of the known analogs, carfentanil. It is 10,000 times more potent than the opioid morphine. Carfentanil, produced under the trade name Wildnil®, is a large animal tranquilizer approved for veterinary use. Some believe that it may have been mixed with the anesthetic gas halothane to increase its potency. The U.S. does not use fentanyl analogs as incapacitants, although these were considered through the 1970s. The identification of signs and symptoms aids in the diagnosis of the patients who succumb to a gas. Maintenance of the patient's airway is critical for his survival as well as the rapid administration of the antidote, an opioid antagonist, naloxone or naltrexone.

HISTORY/MILITARY RELEVANCE

BZ and other glycolates. The use of chemicals to induce altered states of mind dates to antiquity and includes the use of plants such as thorn apple (Datura stramonium) that contain combinations of anticholinergic alkaloids. The use of nonlethal chemicals to render an enemy force incapable of fighting dates back to at least 600 B.C. when Solon's warfighters threw hellebore roots into streams supplying water to enemy troops, who then developed diarrhea. In 184 B.C., Hannibal's army used belladonna plants to induce disorientation, and the Bishop of

Muenster in A.D. 1672 attempted to use belladonna-containing grenades in an assault on the city of Groningen. In 1881, members of a railway surveying expedition crossing Tuareg territory in North Africa ate dried dates that tribesmen had apparently deliberately contaminated with *Hyoscyamus falezlez*. In 1908, 200 French warfighters in Hanoi became delirious and experienced hallucinations after being poisoned with a related plant. More recently, accusations of Soviet use of incapacitating agents internally and in Afghanistan were never substantiated.

Following WWII, the U.S. military investigated a wide range of possible nonlethal, psychobehavioral, chemical incapacitating agents to include psychedelic indoles such as lysergic acid diethylamide (LSD-25) and marijuana derivatives, certain tranquilizers, as well as several glycolate anticholinergics. One of the anticholinergic compounds, 3-quinuclidinyl benzilate, was assigned the NATO code BZ and was weaponized beginning in the 1960s for possible battlefield use. Although BZ figured prominently in the plot of the 1990 movie *Jacob's Ladder* as the compound responsible for hallucinations and violent deaths in a fictitious American battalion in Vietnam, this agent never saw operational use. Destruction of American stockpiles began in 1988 and is now complete.

In February 1998, the British Ministry of Defence released an intelligence report that accused Iraq of having stockpiled large amounts of a glycolate anticholinergic incapacitating agent known as Agent 15. As stated above, this compound is speculated either to be identical to BZ or a closely related derivative. Also in 1998, there were allegations that elements of the Yugoslav People's Army used incapacitating agents that caused hallucinations and irrational behavior against fleeing Bosnian refugees. Physical evidence of BZ use in Bosnia remains elusive, however.

Terms. The term "incapacitation," when used in a general sense, is roughly equivalent to the term "disability" as used in occupational medicine and denotes the inability to perform a task because of a quantifiable physical or mental impairment. In this sense, any of the chemical warfare agents may incapacitate a victim; however, again by the military definition of this type of agent, incapacitation refers to impairments that are temporary and nonlethal. Thus, riot-control agents are incapacitating because they cause temporary loss of vision due to blepharospasm, but they are not considered military incapacitants because the loss of vision does not last long.

Although incapacitation may result from physiological changes such as mucous

membrane irritation, diarrhea, or hyperthermia, the term "incapacitating agent" as militarily defined refers to a compound that produces temporary and nonlethal impairment of military performance by virtue of its psychobehavioral or CNS effects.

Sources other than military. BZ and related anticholinergic compounds can be synthesized in clandestine laboratories, but its illicit use is low possibly because of some unpleasant effects. The anticholinergics atropine, oxybutynin, and scopolamine find use in clinical medicine and are available as pharmaceuticals, as are antihistamines that have prominent anticholinergic side effects. Finally, anticholinergic hallucinogenic compounds are present in plants of the family *Solanaceae*, which include thorn apple (Jimson weed, *Datura stramonium*), black henbane (*Hyoscyamus niger*), belladonna (deadly nightshade, *Atropa belladonna*), woody nightshade (*Solanum dulcamara*), and Jerusalem cherry (*Solanum pseudocapsicum*). These plants contain varying proportions of the anticholinergic glycolates atropine, hyoscyamine, and hyoscine. Finally, BZ itself, now called QNB in the scientific community, is widely used in pharmacology as a muscarinic receptor marker.

Fentanyl derivatives (carfentanil). Fentanyl was first synthesized by Belgian scientists in the 1950s. Since that time more than a dozen analogs of fentanyl have been developed, each more potent than the original fentanyl. Long used as an anesthetic and analgesic for surgery and chronic pain management, fentanyl analogs were considered for use as an incapacitating agents by several nations, including the U.S., up through the 1970s. To date the U.S. has never weaponized fentanyl analogs or used any as an incapacitant. The Russian government admitted that on October 23, 2002, its Special Forces used a knockout gas containing a “fentanyl derivative” to incapacitate 50 Chechen terrorists, who threatened to blow up a Moscow theater and their 800 hostages before storming the building at the end of a 3-day stand-off. At the end of the siege all of the terrorists were dead, shot by the Russian Special Forces after many succumbed to the gas, and 129 of the 800 hostages (16%) died as a result of medical complications from the gas. The hostage death toll would assuredly have been greater if the Russian forces had stormed the theater without the incapacitating gas and the explosives had been successfully detonated by the terrorists.

It is reported by survivors that the gas was released into the theater through a hose or pipe near the floor at the front of the theater. The

release took place in the early morning when most inside the theater were sleeping after a tense night of negotiation. The ventilation system in the theater was not operating, as it had been disabled by the terrorists. Food and water were limited, so hostages were dehydrated. Hostages ranged in age from 13 years old to the elderly, and all were seated at different locations in the theater relative to the source of the gas. These factors resulted in vastly different dosing of the individuals in the theater. While a majority of those sleeping inside the theater succumbed quickly to the gas, not all did so completely or immediately. In fact, one hostage stumbled out of the theater and some of the male terrorists fled to the rear of the theater. Because of this, some survivors have reported that Special Forces allowed the gas to flow into the theater for up to an hour before beginning their entry. This further increased the dose received by the hostages who succumbed early to the gas. While medical providers had some supplies of the antidote naloxone, there reportedly was not enough and it did not work for all patients. Many medical providers at hospitals were unaware of the antidote needed and tried a variety of remedies before discovering that naloxone worked for some of the survivors. Many hostages, upon going unconscious, slumped in their seats with their necks positioned in ways that restricted already suppressed breathing efforts for the long period

between release and rescue. Others became unconscious and then vomited, choking on their own vomit. Some patients, unconscious but still breathing when Special Forces entered the building, were positioned on the pavement outside the theater in supine or on city buses with necks hyper-extended for transport. These actions caused airways to become compromised, resulting in asphyxiation. In one instance a bus driver was unable to find a hospital and became lost, further prolonging medical care.

PHYSIOCHEMICAL CHARACTERISTICS

BZ is the NATO code for 3-quinuclidinyl benzilate (QNB). BZ is odorless. It is stable in most solvents, with a half-life of three to four weeks in moist air; even heat-producing munitions can disperse it. It is extremely persistent in soil and water and on most surfaces. It is also soluble in propylene glycol, DMSO, and other solvents. Agent 15 presumably shares many of the physiochemical properties of BZ.

Fentanyl derivatives (carfentanil). At standard room temperature, fentanyl and carfentanyl exist in liquid form that, while difficult, could potentially be aerosolized. Some surviving hostages near the release area reported seeing a bluish gray cloud that left a

sweet taste in their mouths, which could indicate the addition of halothane, a halogenated anesthetic, to the gas. Halothane was also in the blood of two German hostages who were taken to German hospitals. Halothane is a liquid at standard temperature and pressure that volatilizes rapidly. Other hostages reported that they did not recall seeing any gas with a distinctive taste, odor, or color before passing out. The vapors of the agent used in Moscow quickly dissipated in the open air once the theater was ventilated by opening doors and breaking windows.

DETECTION AND PROTECTION

BZ. Because BZ is odorless and nonirritating, and because clinical effects are not seen until after a latent period of 30 minutes to 24 hours, exposure could occur without the knowledge of casualties. No currently available field military or civilian detector is designed to disclose the presence of BZ or other anticholinergic compounds in the environment. Confirmation of the exact chemical involved in an incapacitating agent exposure would have to await laboratory analysis of environmental specimens containing the agent. The HEPA filter in the canister of the chemical protective mask prevents exposure of the face and respiratory tract to aerosolized BZ. The chemical protective ensemble protects the skin against contact with

BZ or other incapacitating agents dispersed as fine solid particles or in solution. Protection against ingestion would depend upon a high index of suspicion for BZ-contaminated food or drink.

Fentanyl derivatives (carfentanil). There are no chemical detectors for fentanyl derivatives and they have no odor, so they cannot be detected by smell. While the charcoal filter canister of a chemical protective mask should protect against concentrations in a ventilated environment, as they were worn by Special Forces personnel when first entering the theater, the filter effectiveness in an enclosed, unventilated space is unknown.

TOXICOKINETICS

BZ may be dispersed as an aerosolized collection of small particles. Alternately, it may be dissolved in a solvent such as DMSO to enhance percutaneous absorption. Bioavailability via ingestion and by inhalation of one-micron particles approximates 80%, and 40 to 50%, respectively, of a parenterally delivered dose of BZ. Percutaneous absorption of BZ dissolved in propylene glycol yields, after a latent period of up to 24 hours, serum levels approximately 5 to 10% of those achieved with intravenous or intramuscular administration. Although inhalation of aerosolized BZ is

probably the greatest risk on the battlefield, terrorists may choose to disseminate BZ in forms that provide significant opportunities for ingestion and absorption through the skin.

Following absorption, BZ is systemically distributed to most organs and tissues of the body. Its ability to reach synapses and neuromuscular and neuroglandular junctions throughout the body is responsible for its PNS effects, whereas its ability to cross the blood-brain barrier is responsible for its CNS effects. Atropine and hyoscyamine both cross the placenta and can be found in small quantities in breast milk; whether this is also true for BZ is unclear.

Metabolism of BZ would be expected to occur primarily in the liver, with elimination of unchanged agent and metabolites chiefly in the urine.

Fentanyl derivatives (carfentanil). Fentanyl and its derivatives are opioids, synthetic non-opium derived narcotics that perform like opium. They bind to opioid receptors in the CNS and the GI tract. The fentanyl derivative was released in the form of a gas in an enclosed space. It may have been combined with an inhalation anesthetic such as halothane. Once inhaled it is quickly delivered to the blood stream for distribution throughout the

body. While the gas incapacitated most of those in the theater within the first few minutes, there were some who were awake at the time of release, and away from the source of the release, and were able to flee the theater. In the CNS carfentanil binds selectively to mu opioid receptors, which are predominantly found in the gray regions of the brain and the spinal cord. This causes a depression in the function of the CNS. Activation of mu receptors by opioids causes loss of pain sensation, miosis, decreased intestinal paristalsis (constipation), nausea and vomiting, respiratory depression, and diminished mental alertness resulting in a feeling of drowsiness, euphoria, sleepiness, and unconsciousness. With increased dosing this can result in brain-mediated apnea. These products are eventually eliminated in the urine.

TOXICITY

BZ and other glycolates. The characteristic that makes BZ an incapacitating rather than a toxic chemical warfare agent is its high safety ratio. The amount required to produce effects is a thousand or more fold less than a fatal dose of the compound. In terms of Ct products (admittedly a sometimes problematic way of measuring dosage received after aerosol exposure), the ICt_{50} (the Ct product needed to produce incapacitation in 50% of an exposed group) for BZ is 112 mg-min/m^3, whereas the

LCt_{50} is estimated to be 200,000 mg-min/m^3.

Fentanyl derivatives (carfentanil). The effective dose (ED_{50}), the amount needed to cause incapacitation, for carfentanil, a very potent fentanyl derivative and a likely candidate for the agent used in Moscow, is 0.0034 mg/kg while the lethal dose (LD_{50}) is 3.4 mg/kg. This gives carfentanil the relatively wide safety index of 10,000, making it a good choice as an incapacitant. Two German hostages taken to Germany for treatment tested positive for halothane, but it is believed that one of these patients received halothane during life-saving procedures. Therefore, fentanyl could have been mixed with the halogenated anesthetic halothane to make it more potent.

TOXICODYNAMICS (MECHANISM OF ACTION)

BZ and other glycolates. The agent BZ and other anticholinergic glycolates act as competitive inhibitors of the neurotransmitter acetylcholine neurons (1) at postjunctional muscarinic receptors in cardiac and smooth muscle and in exocrine (ducted) glands and (2) at postsynaptic receptors in neurons. As the concentration of BZ at these sites increases, the proportion of receptors available for binding to acetylcholine decreases and the end organ "sees" less acetylcholine. (One way of

visualizing this process is to imagine BZ coating the surface of the end organ and preventing acetylcholine from reaching its receptors.) Because BZ has little to no agonist activity with respect to acetylcholine, high concentrations of BZ essentially substitute a "dud" for acetylcholine at these sites and lead to clinical effects reflective of understimulation of end organs.

Fentanyl derivatives (carfentanil). These analogs act to bind to mu opioid receptors found throughout the brain (hippocampus, cerebral cortex, periaqueductal grey and thalamus) and spinal cord (dorsal horn). When the binding occurs the receptors produce effects such as analgesia, miosis, euphoria, constipation, and respiratory depression.

CLINICAL EFFECTS

BZ and other glycolates:

Peripheral Effects

- Mydriasis, blurred vision
- Dry mouth, skin
- Initially rapid heart rate; later, normal or slow heart rate
- Possible atropine flush

The PNS effects of BZ are, in general, readily understood as those of understimulation

of end organs and are qualitatively similar to those of atropine. Decreased stimulation of eccrine and apocrine sweat glands in the skin results in dry skin (an affected patient can be "dry as a bone") and a reduction in the ability to dissipate heat by evaporative cooling. The skin becomes warm ("hot as a hare") partly from decreased sweating and partly from compensatory cutaneous vasodilatation (the patient becomes "red as a beet," with a so-called atropine flush) as the body attempts to shunt a higher proportion of core-temperature blood as close as possible to the surface of the skin. With decreased heat loss, the core temperature itself rises.

Understimulation of other exocrine glands leads to xerostomia (dry mouth, another way in which the patient is "dry as a bone"), thirst, and decreased secretions from lacrimal, nasal, bronchial, and gastrointestinal glands.

Decreased cholinergic stimulation of pupillary sphincter muscles allows alpha-adrenergically innervated pupillary dilating muscles to act essentially unopposed, resulting in mydriasis. (In fact, the cosmetic effect of mydriasis in women who applied extracts of deadly nightshade topically to their eyes explains the name "belladonna" ["beautiful lady"] given to this plant.) Similar effects on cholinergic ciliary muscles produce paralysis of

accommodation. Classically, the patient is described as being "blind as a bat." Other smooth muscle effects from BZ intoxication include decreased bladder tone and decreased urinary force with possibly severe bladder distention (yet another way in which the patient may be said to be "dry as a bone").

BZ typically raises the heart rate initially, but hours later, depending on the dose of BZ, the heart rate falls to normal or may become slow. Either the peripheral vagal blockade has ceased, or the stimulation of the vagal nucleus has occurred.

Neither atropine nor BZ can act directly at the postjunctional nicotinic receptors found in skeletal muscle, but BZ-exposed patients nonetheless exhibit muscle weakness. This weakness, along with incoordination, heightened stretch reflexes, and ataxia, is probably due to the effects of BZ at CNS sites.

The PNS effects of BZ are essentially side effects that are useful in diagnosis, but incidental to the CNS effects for which the incapacitating agents were developed. These CNS effects include a dose-dependent decrease in the level of consciousness, beginning with drowsiness and progressing through sedation to stupor and coma. The patient is often disoriented to time and place. Disturbances in judgment and insight

appear. The patient may abandon socially imposed restraints and resort to vulgar and inappropriate behavior. Perceptual clues may no longer be readily interpretable, and the patient is easily distracted and may have memory loss, most notably short-term memory. In the face of these deficits, the patient still tries to make sense of his environment and will not hesitate to make up answers on the spot to questions that confuse him. Speech becomes slurred and often senseless, and loss of inflection produces a flat, monotonous voice. References become concrete and semiautomatic with colloquialisms, clichés, profanity, and perseveration. Handwriting also deteriorates. Semiautomatic behavior may also include disrobing (perhaps partly because of increased body temperature), mumbling, and phantom behaviors such as constant picking, plucking, or grasping motions ("woolgathering" or carphology).

Central Effects

- Disturbances in level of consciousness
- Misperceptions and difficulty in interpretation (delusions, hallucinations)
- Poor judgment and insight (denial of illness)
- Short attention span, distractibility, impaired memory (particularly recent)
- Slurred speech, perseveration
- Disorientation

- Ataxia
- Variability (quiet/restless)

Central nervous system mediated perceptual disturbances in BZ poisoning include both illusions (misidentification of real objects) and hallucinations (the perception of objects or attributes that have no objective reality). (Although the phrase "mad as a hatter" refers to poisoning from mercury formerly used by hatters on felt, it can just as well serve as a reminder of CNS effects from anticholinergics.) Anticholinergic hallucinations differ from the often vague, ineffable, and often transcendent-appearing hallucinations induced by hallucinogenic indoles such as LSD. Hallucinations from BZ tend to be realistic, distinct, easily identifiable (often commonly encountered objects or persons), and panoramic. They also have the tendency to decrease in size during the course of the intoxication.

Another prominent CNS finding in BZ poisoning is behavioral lability, with patients swinging back and forth between quiet confusion and self-absorption in hallucinations to frank combativeness. Moreover, as other symptoms begin to resolve, intermittent paranoia may be seen. Automatic behaviors common during resolution include the crawling or climbing

motions called "progresso obstinato" in old descriptions of dementia.

BZ produces effects not just in individuals, but also in groups. Sharing of illusions and hallucinations (*folie à deux*, *folie en famille*, and "mass hysteria") is exemplified by two BZ-intoxicated individuals who would take turns smoking an imaginary cigarette clearly visible to both of them but to no one else.

Fentanyl derivatives (carfentanil). Effects of the gas agent used in the Moscow theater incident were reportedly almost immediate for those near the area of the release. For others there was a period of a feeling of drowsiness which quickly transitioned to unconsciousness. Some not near the release were less affected and showed drowsiness and ataxia, but were able to escape the area. Some of those who were unconscious vomited while sleeping, which caused their airway to be blocked. They also slumped in the theater seats, which placed their airways in restricted positions and blocked them. Those exposed to lethal doses of the agent who continued to breathe it in after becoming unconscious could also have experienced centrally-mediated respiratory depression and finally, apnea.

The general effects of opioids are that they are analgesic and sedative; they commonly

cause euphoria, dysphoria, and drowsiness. They will depress the central respiratory centers, constrict the pupils, and depress the cough reflex. Effective doses of fentanyl will stimulate the chemoreceptor trigger zone that causes nausea and vomiting. Opioids increase smooth muscle tone and therefore slow gastrointestinal propulsion. The effect on urinary smooth muscle is variable, so urinary urgency may increase or decrease.

Halothane is a halogenated hydrocarbon that causes general anesthesia, with a much reduced analgesic effect compared to fentanyl. It causes cerebral vasodilatation, increasing cerebral blood flow, but decreases cerebral consumption of oxygen. There is a decrease in skeletal muscle tone throughout the body, decreased salivation and gastric motility and like fentanyl, respiratory depression, which may produce a decreased tidal volume but increased respiratory rate. It causes the bronchial smooth muscles to dilate. Myocardial contractility and cardiac output also decrease due to vagal stimulation. This causes a drop in mean arterial pressure. It can trigger myocardial dysrhythmias, especially when hypoxia is present.

TIME COURSE OF EFFECTS

BZ and other glycolates.

Clinical effects from ingestion or inhalation of BZ appear after an asymptomatic or latent period that may be as little as 30 minutes or as long as 20 hours; the usual range is 0.5 to 4 hours, with a mean of 2 hours. However, effects may not appear up to 36 hours after skin exposure to BZ.

Once effects appear, their duration is typically 72 to 96 hours and dose-dependent. Following an ICt_{50} of BZ, severe effects may last 36 hours, but mild effects may persist for an additional day.

The clinical course from BZ poisoning can be divided into the following four stages:

1. Onset or induction (zero to four hours after exposure), characterized by parasympathetic blockade and mild CNS effects.
2. Second phase (4 to 20 hours after exposure), characterized by stupor with ataxia and hyperthermia.
3. Third phase (20 to 96 hours after exposure), in which full-blown delirium is seen but often fluctuates from moment to moment.
4. Fourth phase, or resolution, characterized by paranoia, deep sleep,

reawakening, crawling or climbing automatisms, and eventual reorientation.

Fentanyl derivatives (carfentanyl). Immediate unconsciousness given an effective dose.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for irrational and confused patients is a long one and includes anxiety reactions as well as intoxication with a variety of agents, to include hallucinogenic indoles (such as LSD), cannabinoids (such as the delta-9-tetrahydrocannabinol in marijuana), lead, barbiturates, and bromides. All of these conditions can lead to restlessness, lightheadedness (with associated vertigo and ataxia), confusion, and erratic behavior with or without vomiting. Clues that specifically point to BZ or a related compound are the combination of anticholinergic PNS effects ("dry as a bone," "hot as a hare," "red as a beet," and "blind as a bat") with the CNS effects ("mad as a hatter") of slurred and monotonous speech, automatic behavior (perseveration, disrobing, and phantom behaviors ["woolgathering"]), and vivid, realistic, describable hallucinations (decreasing in size over time) in a patient slipping into and out of delirium.

SIGNS AND SYMPTOMS	POSSIBLE ETIOLOGY
Restlessness, dizziness, or giddiness; failure to obey orders, confusion, erratic behavior; stumbling or staggering; vomiting.	Anticholinergics (e.g., BZ) indoles (e.g., LSD), cannabinols (e.g. marijuana), anxiety reaction, other intoxications (e.g., alcohol, bromides, barbiturates, lead).
Dryness of mouth, tachycardia at rest, elevated temperature, flushing of face; blurred vision, pupillary dilation; slurred or nonsensical speech; hallucinatory behavior; disrobing; mumbling and picking behavior; stupor and coma.	Anticholinergics
Inappropriate smiling or laughter, irrational fear, distractibility, difficulty expressing self, perceptual distortions; labile increase in pupil size, heart rate, blood pressure. Stomach cramps and vomiting may occur.	Indoles (Schizophrenic psychosis may mimic in some respects.)

Euphoric, relaxed, unconcerned daydreaming attitude, easy laughter; hypotension and dizziness on sudden standing.	Cannabinols
Tremor, clinging or pleading, crying; clear answers, decrease in disturbance with reassurance; history of nervousness or immaturity, phobias.	Anxiety reaction
Sleepiness, ataxia, rapid unconscious, miosis, reduced quality of respirations decrease with resulting respiratory depression.	Fentanyl (carfentanyl)

Atropine intoxication from MARK I autoinjector use in a patient not exposed to nerve agents may create similar PNS effects to those seen in BZ intoxication. However, marked confusion from atropine is not normally seen until a total of six or seven autoinjectors have been given (in a hot, dehydrated, or battle-stressed individual, less atropine would probably suffice). Circumstantial evidence may be helpful in this situation. Heat stroke may also generate hot, dry, and confused or stuporous casualties and needs to be considered in the differential

diagnosis. Patients with anxiety reactions are usually oriented to time, place, and person but may be trembling, crying, or otherwise panicked. The classic picture of unconcern, or "la belle indifférence," may characterize a patient with a conversion reaction, but these patients are also likely to be oriented and lack the anticholinergic PNS signs of BZ poisoning.

MEDICAL MANAGEMENT

Guidelines for general patient care are not intended to take the place of sound clinical judgment, especially in the management of complicated cases.

BZ and other glycolates. The admonition to protect oneself first may be difficult when dealing with any intoxication involving a latent period, since initially asymptomatic exposure to health care providers may already have occurred during the same time frame in which patients were exposed. Protection of medical staff from already absorbed and systemically distributed BZ in a patient is not needed.

General supportive management of the patient includes decontamination of skin and clothing (ineffective for already absorbed agent but useful in preventing further absorption of any agent still in contact with the patient), confiscation of weapons and related items from

the patient, and observation. Physical restraint may be required in moderately to severely affected patients. The greatest risks to the patient's life are (1) injuries from his or her own erratic behavior (or from the behavior of similarly intoxicated patients) and (2) hyperthermia, especially in patients who are in hot or humid environments or are dehydrated from overexertion or insufficient water intake. A severely exposed patient might be comatose with serious cardiac arrhythmias and electrolyte disturbances. Management of heat stress assumes a high priority in these patients. Because of the prolonged time course in BZ poisoning, consideration should always be given to evacuation to a higher care level.

As a competitive inhibitor of acetylcholine, BZ effectively decreases the amount of acetylcholine "seen" by postsynaptic and postjunctional receptors throughout the body. Specific antidotal therapy in BZ poisoning is therefore geared toward raising the concentration of acetylcholine in these synapses and junctions. Any compound that causes a rise in acetylcholine concentration can potentially overcome BZ-induced inhibition and restore normal functioning; even the nerve agent VX has been shown to be effective when given under carefully controlled conditions. The specific antidote of choice in BZ poisoning is the carbamate anticholinesterase physostigmine

(eserine; Antilirium®), which temporarily raises acetylcholine concentrations by binding reversibly to anticholinesterase on the postsynaptic or postjunctional membrane. Physostigmine is similar in many ways to pyridostigmine and is equally effective when used as a pre-exposure antidotal enhancer ("pretreatment") in individuals at high risk for subsequently encountering soman. However, physostigmine is not used for this purpose because the doses required cause vomiting through CNS mechanisms. In the case of BZ poisoning, a nonpolar compound such as physostigmine is used specifically because penetration into the brain is required in those individuals who already have CNS effects from BZ.

In BZ-intoxicated patients, physostigmine is minimally effective during the first four hours after exposure but is very effective after four hours. Oral dosing generally requires one and a half times the amount of antidote as does IM or IV administration. However, effects from a single intramuscular injection of physostigmine last only about 60 minutes, necessitating frequent re-dosing. It must be emphasized that physostigmine does not shorten the clinical course of BZ poisoning and that relapses will occur if treatment is discontinued prematurely. The temptation to substitute a slow intravenous infusion for intramuscular injections should be

tempered by the awareness that IV infusion may lead to nerve-agent-like bradycardia, and too rapid infusion might cause arrhythmias, excessive secretions (to the point of compromising air exchange), and convulsions. Moreover, the sodium bisulfite in commercially available preparations of physostigmine may cause life-threatening allergic responses.

Suggested dosages for physostigmine in the treatment of BZ poisoning follow:

Test dose. If the diagnosis is in doubt, a dose of 1 mg might be given. If a slight improvement occurs, routine dosing should be given.

Routine dosing. Adult doses of about 45 mcg/kg have been recommended. This might be modified by the response. A mental status examination should be done every hour, and the dose and time interval of dosing should be modified according to whether the mental status is improved or not. As the patient improves, the dosage requirement will decrease. Oral dosing is the preferred route after the initial IV dose. This will decrease the risk of overdose that could be created by further IV administration.

Routes of administration.

IM: 45 mcg/kg in adults (20 mg/kg in children)

IV: 30 mcg/kg slowly (1 mg/min)

PO: 60 mcg/kg if patient is cooperative (because of bitter taste, consider diluting in juice)

For each route, titrate about every 60 minutes to mental status.

Fentanyl derivatives (carfentanil). It is critical to assess the patient's respiration and insure that his airway is not obstructed. A quick check of the patient's head and body should also be conducted to insure that he did not bump his head or other parts of his body on the floor or on objects if he collapsed quickly. Check for airway obstruction from vomitous and clear the airway and position the patient on his side. Administer the antidote, naloxone, and check for improved respiration. Even after the administration of the antidote, the patient's breathing must be checked periodically, as naloxone effects are known to be short-lived with a potent opioid such as carfentanil. Pre-existing medical conditions may also interfere with breathing, which could be exacerbated as fentanyl derivatives tend to depress respiration.

The individual who has aspirated vomit may be at risk for aspiration pneumonia, so a follow-up chest radiograph may be indicated. A CT head scan should be ordered if a fall injury to the head is suspected. Patients with preexisting cardiac disease may be at risk for cardiac ischemia, so an ECG should be ordered for these patients. Urine drug screenings must specifically detect for fentanyl and carfentanyl as they will not be identified by morphine screens. Other precautionary lab tests to assess rhabdomyolysis, electrolytes, renal and liver function and clotting are appropriate to assess clearance and general status. If the agent is unknown, then it is wise to collect additional blood and urine samples for testing to help rule out other analgesics, sedatives, or toxins.

The antidotes for opioids, naloxone hydrochloride (Narcan), or naltrexone have long been used to treat heroine addicts. These are opioid receptor antagonists that bind to the opioid receptors more strongly than a fentanyl derivative, but do not activate the receptor. As these replace the opioid on the opioid receptor the signs and symptoms are quickly reversed, especially life-threatening respiratory depression. It is important to remember that these have a relatively short half life, so symptoms may return in an apparently stabilized patient. This means that the patient, though

recovering, must be monitored closely and airway support maintained until full consciousness is achieved. If the agent is composed of multiple substances, then naloxone, an opioid inhibitor, may demonstrate only limited effectiveness. The administration of naloxone can be diagnostic as well as therapeutic. The dosage guidelines noted here are for those with opioid dependency. Some individuals may be hypersensitive to naloxone; otherwise there are no life-threatening side effects with normal dosing. When uncertain of the agent, naloxone should be titrated slowly and the patient observed closely for change.

The adult dose is 0.4 to 2 mg IV, repeated every 2 to 3 minutes as needed for stabilization of breathing. IV administration will bring the quickest improvement. If injected either IM or subcutaneously, increments of 0.1 to 0.2 mg are recommended at 2- to 3-minute intervals until adequate patient ventilation is achieved. Larger than suggested dosage may result in increased blood pressure, sweating, and vomiting. Dosages may need to be repeated every 20 to 60 minutes after initial stabilization if signs return. Supplemental IM doses may have longer effect. If no response is noted after 10 mg of naloxone have been administered, the toxic agent should be questioned.

The pediatric dose is 0.1 mg/kg of body weight if given IV. If this dose does not allow for adequate patient ventilation, a follow-on dose of 0.1 mg/kg of body weight can be administered. If IV is not available, it can also be administered IM or subcutaneously. Titrate to respiratory stabilization.

HISTORY AND TOXICITY OF THE BZ ANTIDOTE PHYSOSTIGMINE

The antagonism between physostigmine (the elixir or calabar bean) and atropine (tincture of belladonna) was first reported in 1864 by a physician who successfully treated prisoners who had become delirious after drinking tincture of belladonna. Physicians did not notice this report until the 1950s when atropine coma (in which 50 mg or so of atropine were given to certain psychiatric patients) was successfully treated with physostigmine after the "therapeutic benefit" had been attained. Again, this went unnoticed until a controlled study reported in 1967 indicated that anticholinergic intoxication could be successfully, albeit transiently, reversed by physostigmine.

A recent textbook of emergency medicine stated that physostigmine should be used only as "a last resort." It would seem that when a patient needs "last resort" care is the absolute

wrong time to administer a potent cholinesterase inhibitor.

The administration of physostigmine by the IV route in a delirious but conscious and otherwise healthy patient is not without peril. It is sometimes difficult to keep a delirious patient quiet long enough to administer the drug (at 1 mg/min is the marketed solution of 1 mg/ml). Even if administered correctly (very slowly), the heart rate may decline from 110 bpm to 45 bpm over a period of 1 to 2 minutes. The difference in the onset of the effects after IM and IV administration of physostigmine is a matter of only several minutes. Since its use is rarely lifesaving, this slight difference in time of response is inconsequential.

Physostigmine is a safe and effective antidote if used properly. In a conscious and delirious patient it will produce very effective but transient reversal of both the peripheral and central effects of cholinergic blocking compounds. Its use by the IV route is not without hazards. It absolutely should NOT be used in a patient with cardiorespiratory compromise, hypoxia, or acid-base imbalance with a history of seizure disorders or arrhythmias.

BZ and other glycolates.

An **immediate** casualty (possible, but unlikely) would be one with cardiorespiratory compromise or severe hyperthermia. Immediate attention to ventilation, hemodynamic status, and temperature control could be lifesaving. Because of its dangers in a hypoxic or hemodynamically challenged patient, physostigmine should be considered a second-line management option to be used only if adequate attention can simultaneously be given to temperature and other vital signs.

The **delayed** casualty would present with pronounced or worsening anticholinergic signs. Physostigmine should definitely be considered in this kind of patient.

A **minimal** casualty from a strictly medical standpoint might have mild PNS or CNS anticholinergic effects. Given the time course of BZ intoxication, however, these patients should not be considered able to manage themselves or capable of routine return to duty and should be relieved of their weapons, observed, and if the holding capacity at the current echelon is exceeded, evacuated.

An **expectant** casualty (also possible, but unlikely) would have severe cardiorespiratory compromise in a situation in which treatment or evacuation resources are too limited to allow the necessary attention to be directed to him or her.

Fentanyl derivatives (carfentanyl).

An **immediate** casualty would be one with cardiorespiratory compromise due to central apnea or airway obstruction. Immediate attention to maintenance of the airway, supplemental oxygen and ventilatory support, and administration of antidotes will be lifesaving. These patients should be considered a priority for rapid evacuation to a facility with respiratory care assets if their condition does not improve with the administration of antidotes and supplemental oxygen at the incident scene.

The **delayed** casualty would present with independent breathing that could be compromised if the patient vomits or central dyspnea increases. Respiration needs to be monitored closely. This patient must be placed in a side-lying position to help maintain a clear airway. Supplemental oxygen and administration of antidotes should be considered for this patient.

A **minimal** casualty might be an individual who has walked out of the contaminated area

unassisted and is able to converse, but may be somewhat drowsy. In these patients breathing is not restricted. They should be placed in an area where they can be observed for 24 hours before being released.

An **expectant** casualty (also possible, but unlikely) would have severe cardiorespiratory compromise in a situation in which antidotal treatment, respiratory assistance, and evacuation resources are too limited to allow the necessary attention to be directed to him or her.

RETURN TO DUTY

BZ and other glycolates. Given the time course of the intoxication, early return to duty is probably not a realistic possibility for the majority of casualties who may require observation and management for several days at the least.

Fentanyl derivatives (carfentanil). Those with mild to moderate symptoms should be able to return to duty within 24 hours. Those who require ventilatory support will require longer periods of hospitalization until cardiopulmonary function is normalized, barring any complications such as pulmonary infection or brain damage from hypoxia following periods of sustained apnea.

RIOT-CONTROL AGENTS
CS, CN, CR, DM, and OC

SUMMARY

Signs and Symptoms: Burning and pain on exposed mucous membranes and skin, eye pain and tearing, burning in the nostrils, respiratory discomfort, and tingling of the exposed skin. **DM:** Will cause prolonged periods of vomiting and a feeling of malaise.

Field Detection: No field detector is available for any of the RC Agents.

Field Decontamination: **Eyes:** Thoroughly flush with water, saline, or similar substance. **CS, CN, CR, DM: Skin:** Flush with copious amounts of water, soap and water, or a mildly alkaline solution (sodium bicarbonate or sodium carbonate). Generally, decontamination is not needed if the wind is brisk. **OC:** The pain from OC will increase with water, especially warm water. It is best decontaminated with baby shampoo, milk, alcohol, or vegetable oil. Without decontamination pain will subside over time.

Immediate management: Usually none is necessary; effects are self-limiting and diminish or cease within 45 minutes. DM is the exception, where effects may last several hours.

OVERVIEW

Riot-control agents, also called irritants, lacrimators, and "tear gas," produce transient discomfort and eye closure to render the recipient temporarily incapable of fighting or resisting. Law enforcement agencies use them for riot control, and military forces use them for training and in combat (see below). They have a high LCt_{50} and a low effective Ct_{50}, and therefore have a high safety ratio. Their major activity is to cause pain, burning, or discomfort on exposed mucous membranes and skin; these effects occur within seconds of exposure but seldom persist more than a few minutes after exposure has ended.

HISTORY/MILITARY RELEVANCE

Paris police used riot-control agents to dispel rioters before WWI, and these compounds were the first chemical agents deployed during that war. French warfighters used them with limited success in small skirmishes. About 30 riot-control agents were developed and used, but their use decreased following the advent of more potent compounds.

After WWI, military and law enforcement agencies used CN for various purposes until CS,

a more potent and less toxic compound, synthesized by Corson and Stoughton (hence the nomenclature) in 1928, replaced it in about 1959. Today CN is in commercially available devices for self-protection (Mace®), but CS and oleoresin capsicum (OC) are fast becoming the favorites among law enforcement agencies. The military forces of most countries use CS in training as a confidence builder for the protective mask (the "gas chamber exercise"), and the U.S. used it extensively in Vietnam primarily for tunnel denial. Worldwide, police forces of many countries, e.g., Ireland, France, Russia, and the U.S., use it for crowd control or during riots.

Introduced in 1974 in the U.S., oleoresin capsaicin (OC) is quickly replacing other riot control agents used in law enforcement across the U.S. because of its safety and the fact that it can be dispensed as a liquid, foam, aerosol, or as a powder in paint ball delivery systems. It is best used for individual protection. Derived from the resin of cayenne peppers, it has been used in various ways. In small amounts it is used for pain relief; in larger amounts it is used as an irritant and as a tool of torture. Examples include its use historically by the Japanese police and the ninja, in the form of a powder, metsubishi, blown on an attacker, and as an irritant during the Indo-China wars of 2,000 B.C. The U.S. excludes these agents from international treaty provisions. They may be used in military

situations by Presidential Order. OC can be used by military law enforcement.

The agents in use today are OC, CS, and CN. CA is a very old and outmoded riot-control agent because of its low safety margin and caustic nature, so it will not be discussed here; CR is a British agent similar to CS, and DM (Adamsite) is an irritant as well as a vomiting agent. While OC, CS, and CN are used in the U.S., CA and DM are not, but may be used by other nations for riot control.

PHYSIOCHEMICAL CHARACTERISTICS

Unlike most agents, which are liquids under temperate conditions, the riot-control agents CS, CN, CR, and DM are crystallized solids with low vapor pressures and are dispersed as fine particles or in solution. Dispersion devices include small, handheld spray cans, large spray tanks, grenades, and larger weapons. OC is an oily resin, which can be dried to form an off-white powder and aerosolized, or combined with a medium such as alcohol or oil to make a liquid or foam spray.

MECHANISM OF TOXICITY

The mechanism of biological activity is less well characterized for riot-control agents than for most other agents. Fortunately, a detailed

knowledge of the mechanism of action is not necessary for appropriate medical management.

CS and CN are SN_2 alkylating agents (mustard, in contrast, is an SN_1 alkylator) and react readily at nucleophilic sites. Prime targets include sulfhydryl-containing enzymes such as lactic dehydrogenase. In particular, CS reacts rapidly with the disulfhydryl form of lipoic acid, a coenzyme in the pyruvate decarboxylase system. It has been suggested that tissue injury may be related to inactivation of certain of these enzyme systems.

Pain can occur without tissue injury and may be bradykinin mediated. CS causes bradykinin release *in vivo* and *in vitro*, and elimination of bradykininogen *in vivo* abolishes the systemic response to CS.

The initial response to aerosolized CS is an increase in blood pressure and irregular respiration, suggestive of the Sherrington pseudoaffective response. Bypassing the pain receptors of the nose and upper airway by endotracheal administration of CS leads to the same decrease in blood pressure and in respiration seen after IV injection. This suggests that the initial pressor effect and irregular respiration are responses to a noxious stimulus rather than pharmacological effects of CS.

OC produces a burning and painful sensation in the individual it contacts as it binds to ion-channel receptors in the nervous system resulting in rapid cell depolarization and a massive release of substance P, a neuropeptide that is a neurotransmitter of pain. This release of high levels of substance P signals the brain that the body is experiencing extreme pain and burning. This continues until the substance P is used up. The body then releases endorphins to inhibit the pain sensation, which can create a period of euphoria, a “runners high” as an after effect for the individual exposed. Those who regularly eat hot peppers are less susceptible to the painful effects of pepper spray. The use of capsaicin in pain relieving ointments causes a slower release and depletion of substance P, creating an analgesic effect without causing the extreme pain sensation experienced with OC exposure.

CLINICAL EFFECTS

The main effects of riot-control agents are pain, burning, and irritation of exposed mucous membranes and skin. These effects do not differ appreciably from one agent to another except in the case of DM, which will be discussed in a separate section.

Eyes. The eye is the organ most sensitive to riot-control agents. Contact with any of the riot-

control agents produces a sensation of conjunctival and corneal burning and leads to tearing, blepharospasm, and conjunctival injection. The severe blepharospasm causes the lids to close tightly and produces transient "blindness," an effect that could inhibit the recipient's ability to fight or resist. However, if the recipient opens his eyes, his vision is near normal even if a significant concentration of the agent persists.

Because these compounds are solids, with the exception of OC, it is possible for a particle or clump to become embedded in the cornea or conjunctiva to cause tissue damage. With the caveat noted below, there is no evidence that this complication has ever occurred; however, a recipient seeking medical care for eye pain after exposure should have his eyes thoroughly decontaminated and undergo thorough ophthalmic examination. It could be necessary to pick out the particles of agent from tissue. The carriers' solvents in some OC sprays can cause corneal erosion, so eyes should be decontaminated with copious amounts of water or baby shampoo and water. Follow-up with an ophthalmologist is indicated if complications appear.

Reviewers examined the evidence for permanent eye damage from riot-control agents. In each instance, the damage was from a

weapon fired from close range (about 50% were self-inflicted). The reviewers concluded that the blast force driving the agent deep into tissue (with or without the wadding of the weapon) was the major cause of permanent injuries. This should not happen under normal use.

Nose and mouth. Contact with the delicate mucous membranes of the nose produces a burning sensation, rhinorrhea, and sneezing; a similar burning sensation accompanied by increased salivation occurs after contact with the mouth.

Airways. Inhalation causes burning and irritation of the airways with bronchorrhea, coughing, and a perception of a "tight chest" or an inability to breathe. In research studies, pulmonary function studies done immediately after exposure have shown minimal alterations. In one reported instance, nine Marines who were without respiratory protection were exposed to high CS concentrations during training and developed transient pulmonary syndrome. All had coughs and shortness of breath, five had hemoptysis, and four who needed to be hospitalized had hypoxia. It was discovered that those hospitalized underwent strenuous physical exercise within 36 to 84 hours after exposure to high amounts of CS. One week after exposure all nine had normal

lung function measured by spirometry before and after exercise.

An inhaled irritating compound might be expected to exacerbate a chronic pulmonary disease such as asthma, emphysema, or bronchitis, but this appears not to happen after CS or CN, even though these agents have been used widely in mixed populations. The medical care provider should nevertheless anticipate airway problems in individuals with lung disease, particularly if they are exposed to higher than the average field use concentrations. The onset of extreme pain for those exposed to OC has resulted in a few cases of laryngospasm as a reaction to the pain caused by the OC. These cases have responded well with standard airway management.

There is no evidence that CS causes permanent lung damage after one or several exposures to field concentrations. Following inhalation of lethal amounts, animals died from severe airway damage 12 to 24 hours post-exposure, but survivors from large exposures had minimal or no pulmonary abnormalities. After multiple (50 or more) daily exposures to smaller amounts, animals developed laryngitis and tracheitis.

Skin. Contact with the skin causes a tingling or burning sensation and may cause erythema,

particularly if the skin is raw or freshly abraded (e.g., shortly after shaving). The erythema begins several minutes after exposure and generally subsides 45 to 60 minutes after termination of exposure.

Under conditions of high temperature, high humidity, and high concentration of agents such as CS, there may be more severe dermatitis starting with erythema hours after exposure and followed by vesication. Generally these are second-degree burns not unlike, but more severe than sunburn. Firemen who entered buildings contaminated with CS after summer riots several decades ago developed these lesions. After stirring up the contaminating particles, they later developed erythema and blisters on their exposed skin. Hypersensitivity may develop for those who have reacted to CS in the past. In one instance, an individual developed generalized vesication and high fever after an uneventful exposure to CS more than 20 years after his only and equally uneventful previous exposure. OC will cause skin redness and irritation and with prolonged exposure to high concentrations can cause skin rash, but not the blistering seen with CS or CN.

GI tract. Gastrointestinal effects usually do not occur with most riot-control agents (DM is an exception), although there may be retching or vomiting if the agent concentration is high,

exposure is prolonged, or the individual is sensitive.

Cardiovascular. A transient increase in heart rate and blood pressure has occurred in people immediately prior to an exposure to all of these riot-control agents or immediately after onset of exposure. The heart rate and blood pressure returned essentially to pre-test ranges while exposure continued and may have been caused by the anxiety or the initial pain rather than a pharmacological effect of these agents. This "alarm reaction" may cause adverse effects in one with pre-existing cardiovascular disease.

Oral ingestion. Taking large amounts of capsicum orally can increase the production of stomach acid, but this would not be caused by ingestion of minute amounts of OC from a spray or foam. Children occasionally eat CS, and several adults have swallowed CS pellets. Aside from bouts of diarrhea and abdominal cramps (which might have been from the cathartics and antacids used as therapy), their courses have been uneventful. In animals, the LD_{50} for CS is about 200 mg/kg (which is about 14 grams/70-kg person), an amount unlikely to be ingested, even deliberately. A few animals fed lethal amounts (or greater) of CS had gastric irritation or erosions, and several had signs of intestinal perforation. Recommended therapy after

ingestion consists of cathartics, antacids, and surgical observation.

Lethality. CN, occasionally in combination with DM, has caused deaths in people who refused to exit a confined space. In each case the agent was used in excess. Death generally occurred hours after initial exposure, and post-mortem findings were those of severe airway damage similar to that seen in animals. Deaths directly attributed to OC exposure are not easily found in the literature, though theoretically severe laryngospasm could cause respiratory obstruction, hypoxia, and unconsciousness. Those in police custody who died after exposure to OC died from pre-existing cardiopulmonary conditions, drug intoxication, or positioning while restrained that restricted breathing, but no deaths were directly attributed to the actions of OC.

Metabolism. Animals given lethal amounts of CS by IV or intraperitoneal (IP) administration developed increased blood thiocyanate concentrations hours later, indicating that the malononitrile portion of CS had been metabolized to cyanide. Cyanide was not a factor in causing death (lung damage was). A significant increase in blood concentration of thiocyanate has not been noted after aerosol administration of CS. Several popular databases mention this cyanogenic potential of CS and

suggest that treatment of a CS casualty might require therapy for cyanide poisoning (this recommendation is apparently based on the IV or IP administration data). After receiving lethal amounts of CS by inhalation, animals died 12 to 24 hours later from severe airway damage; cyanide was not implicated in their deaths.

OC will cause depletion of substance P over time; once substance P is depleted, pain stops. The body rebuilds substance P stores over several hours to days. OC is absorbed in the body. Those who are exposed to capsaicin for long periods of time, such as in food or topical salves for pain management, have depleted stores of substance P and may not demonstrate as extreme a pain reaction to the administration of OC.

ADAMSITE (DM)

DM typically appears as a canary yellow crystalline solid and has the same unique color when dispensed as a cloud. The effects of usual field concentrations of DM are similar to those of the other riot-control agents, except that DM has little irritancy to the skin. However, at higher concentrations, DM causes nausea, vomiting, and a feeling of generalized malaise. For this reason, it is called a vomiting agent.

TIME COURSE OF EFFECTS

Except for those produced by DM, the biological effects from these agents begin seconds after exposure and continue for 15 minutes or so after one exits the contamination to fresh, clean air. The effects from DM begin two to four minutes after the onset of exposure and may last an hour or two. (This is advantageous militarily, as an individual unaware of the agent will continue to inhale it for several minutes and absorb a larger dose. He may then vomit, requiring mask removal, which leads to continued inhalation of agent.)

DIFFERENTIAL DIAGNOSIS

Usually the circumstances of exposure make the diagnosis obvious. The history and the few physical signs (conjunctival injection with normal pupils, tearing, etc.) are usually adequate. On a battlefield, the sudden onset of burning pain and irritation might lead one to consider Lewisite or phosgene oxime exposure, but the signs and symptoms of riot-control agents gradually recede, whereas those from the vesicants worsen.

LABORATORY FINDINGS

There are no specific laboratory tests that will confirm the diagnosis. Complications, e.g., infection of a skin lesion, will produce the laboratory findings characteristic of the complication.

MEDICAL MANAGEMENT

The effects of exposure to these agents under the usual field conditions generally are self-limiting and require no specific therapy. Most will disappear in 15 to 30 minutes, although erythema may persist for an hour or longer.

The following section discusses potential complications occurring only under exceptional circumstances, such as exposure to a very large amount of agent (as in an enclosed space), exposure in adverse weather, or experimental studies in humans or animals. They are not to be expected with normal use of these agents.

Less than 1% of exposed people will have effects severe or prolonged enough to cause them to seek medical care. Those who do probably will have eye, airway, or skin complaints. Because there is no antidote for these agents, treatment consists of symptomatic management.

Eyes. The eye should be carefully flushed with water or saline and impacted particles should be sought. General care consists of a topical solution (many are available) to relieve the irritation and topical antibiotics. An ophthalmologist should be consulted for further evaluation and care. With exposure to OC, milk, vegetable oil, or baby shampoo and copious amounts of water can be effective for eye washing.

Pulmonary. These agents may exacerbate chronic disease or unmask latent disease, although there is little evidence of this. Bronchospasm with wheezing and mild distress continuing hours after exposure may occur in a latent asthmatic. More severe effects and respiratory distress may occur in one with chronic bronchitis or emphysema. Management includes oxygen administration (with assisted ventilation, if necessary), bronchodilators if bronchospasm is present, and specific antibiotics dictated by the results of sputum studies (Gram stains of smears followed by culture). A specialist skilled in the treatment of inhalation injury should be consulted early. Animal studies and very limited human data indicate that maximal effects occur 12 hours after exposure.

Skin. The early erythema requires reassurance, but no specific therapy unless

severe and prolonged more than an hour or two. The later onset erythema precipitated by a larger exposure in a hot and humid atmosphere is usually more severe and less likely to resolve quickly. It may require the use of soothing compounds such as calamine, camphor, and mentholated creams. Small vesicles should be left intact, but larger ones will ultimately break and should be drained. Irrigation of denuded areas several times a day should be followed by the application of a topical antibiotic. Large, oozing areas have responded to compresses containing substances such as colloidal oatmeal, Burrow's solution, and other dermatologic preparations.

DECONTAMINATION

The crystallized solids, CS, CN, CR, and DM, can be released from hair, skin, and clothing by flapping the arms or the use of fans. While many of these are not directly soluble in water, washing with soap and water will effectively remove these agents from the skin. Washing with soap and water is particularly important for those exposed to these agents for long periods of time, such as an individual operating a mask confidence chamber. Water will increase the pain of OC, so if it is used it should be with copious volumes to help remove the OC from the skin surface. A mild soap, such as baby shampoo, will help loosen resin, as will

vegetable oil or alcohol. Casein in milk will help to reduce the further release of substance P. Milk, with its lipophilic casein, will also effectively combine with the capsaicin resin to help wash it away.

TRIAGE

A person exposed to the usual field concentrations of riot-control agents will probably not be seen at a triage area. Those presenting with complications should be triaged according to the nature of their injuries.

RETURN TO DUTY

Because the effects of field concentrations clear within minutes, the casualty should be returned to duty as soon as possible. Casualties with complications may require evacuation and further medical treatment before returning to duty.

DECONTAMINATION

OVERVIEW

Decontamination is the reduction or removal of hazardous agents. Decontamination of chemical agents may be accomplished by removal of these agents by physical means or by chemical neutralization or detoxification.

There are three levels of patient decontamination: (1) immediate decontamination primarily protects the patient by reducing or eliminating the amount of chemical agent on the skin; (2) patient operational decontamination reduces the spread of contamination from the contaminated patient to "dirty" designated transport vehicles; and (3) patient thorough decontamination primarily protects the medical facility and its staff from cross contamination by agent on the patient's skin and clothing.

Prompt removal of an agent from the skin by any nontoxic means is the key to patient decontamination. Immediate decontamination by the warfighter can mean the difference between minor and significant medical effects from agent exposure.

The M291 Skin Decontamination Kit (SDK) or soap and water are currently the best decontaminants for skin. Reactive Skin Decontamination Lotion (RSDL), a liquid skin decontaminant, is expected to replace the M291 (SDK), a dry resin-based decontaminant, in the future.

INTRODUCTION

Decontamination is the process of removing or reducing a hazardous agent, whether chemical, biological, or radiological, from a person or object. Chemical contamination can present as a vapor, aerosol, liquid, or dry solid. The key reference for patient decontamination in the military is FM 4-02.7, Health Service Support in an NBC Environment.

Decontamination is not as critical for those contaminated only by vapor exposure, because vapor will continue to volatilize in the open air; rapidly brushing the hair if vapor is trapped in it and removing a patient's clothing where vapors can be trapped will usually remove the vapor hazard. Exposures to aerosols, liquid, and dry solids on the other hand will require more thorough decontamination if a patient is to enter a facility where staff are not in protective clothing.

In the military there are three levels of patient decontamination. (1) Immediate decontamination is performed by the exposed individual or a buddy if that individual is unable to decontaminate themselves. It reduces the dose of agent the patient receives by removing agent from the skin. It also reduces agent on equipment worn by the patient. (2) Patient operational decontamination is decontamination that is performed at the unit level to reduce the spread of contamination from a patient during evacuation in a “dirty vehicle.” Here the patient remains in full protective ensemble, and gross contamination is removed from the ensemble prior to transport. (3) Finally, patient thorough decontamination occurs prior to a contaminated patient being allowed into a medical treatment facility (MTF). A patient decontamination station (PDS) is usually established to perform this task. Here the patient’s equipment and clothing are removed and a more thorough decontamination of contaminated skin is performed.

Other techniques of decontamination include detailed troop decontamination (DTD), which is performed by units for those who are not casualties but need to exit the contaminated battlefield, and technical decontamination, which is the decontamination of PDS operators before they cross to the uncontaminated side of the PDS.

The most important decontamination to minimize injury to the patient is immediate decontamination since it reduces the patient's exposure to a toxic agent. It is most effective if performed within one or two minutes after exposure, particularly with HD, but the dose will still be reduced to some degree if decontamination is performed later than this. The key is to remove the agent from the skin with whatever means are available that are not toxic or abrasive to the skin.

Decontamination studies have been conducted using common household products. The goal of these studies was identification of decontaminants for civilians, as well as field expedients for the warfighters. Timely use of water, soap and water, or flour, followed by wet tissue wipes, produced results equal, nearly equal, or, in some instances, better than those produced by the use of Fuller's Earth, Dutch Powder, and other compounds. (Fuller's Earth and Dutch Powder are decontamination agents currently fielded by some European countries.) This is easily understood because (1) no topical decontaminant has ever shown efficacy with penetrated agent, (2) agents in large enough quantity, especially vesicants, may begin penetrating the skin before complete reactive decontamination (detoxification) takes place, and (3) early physical removal is most important.

Copious amounts of water or soap and water are effective for washing away most agents. A low pressure, high volume of water should be used combined with wiping of the skin. Fat-based soaps should be used and not detergents. The fat-based soaps, such as castile soap or other mild liquid soaps, help to emulsify thickened agents such as VX and HD.

Physical removal is imperative because none of the chemical means of destroying these agents does so instantaneously. While decontamination preparations such as fresh hypochlorite react rapidly with some agents (e.g., the half time for destruction of VX by hypochlorite at a pH of 10 is 1.5 minutes), the half times of destruction of other agents, such as mustard, are much longer. If a large amount of agent is present initially, a longer time is needed to completely neutralize the agent to a harmless substance.

The preferred methods of skin decontamination in the military are:

1. The use of the M291 Skin Decontamination Kit (SDK), which contains pads impregnated with Ambergard-555 Resin. As the pad is scrubbed over the contaminated skin, the chemicals are rapidly transferred into and trapped in the interior of the resin particles. The presence of acidic and basic groups in the resin

promotes the destruction of trapped chemical agents by acid and base hydrolysis. Because the resin is black, it maps out the areas that have been decontaminated. It is used on intact skin, but not in open wounds. Since the M291 Kit is small and dry and easily carried by the warfighter, it is well suited for field use. It will be the early intervention with the use of this kit that will reduce chemical injury and save lives in most cases. Decontamination of the casualty using an M291 Kit does not obviate the need for decontamination at a field facility.

2. Soap and water is a low-cost decontaminant that removes agents by washing them off the skin. It is effective for removing chemical, biological and radiological contaminants. It does not destroy biological agents or neutralize radioactive particles. Both fresh water and sea water have the capacity to remove chemical agents not only through mechanical force, but also via slow hydrolysis; however, the generally low solubility and slow rate of diffusion of chemical warfare agents in water significantly limit the agent hydrolysis rate. The predominant effect of water and water/soap solutions is the physical removal or dilution of agents; however, slow hydrolysis does occur particularly with alkaline soaps. Fat-based liquid soap (e.g. baby shampoo, castile liquid soap, or soft soap) attract and help emulsify chemical agent so that the action of the water can wash it

away. Detergents that can dry the skin should not be used. Clean water can be used to irrigate wounds; only copious amounts of water, normal saline, or eye solutions are recommended for the eye.

3. A 0.5% hypochlorite solution with an alkaline pH is an alternate skin decontaminant that can be used when the others are not available and water is limited. It is a solution of nine parts water to one part bleach which at this dilution is not harmful to the skin. It is wiped on the skin and can be rinsed off several minutes later with fresh water. This chemical decontamination reaction involves very slow oxidative chlorination and hydrolysis. The term oxidative chlorination covers the "active chlorine" chemicals like hypochlorite. Hypochlorite solutions act universally against the organophosphorus and mustard agents. Both VX and HD contain sulfur atoms that are readily subject to oxidation and hydrolysis. The decontamination effectiveness of these solutions increases as the hypochlorite pH levels go above 8; but this is harmful to the skin, so at the dilution level of 0.5% the oxidation and hydrolysis effects are present but very limited. Hypochlorite should not be used in abdominal wounds, open chest wounds, on nervous tissue, or in the eye. Irrigation of the abdomen may lead to adhesions and is therefore also contraindicated. The use of hypochlorite in the

thoracic cavity may be less of a problem, but the hazard is still unknown.

4. Finally, while not yet in the inventory, Reactive Skin Decontamination Lotion (RSDL) is a new decontaminant that is expected to replace the M291 in the future. It is a packaged sponge that contains a liquid solution that effectively wipes away chemical agents and simultaneously provides oxime protection against nerve agents.

CERTIFICATION OF DECONTAMINATION

Certification of decontamination for chemical agents is accomplished by any of the following: processing through the decontamination station, M8 paper, or the ICAM (Improved Chemical Agent Monitor). If proper procedure is followed, the possibility of admitting a contaminated casualty to an MTF is extremely small. Fear is the worst enemy, not the contaminated warfighter.

WOUND DECONTAMINATION

The initial management of a casualty contaminated by chemical agents will require removal of IPE and skin decontamination before treatment within an MTF.

When thorough decontamination is performed, contaminated bandages are

removed and wounds are flushed with sterile water. Any contaminated debris (such as clothing in the wound that may hold agent) is irrigated and removed from the wound by decontaminated gloved hand, instruments, or other no touch technique. The bandages are then replaced only if bleeding recurs or the wound needs to be protected from further contamination. Contaminated tourniquets are replaced with clean tourniquets and the sites of the original tourniquets decontaminated. Splints are thoroughly decontaminated by rinsing with a 0.5% hypochlorite solution or copious amounts of soap and water, but removed only by a physician or a medical aidman directly supervised by a physician.

General consideration. Of the agents discussed, only two types, the vesicants and nerve agents, might present a hazard from wound contamination. Cyanide is quite volatile, so it is extremely unlikely that liquid cyanide will remain in a wound, and it requires a very large amount of liquid cyanide to produce vapor adequate to cause effects.

Mustard converts to a cyclic compound within minutes of absorption into a biological milieu, and the cyclic compound rapidly (minutes) reacts with blood and tissue components. These reactions will take place with the components of the wound -- the blood,

the necrotic tissue, and the remaining viable tissue. If the amount of bleeding and tissue damage is small, mustard will rapidly enter the surrounding viable tissue where it will quickly biotransform and attach to tissue components (and its biological behavior will be much like an intramuscular absorption of the agent).

Although nerve agents cause their toxic effects by their very rapid attachment to the enzyme acetylcholinesterase, they also quickly react with other enzymes and tissue components. As they do with mustard, the blood and necrotic tissue of the wound will "buffer" nerve agents. Nerve agent that reaches viable tissue will be rapidly absorbed, and since the toxicity of nerve agents is quite high (a lethal amount is a small drop), it is unlikely that casualties who have had much nerve agent in a wound will survive to reach medical care.

Potential risk to the surgeon from possibly contaminated wounds arises from agent on foreign bodies in the wound and from thickened agents.

Thickened agent. Thickened agents are chemical agents that have been mixed with another substance (commonly an acrylate) to increase their persistency. They are not dissolved as quickly in biological fluids, nor are they absorbed by tissue as rapidly as other

agents. VX, although not a thickened agent, is absorbed less quickly than other nerve agents and may persist in the wound longer than other nerve agents.

Thickened agents in wounds require more precautions. Casualties with thickened nerve agents in wounds are unlikely to survive to reach surgery. Thickened HD has delayed systemic toxicity and can persist in wounds even when the large fragments of cloth have been removed. Though the vapor hazard to surgical personnel is extremely low, contact hazard from thickened agents does remain and should always be assumed.

No country is currently known to stockpile thickened agents. In a chemical attack, the intelligence and chemical staffs should be able to identify thickened agents and to alert medical personnel of their use.

Off-gassing. The risk from vapor off-gassing from chemically contaminated shrapnel and cloth in wounds is very low and not significant. Further, there is no vapor release from contaminated wounds without foreign bodies. Off-gassing from a wound during surgical exploration will be negligible (or zero). No eye injury will result from off-gassing from any of the agents. A chemical-protective mask is not required for surgical personnel.

Foreign material. The contamination of wounds with mustard or nerve agents is basically confined to the foreign material (e.g., battle dress uniform (BDU) and protective garment in the wound). The removal of this cloth from the wound effectively eliminates the hazard. There is little chemical risk associated with individual fibers left in the wound. No further decontamination of the wound for chemical agent is necessary.

Wound exploration/debridement. Surgeons and assistants are advised to wear a pair of well-fitting (thin), butyl rubber gloves or double latex surgical gloves and to change them often until they are certain there are no foreign bodies or thickened agents in the wound. This is especially important where puncture is likely because of the presence of bone spicules or metal fragments.

The wound should be explored with surgical instruments rather than with fingers. Pieces of cloth and associated debris must not be examined closely, but quickly disposed of in a container of 5% hypochlorite. The wound can then be checked with the ICAM, which may direct the surgeon to further retained material. It takes about 30 seconds to get a stable reading from the ICAM. A rapid pass over the wound will not detect remaining contamination. The wound is debrided and excised as normal, maintaining

a no-touch technique. Removed fragments of tissue are dropped into hypochlorite. Bulky tissue such as an amputated limb should be placed in a plastic or rubber bag (chemical-proof), which is then sealed.

Hypochlorite solution (0.5%) may be instilled into noncavity wounds following the removal of contaminated cloth. This solution should be removed by suction to an appropriate disposal container. Within a short time, i.e., five minutes, this contaminated solution will be neutralized and nonhazardous. Subsequent irrigation with saline or other surgical solutions should be performed.

Penetrating abdominal wounds caused by large fragments or containing large pieces of chemically contaminated cloth will be uncommon. Surgical practices should be effective for the majority of wounds in identifying and removing the focus of remaining agent within the peritoneum. Saline, hydrogen peroxide, or other irrigating solutions do not necessarily decontaminate agents, but may dislodge material for recovery by aspiration with a large bore sucker. The irrigation solution should not be swabbed out manually with surgical sponges. The risk to patients and medical attendants is minuscule. However, safe practice suggests that any irrigation solution should be considered potentially contaminated.

Following aspiration by suction, the suction apparatus and the solution should be disposed of in a solution of 5% hypochlorite.

Superficial wounds should be subjected to thorough wiping with soap and water or a 0.5% hypochlorite solution and subsequent irrigation with normal saline.

Instruments that have come into contact with possible contamination should be placed in 5% hypochlorite for ten minutes prior to normal cleansing and sterilization. Reusable linen should be checked with the ICAM or M-8 paper for contamination; if found to be contaminated, it should be disposed of in a 5 to 10% hypochlorite solution.

CONCLUSIONS

Decontamination at the MTF is directed toward (1) eliminating any agent transferred to the patient during removal of protective clothing, (2) decontaminating or containing contaminated clothing and personal equipment, and (3) maintaining an uncontaminated treatment facility.

Current doctrine specifies the use of the M291 (SDK), soap and water, or a 0.5% hypochlorite solution. These decontaminants

have been tested and found to be effective when used appropriately.

CASUALTY MANAGEMENT IN A CONTAMINATED AREA

OVERVIEW

In a contaminated environment, casualties enter an MTF through the patient decontamination station (PDS). This occurs at all levels of medical care where contaminated casualties might be received. The purpose of the PDS is to remove all contamination, or as much as possible, from the casualty before he/she enters the clean MTF; this ensures that unprotected medical staff inside the facility are not made ill or become cross-contaminated by the agent that is on the arriving patient. The key military reference for the decontamination of patients is FM 4-02.7, Health Service Support in an NBC Environment, which offers detailed instructions on the establishment and operation of a PDS. The key reference for the establishment of stateside MTF patient decontamination is the Occupational Safety and Health (OSHA) Best Practices for Hospital-based First Receivers of Victims from Mass Casualty Incidents Involving the Release of Hazardous Substances, January 2005. This section outlines the key points found in those documents. More detailed PDS procedures are also found in Appendix A.

ZONES OF CONTAMINATION

Hot Zone. This is the area that is directly contaminated by CBRN agents. In combat, this is the contaminated battlefield or TIM release site (e.g., factory storage tank or terrorist bomb). Casualties usually undergo immediate decontamination in the hot zone or on the periphery of it.

Warm Zone. This is an area where low levels of dry, liquid, and vapor contamination can be expected once contaminated individuals enter this area. The contamination hazard is essentially the agent that remains on the patients that are brought into this area. In this zone immediate, patient operational, and patient thorough decontamination take place. The PDS is initially set up in an area free of contamination. This area becomes part of the warm zone once contaminated casualties begin to arrive.

Cold Zone. The cold zone is an area free from liquid, dry, and vapor contamination. All personnel and patients entering this zone have been decontaminated. Protective ensemble and mask are usually not required for personnel in the cold zone unless the area becomes contaminated. Standard precautions must be

practiced if the patient is infectious from a biological agent.

The principal components of the PDS are:

- Entry control point (ECP)/arrival point
- Triage area
- Emergency treatment area
- Decontamination area(s)
- "Hot line"

After crossing the hot line, the casualty enters the clean triage and treatment area. From there, the now contamination-free casualty is brought into that MTF or prepared for “clean” disposition to another MTF.

The size of the MTF will dictate the personnel support needed to staff the PDS to ensure that it is functional. At a Battalion Aid Station (BAS), for example, staffing is limited and the same senior medical NCO serving as the triage officer may also provide emergency care. The decontamination areas will be staffed by a limited number of augmented personnel, and very limited medical care can be provided in the clean treatment area. At larger MTFs there will be a medical professional to triage and others to provide emergency care. There will be more decontamination lanes and more augmentees to man these lanes. If augmented personnel are not plentiful, the decontamination

team will be supplemented by non-medical personnel from the hospital staff. Manning a PDS can take 19 to 45 people, depending on its size and the time that the PDS needs to be in continuous operation.

The following is intended as an introduction to each of these stations. More detailed information can be found in the Appendix or in the references noted previously.

KEY COMPONENTS OF A PDS

All PDS operations must have the following key components to operate effectively.

WARM SIDE

Protection. All staff on this side of the PDS wear MOPP Level IV, roughly equivalent to OSHA Level C.

Entry Control Point (ECP) and Arrival Area. Patients pass through the ECP where access is controlled to the PDS. Vehicles with casualties then proceed to the arrival point where they are unloaded. This is manned by augmentees. Key activities here are (1) routing of vehicles, (2) unloading of vehicles, and (3) quick pat down searches to remove ordinance from patients. All staff are in MOPP Level IV when contaminated patients arrive.

Warm Side Triage Area. This is located near the arrival area; patients are moved to this area from the arrival point. Here casualties are simultaneously triaged as to their need for medical care, their priority for patient thorough decontamination, and their priority for evacuation to the next level of care. Within the triage area casualties are moved to either the immediate [warm side Emergency Medical Treatment (EMT)], delayed, minimal, or expectant treatment areas. A patient is retriaged as their condition changes. The placement of treatment areas in relation to the decontamination lanes is suggested to improve patient flow through the PDS.

- The immediate patients are moved to the warm side, EMT. This area is located between patient triage (closer to triage to minimize the time it takes to move from triage to dirty EMT) and the entrance to the litter decontamination lanes. This way a patient can be moved to litter decontamination without interfering with the traffic flow from other patient groups.

- The delayed patient area should be positioned nearer to the entrance to both the litter and ambulatory decon lines. This way delayed patients can be processed through

either the litter or ambulatory lanes when the lanes become available.

- Minimal patients should be positioned near to the ambulatory patient area so that if medical care on the clean side of the hot line is needed they can process through the ambulatory lane when it becomes available and not interfere with the flow to the litter lanes.

- Expectant patients should be located near to the EMT area, but farther away from the decontamination lanes so that they can be retriaged and stabilized for decontamination if the EMT area no longer has patients in it.

Casualties are retriaged as they progress through the EMT and decontamination process.

Warm Side Emergency Treatment (EMT) Area. Patients triaged as immediate for medical treatment are sent to this area until their condition is stabilized for patient thorough decontamination at this PDS or for dirty evacuation to another medical facility. An initial quantity of medical supplies are located in this area to provide antidotes to chemical agents, bandages for wounds, equipment to establish IV access, intubation equipment to establish

emergency airways, and decontamination kits to provide immediate or operational decontamination to patients. It is important to only put enough supplies here for the anticipated number of patients so that unused supplies are not in danger of contamination. The warm side EMT area should be large enough to expand and handle an influx of patients. Staffing should consist of trained and experienced aidmen (EMT, corpsman, etc.), nurses, or physician assistants.

Warm Side Disposition (Dirty Evacuation). Located in the vicinity of the warm side EMT. Here patients remain in protective ensemble and undergo operational decontamination and staging for “dirty” evacuation (ground, water, or rotor wing aircraft) to another MTF where adequate resources are available to care for them.

Contaminated Waste Dump. Located at least 75 meters down wind from the hot line. Bags of contaminated clothing and bandages are taken to this area where they are buried and marked with the appropriate hazard markers. The position is communicated to headquarters so that the waste can be disposed of properly.

Temporary Morgue. A shaded area located on the warm side where the

contaminated remains of those who die while being processed through the PDS are stored. These remains stay on the warm side of the hot line and are handled in accordance with theater policy until they are retrieved by the services unit that turns them over to mortuary affairs.

Litter Patient Decontamination Lane(s). This area is located between the warm side EMT and the hot line where litter patients have their clothing removed, contaminated bandages and splints replaced, and personal effects and field medical card (FMC) placed in plastic, zip-lock bags, and where they are decontaminated. Patients must be medically stable enough to undergo decontamination before they are brought to this area. Those performing decontamination also wear a toxicological agent protective (TAP) apron over their protective ensemble to keep their protective ensemble dry and to allow them to decontaminate their aprons before conducting patient transfers. With the exception of the Air Force and some Navy units who have trained medical teams throughout the decontamination process, this area is manned by augmentees who are closely supervised by an aidman.

Ambulatory Patient Decontamination Lane. This area is usually located parallel to the litter patient decontamination lane. Ambulatory

patients who need to see the physicians at the MTF are processed through this area where they have their clothing removed, contaminated bandages and splints replaced, and personal effects and FMC placed in plastic, zip-lock bags, and they are decontaminated. Ambulatory individuals who do not have medical complaints needing to be seen at this MTF are treated in the treatment area and returned to their unit without undergoing decontamination or crossing the hot line, or they are processed through troop decontamination lanes and not through the medical ambulatory decontamination lane. Those performing decontamination also wear a TAP apron to keep their protective ensemble dry. This area is usually manned by at least one aidman, and other augmentees if available, to supervise ambulatory patients as they process through the line and assist one another.

Contamination Check Area. This area is located between the decontamination lanes and the hot line. Here, completeness of decontamination is checked using the appropriate monitoring devices (e.g., chemical: ICAM or M8 paper). Zip-lock bags containing the patient's personal items can also be unzipped and the monitors used to check for contamination of the items inside them. The decontamination check of patients may not be necessary where fully plumbed decontamination

tents provide adequate soap and water for a thorough wash.

Litter Decontamination Station. Here, warm side litters are washed and readied for use again. Buckets and sponges with 5% hypochlorite solution are available as well as water to rinse the litters. With a shower/roller system, litters only need to be sent back through the decon station for a wash with soap and water.

Weapons and Contaminated Personal Effects Storage Area. Here, patient weapons and personal effects are secured and inventoried. Items from this area are decontaminated and moved through the contamination check area before being sent across the hot line. If personnel are limited, this area may need to be well organized and under the observation of personnel serving as security augmentees.

Warm Side Rest Area. A shaded rest area where PDS team members can rest and drink water while remaining in their protective ensemble.

Hot Line and Shuffle Pit. The hot line separates the PDS warm zone (dirty side) from the cold zone (clean side) where the MTF is

located. **No liquid or solid contamination crosses the hot line.** The line must be indicated in some way (e.g., by a barrier, tape line, or air lock) so that all personnel know they cannot cross the line until they are properly decontaminated. In the battlefield it is best to indicate this area with a specific barrier, such as concertina wire, to protect the medical facility. Shuffle pits or boot rinses are located at openings along the hot line to ensure that footwear worn by individuals moving across the hot line is decontaminated. At the hot line, information on the patient's FMC is transferred to a clean card, and litter patients are transferred to a clean litter to ensure that no contaminated cards or litters cross the hot line. A blanket is also placed on the patient once they are transferred to a clean litter. Team members on the clean side receive the patient. Staffing on the dirty side consists of the team in TAP aprons that decontaminated the patient and the warm side aidman, if available. Receiving members consist of one medic and at least two augmentees for litter patients and one augmentee for ambulatory patients.

Vapor Control Line (VCL). This line is typically upwind of the hot line by approximately 10 meters. Patients and PDS team members remain masked until they cross this line. This line can be established using chemical vapor detectors such as the ACADA.

CLEAN SIDE

Protection. Personnel assigned to this area do not need to wear protective equipment as the patients in this area are free from contamination. When processing infectious biological casualties, staff should practice universal precautions and wear appropriate respiratory protection.

Triage/EMT Area (Cold Zone). This contamination-free area is located beyond the hot line and vapor control line where patients are retriaged and treated. It can be a holding and staging area for admission to an MTF, for clean evacuation to another MTF, or for ambulance transport from a co-located (troop and patient) PDS to a nearby MTF.

Disposition (Cold Zone - Clean Evacuation). This area is adjacent to the cold zone triage/EMT. From this area, contamination-free patients that have been stabilized are staged for transport to another treatment facility.

Supply Point. This point is located outside the VCL. PDS supplies are kept here and are handed across the hot line to the warm side when needed.

PDS CRITICAL CONCERNS

Warm Side Triage and Treatment

It is important that the triage officer be practiced enough to effectively triage patients so that the PDS is not overwhelmed with patients who can be treated on the warm side and returned to their unit; should be dirty evacuated to a larger MTF (if that is possible); or stabilized in the warm area until they are ready for decontamination. The triage officer might be a senior medic in a BAS. In larger medical units the triage officer might be a physician or physician's assistant. His ability to evaluate the casualty will be limited because both he and the casualty will be in MOPP Level IV.

Warm Side EMT Care

Those casualties needing immediate care will be sent to the warm side EMT. Casualties classified as minimal might also be sent to this area, if the appropriate care can be provided in a contaminated environment. The purpose of this is to return them to duty quickly and to lessen the workload on the decontamination teams.

However, the types of injuries that can be treated without breaking the integrity of the protective garment are small; once the integrity of the protective garment is violated, the minimal casualty will need to be treated and sent through troop decontamination to don replacement IPE before being returned to the battle area. For those patients being processed through patient decontamination who might be returned to their unit, arrangements will need to be made with supported units to have replacement IPE available for these casualties. Decontaminated litter patients will be placed in a patient protective wrap (PPW) if they need to be transported on dirty evacuation assets or through contaminated areas. Administration of MARK I Kits is an example of treatment that can be given without breaking the seal of the protective garment.

Those casualties classified as delayed will be sent through the PDS for decontamination if they require care in the clean treatment area. Otherwise, they will be dirty evacuated to the next level of care that can better handle them.

The expectant casualty will be temporarily set aside, adjacent to the warm side EMT, for later re-evaluation as there are no more patients in the EMT station.

The amount of vapor arising from patient IPE should not be enough to preclude an apneic patient from being ventilated. Ideally, in a battlefield environment a resuscitation device individual chemical (RDIC) with its filtered ambu-bag should be used. Ventilation of a newly apneic patient will be limited more by the lack of personnel to squeeze the ambu-bag than by the risk of forcing more chemical vapor into the casualty's lungs. Intravenous injections can be given and IV fluids can be started after decontamination of the skin at the IV insertion site and the care provider's gloves. Minor suturing can be done in this area using the same precautions. The time needed by the single medical care provider to perform these procedures is probably the limiting consideration, not the risk of further contamination.

Preventing Musculoskeletal and Heat Injury

A safety officer must be appointed for operations on the warm side of the PDS. This can be the NCOIC, OIC, or some other individual. It must be someone who can observe the PDS workers, travel freely around the PDS, and manage work/rest cycles.

Worker musculoskeletal injury can easily occur from patient lifting and litter carrying or injuries caused by falls while wearing protective

ensemble. To reduce these, clear routes within the PDS to reduce tripping hazards; establish decontamination lanes far apart to reduce clutter; enforce frequent garbage bag removal to reduce trip hazards; train and enforce safe-lifting techniques; ensure there are adequate rest breaks; and use work-saving equipment, e.g., NATO litter carriers, if available.

Patient triage, treatment, and decontamination involve moderate and heavy work. Wearing IPE generates heat that is not easily dissipated by the process of sweating since the wearer's skin does not contact the air to cool. This can create heat injury and increase accident frequency as overheated workers overlook safety procedures.

Heat Stroke. *Cause:* The body's temperature regulatory system fails and sweating becomes inadequate. *Signs and Symptoms:* Body temperature is usually 105°F (40.5°C) or higher. The victim is mentally confused, delirious, perhaps in convulsions, or unconscious. *Medical Attention:* First-aid must be administered immediately since death can occur without quick treatment. Move the victim to a cool shaded area. Process the victim quickly across the hot line. Remove the victim's IPE, soak the underclothing with water, and fan the body to increase cooling. Evacuate to

nearest MTF for medically monitored fluid replacement.

Heat Exhaustion. *Cause*: Loss of large amounts of fluid by sweating, sometimes with excessive loss of salt. *Signs and Symptoms:* Sweating, extreme weakness or fatigue and may show giddiness, nausea, or headache; can resemble the early heat stroke symptoms. The skin is clammy and moist, the complexion is pale and flushed, and the body temperature is normal or slightly elevated. The victim may lose consciousness. *Medical Attention:* Notify medical personnel immediately. Move the victim to a cool place and rest. Process the victim across the hot line as operation tempo permits. Have him drink plenty of liquid. His status should be monitored by an aidman.

Heat Cramps. *Cause:* Painful spasms of the muscles in those who sweat profusely, drink large quantities of water, but don't adequately replace salt loss. *Medical Attention:* Cramps may occur during or after work hours and may be relieved by taking salted liquids by mouth. Move the individual to the warm side rest area and seek medical attention. Return to work; can be put in a less physically demanding position if contention improves; or process across the hot line when operations tempo allows.

Fainting. *Cause:* Typically seen in a worker who is not accustomed to hot environments. The heat will cause blood vessels in the skin and the lower part of the body to enlarge to try to cool the body. The blood may pool there rather than return to the heart to be pumped to the brain causing the person to faint. *Medical Attention:* The worker should be guided to lie down in the warm side rest area. Elevate the legs. Seek medical advice. He should recover quickly. Return the individual to duty when recovered, or process across the hot line when operations tempo allows.

Work/Rest Cycles. These are carried out on the warm side of the hot line. Enforcing adequate worker rest ensures adequate worker hydration, gives the body an opportunity to get rid of excessive heat, slows down the production of internal body heat that is created during physical work, and provides greater blood flow to the skin. Wearing protective overgarments adds 10°F (5.6°C) to the wet bulb globe temperature (WBGT) index, and wearing body armor increases this by another 5°F (2.8°C).

HEAT CATEGORY	WBGT INDEX °F	MODERATE WORK		HARD WORK	
		WORK/ REST MIN	WATER INTAKE QT/HR	WORK/ REST MIN	WATER INTAKE QT/HR
1 (WHITE)	78-81.9	NL	3/4	40/20	3/4
2 (GREEN)	82-84.9	50/10	3/4	30/30	1
3 (YELLOW)	85-87.9	40/20	3/4	30/30	1
4 (RED)	88-89.9	30/30	3/4	20/40	1
5 (BLACK)	> 90	20/40	1	10/50	1

CAUTION:

Drinking Fluids. A worker may produce as much as two to three gallons of sweat in the course of a day's work. A worker, therefore, should not depend on thirst to signal when and how much to drink. Five to 7 ounces of liquid should be consumed every 15 to 20 minutes to replenish the necessary fluids in the body. Water intake should not exceed 1 quart per hour or 12 quarts per day, since excessive water consumption can dilute the salt content of the blood to the point where it interferes with brain, heart, and muscle function. This "water poisoning" can result in heat attack and seizures.

CHEMICAL DEFENSE EQUIPMENT

This overview is divided into four sections:

- Individual Protection
- Individual Decontamination
- Detection and Alarms
- Patient Protective Equipment

INDIVIDUAL PROTECTION

This section includes standard "A" chemical defense equipment (CDE) issued to each warfighter depending on their MOS and consisting of the following:

- M40 Series Field Protective Mask
- M42A2 Combat Vehicle Protective Mask
- M45 Air Crew/Land Warrior Chem-Bio Mask System
- MCU-2A/P Protective Mask
- Battle Dress Overgarment
- Joint Service Lightweight Integrated Suit Technology
- Suit Contamination Avoidance and Liquid Protective
- Chemical Protective Gloves and Overboots

- Skin Exposure Reduction Paste Against Chemical Warfare Agents (SERPCWA)

Chemical-Biological Mask: Field M40/M42A2/ M45
TM 3-4240-346-10
TM 3-4240-348-10

This section focuses on three variations of protective masks, the M40A1, the M42A2, and the M45. These masks share many of the same design characteristics, capabilities, and features. Each mask has been designed for very specific mission requirements, such as aircraft or combat vehicle operation. This section is not designed to highlight specific mask operational capabilities; it is intended to provide technical information pertaining to overall mask operations.

Protective masks provide users with respiratory, eye, and face protection against CB agents and radioactive fallout particles. If a mask is properly fitted and worn correctly it provides a gas-tight face seal, which prevents contaminated air from reaching the wearer's respiratory, ocular, and dermal systems.

Masks described in this section have not been designed for use TIC environments and are known to be ineffective against chemicals such as ammonia and carbon monoxide. The

masks are also not suitable for confined spaces where oxygen is insufficient to support life.

Each mask is constructed of silicone rubber with an in-turned sealing surface so it can form a comfortable seal on the wearer's face, an external second skin for additional protection. Binocular eye lens system is used for improved vision, clear and tinted outserts provide eye protection against laser and low speed fragmentation, and optical inserts may be inserted if the user requires corrective lenses. An elastic head harness secures the mask to the user's face. Other common features include front and side voicemitters to allow for face-to-face and phone communications. Each of the masks is furnished with drinking tubes to allow for hydration. A key design feature of each of the discussed protective masks is the use of a standard C2/C2A1 NATO threaded filter canister. The C2 filter canister contains Chromium VI; damaged or unusable canisters are considered hazardous waste and are known to be carcinogenic if inhaled or swallowed. The C2A1 canister is chromium free but must be disposed of in accordance with state and local environmental laws. Both canisters are qualified to withstand and protect against a maximum of 15 nerve, choking, and blister agent attacks. The canister is externally mounted and may be mounted on the left or right side of the user's face depending on the preference of the user.

Additionally a quick-doff hood is used to provide protection to the user's head and neck.

MCU-2A/P Protective Mask
Air Force Technical Order 14P4-15-1

The MCU-2A/P Series Mask is designed to protect the face, eyes and respiratory tract of the user from tactical concentrations of chemical and biological agents, toxins and radioactive fallout particles. The mask has a unimolded, silicone rubber face piece and a single flexible lens bonded onto the face piece. The large lens gives the user a wide field of vision. It has a single filter and two voicemitters, one on the front of the mask for speaking directly into a telephone or radio handset and one at the side to allow personnel nearby to hear. A nosecup with two inlet valves fits over the nose and mouth. It directs incoming air across the inside of the lens to reduce fogging. The mask has a drinking tube that connects to a canteen with an M1 cap. The mask is not authorized for use during TIC spills, and the mask is not effective against chemicals such as ammonia, chlorine, or even carbon monoxide fumes. The mask is not effective in confined spaces where oxygen levels are insufficient to sustain life.

Battle Dress Overgarment (BDO)
TM 10-8415-209-10

The Battle Dress Overgarment will hereafter be referred to as the BDO. The BDO chemical protective overgarment is a two-piece overgarment consisting of a coat and separate trousers. The BDO is available in a factory-sealed, vacuum-packaged, vapor-barrier (VB) bag. The VB bag assists in protecting the BDO from environmental impacts associated with storage. Each VB bag contains a jacket and trousers. The suit is composed of an outer layer constructed of 50/50 nylon cotton tightly woven material that has been treated with water-resistant sealant. The liner or inner layer is constructed of charcoal-impregnated polyurethane foam, nylon tricot laminate. BDOs are available in various camouflage patterns with sizes ranging from extra extra small (XXXS) to double extra large (XXL). Once the BDO has been removed from the vacuum-sealed packaging, the suit offers 22 days of wear. With slight increase in risk commanders may increase wear time to 30 days. Wear time for the BDO begins when the seal is broken on the VB bag. To properly maintain the BDO when not in use, it should be sealed in the original VB bag or other similar material bag. The bag can be sealed with duct tape, 100 mile per hour tape, or other suitable tape. Donning the BDO regardless of the amount of time within a 24-hour period

constitutes a day of use. The BDO is currently qualified to offer 24 hours of protection against CB agents in solid, liquid, or vapor form; the suit also protects against alpha and beta radioactive particles.

Joint Service Lightweight Integrated Suit Technology (JSLIST)
TM 10-8415-220-10

The Joint Service Lightweight Integrated Suit Technology (JSLIST) Chemical Protective Overgarment (CPO) has been designed to replace its predecessor, the battle dress overgarment (BDO). The JSLIST CPO is a two-piece overgarment consisting of a coat with an integrated hood and separate trousers. The CPO has been designed to be lighter weight, more flexible and have the ability to be laundered up to six times. In addition the system has also been designed to reduce the stresses of protective gear. The JSLIST CPO is available in four- color woodland and three-color desert camouflage patterns. The JSLIST suit is composed of an outer layer of 50/50 nylon/cotton poplin, rip-stop material with camouflage pattern facing outward. The liner or inner layer is polyester knit coated with activated carbon spherical absorbers covered by a nonwoven laminate that is bonded to a tricot knit back. Unlike the BDO, JSLIST suits are not packaged as sets; JSLIST suits consist of a coat

and trousers. Each component is separately packed in a factory-sealed vacuum bag containing the ensemble item and a resealable bag. JSLIST suits are available in seven sizes ranging from short extra small (SXS) to large long (LL). The JSLIST overgarment is currently qualified to offer 24 hours of protection against CB agents in solid, liquid, or vapor form; the suit will also protect against alpha and beta radioactive particles.

Once the CPO has been removed from the vacuum- sealed packaging, the suit offers 45 days of wear and 120 days of service life. To be properly maintained and stored when not in use, the JSLIST CPO should be placed in the resealable bag that is furnished with each component of the ensemble.

Both the BDO/JSLIST ensemble will be worn in all environments when under threat of an imminent nuclear, biological, or chemical attack or after chemical operations have been initiated.

Once the suit has been contaminated, the Warfighter must replace the suit by using the MOPP gear exchange procedure described in STP 21-1-SMCT, Warfighter's Manual of Common Tasks, October 2003, Task #031-503-1023, Exchange MOPP Gear. The BDO/JSLIST adds weight to the Warfighter’s

workload. In addition, the BDO/JSLIST prevents heat exchange with the environment and may add, depending on the Warfighter's level of exertion, 10°F to 15°F to his ambient temperature and heat burden. When wearing the BDO/JSLIST at MOPP 1 or MOPP 2, and complete encapsulation is not required, certain modifications to the uniform are authorized:

- The trouser leg closures may be unzipped.
- The waist tabs may be loosened.
- The jacket may be unzipped.
- The sleeve Velcro closures may be opened.

This overall loosening of the BDO/JSLIST will allow heat to escape as walking and other movements induce a bellows action of the suit against underlying clothing and skin.

Suit, Contamination Avoidance and Liquid Protective (SCALP)
TM 10-8415-209-10

The SCALP is an impermeable, lightweight, inexpensive, disposable ensemble that provides supplemental liquid protection. The SCALP is a four- piece ensemble that consists of a jacket, trousers, and two footwear covers. It is designed to be worn over the BDO or JSLIST with protective overboots. The footwear covers are constructed with 12 mil embossed polyethylene

soles. The SCALP ensemble provides protection from gross liquid contamination for up to one hour. Operationally the SCALP is used to protect personnel who are conducting decontamination procedures from becoming soaked during decon operations.

Chemical Protective Gloves and Overboots

- Green/Black Vinyl Overboots (GVO) (BVO)
- The Multipurpose Overboot (MULO)
- Gloves, 0.025-inch thickness
- Gloves, 0.014-inch thickness
- Gloves, 0.007-inch thickness

Green or Black Vinyl Overboots (GVO) (BVO)

The overboots have been designed to be worn over combat boots to protect the user's feet. The boots are available in sizes 3-14. The GVO/BVO are constructed of vinyl, making them impervious to all known chemical and biological agents and alpha and beta radiological particles. They also protect against environmental effects such as rain, mud, and snow. Both boots are similar except for the color and the enlarged, elastic pull-tab fasteners on the BVO. The GVO/BVO are qualified to offer 60 days of protection. If the GVO/BVO become contaminated, they provide 24 hours of protection. Following contamination, use a 5%

HTH and water solution or a 5% household bleach and water solution to decontaminate the GVO/BVO. Ensure boots are serviceable and no signs of deterioration are present after the decontamination process. If boots are deemed to be unserviceable, replace them.

Multipurpose Overboot (MULO)

The multipurpose rain/snow/CB overboot (MULO) replaces the older black vinyl overboot/green vinyl overboot (BVO/GVO). The MULO is made by injection molding an elastomer blend, compounded to provide the characteristic chemical and environmental protection required. It incorporates two quick-release side buckles and is designed to be worn over the standard issue combat boot, jungle boot, and intermediate cold/wet boot. The MULO provides 60 days of durability and 24 hours of protection against liquid chemical agents. The MULO is capable of being decontaminated to an operationally safe level using standard field decontaminants. Environmental protection is provided against water, snow and mud, in addition to petroleum, oil, and lubricant (POL). Additionally, the MULO is flame resistant.

Chemical Protective Glove Set

Chemical protective glove sets are qualified to offer protection against chemical and biological agents, as well as alpha and beta radiological particles. The chemical protective glove set consists of an outer glove for protection and an inner glove for absorption of perspiration. The outer gloves are made from black butyl rubber and are impermeable to chemical agents. The inner gloves are made of thin, white cotton.

Glove sets are available in three thicknesses, 7 mil to 25 mil, and in sizes ranging from extra small (XS) to extra large (XL).

The 25-mil glove offers the most durable protection and may be utilized to perform close combat tasks or other types of heavy labor. The 14-mil glove is less durable and is used in an environment in which much less physical demand is placed on the glove. Such users could include vehicle mechanics, aviators, or weapons crews.
If either glove becomes contaminated, decontaminate or replace within 24 hours after exposure. Contaminated gloves may be decontaminated with a 5% chlorine solution or a 5% HTH and water solution.

The 7-mil gloves offer the most tactility and are used by individuals who require extreme sensitivity to accomplish tasks without subjecting the glove to harsh treatment. If the 7-mil glove becomes contaminated, replace or decontaminate it after six hours of exposure. Contaminated gloves may be decontaminated with a 5% chlorine solution or a 5% HTH and water solution.

Skin Exposure Reduction Paste Against Chemical Warfare Agents (SERPACWA)
NSN 6505-01-483-7162

SERPACWA is used by service members in conjunction with MOPP) gear to enhance protection against chemical warfare agents. Approved by the FDA for military use only, it is a cream containing chemically inert perfluorinated polymers. It is applied to susceptible areas of the skin before donning MOPP gear. Not intended for use by itself, SERPACWA will provide additional protection at those locations where the MOPP is susceptible to leakage or separation, including the waist, groin, armpits, wrists, ankles, and neck. In conjunction with personal decontaminating materials, the use of SERPACWA will prevent or reduce the toxicity resulting from exposure to chemical warfare agents. When used as directed, SERPACWA will provide between five and eight hours of protection.

For further information on these items, see Multiservice Tactics, Techniques, and Procedure for Nuclear, Biological, and Chemical (NBC) Protection FM 3-11.4 June 2003, Chapters VI, Appendix A.

INDIVIDUAL DECONTAMINATION

The preceding section provided an overview of the primary items of chemical defense equipment which, when used correctly, will prevent contact with agent in typical battlefield concentrations. The problem of decontamination arises when some warfighters, because of bad training, bad discipline, or bad luck, become exposed to liquid agent despite the availability of protective masks and clothing.

This section addresses two decontamination kits currently in the inventory--the M291 Decontaminating Kit Skin and the M295 Decontamination Kit Individual Equipment. The kits are fairly simple in design and function, and instructions for their use are straightforward and easily committed to memory. Because of the potency of liquid nerve agents and the rapidly occurring tissue damage caused by vesicants, every warfighter must be able to conduct an effective decontamination of all exposed skin without referring to the instructions printed on the kits.

Decontaminating Kit, Skin: M291
NSN 4230-01-276-1905
TM 3-4230-229-10

The M291 Skin Decontamination Kit consists of a wallet-like carrying pouch containing six individually sealed decontamination applicator pads. Each pad is filled with FDA-approved Ambergard XE-555 decontaminating resin. Each kit allows personnel to decontaminate their skin of liquid nerve and vesicant agents through physical removal, absorption, and neutralization of chemical agents. The M291 kit is nontoxic and has been designed for external use. Since it may be slightly irritating to eyes or skin, decontamination powder should be kept out of eyes, cuts, and wounds. Inhalation of the powder should be avoided as it can irritate the lungs.

Each of the individual decontamination pads is capable of providing decontamination coverage to the face and hands or an equivalent area of skin from exposure to chemical agents. Effective decontamination will result in blacking of decontaminated skin areas.

Decontamination Kit, Individual Equipment: M295 (DKIE)
NSN 4230-01-357-8456
TM 3-4230-235-10

The M295 DKIE consists of a pouch containing four individually sealed wipe down mitts. Each wipe-down mitt is comprised of a sorbent decontaminating powder containing alumina and silica. Each kit allows personnel to decontaminate their individual equipment through sorption of contamination by both the pad and the decontaminating powder. Decontamination is effective against liquid nerve and vesicant chemical agents. Kits are worn over protective ensembles and are capable of decontaminating approximately 1,200 square feet. (M291 kits may be used to decontaminate such items as the CB mask and hood, gloves, footwear, weapons, helmet, and load-bearing vest.)

M295 kits are issued in boxes of 20 kits. The kits should be stored at the squad level in a box capable of being decontaminated.

DETECTION AND ALARMS

This section will describe the equipment issued for detection and identification of chemical agent liquid and vapor in the environment. For both the individual warfighter

and the unit, these items of equipment (listed below) are the primary means of identifying the presence and type of chemicals on the battlefield and determining when a safe condition exists.

- Paper, CM Agent Detector: M9
- Paper, CM Agent Detector: M8
- Chemical Agent Detector Kit: M256A1
- Chemical Agent Monitor
- Automatic Chemical Agent Alarm: M8A1
- Water Test Kit, Chemical Agents: M272

Paper, CM Agent Detector: M9
NSN 6665-01-049-8982
TM 3-6665-311-10

M9 detector paper is placed on personnel and equipment to detect and identify the presence of liquid nerve or blister agents in exposures as small as 100 microns in diameters. The paper contains an indicator chemical dye that will turn pink, red, reddish brown, or red purple when exposed to liquid agents. The paper is capable of detecting but cannot identify specific agents. M9 paper is manufactured in 30-feet x 2-inch adhesive-backed rolls of dull, off-white/cream-colored paper. The rolls are packaged with a reusable plastic storage bag in a vacuum-sealed, vapor-barrier package. The detector paper dye may be a potential carcinogen, so chemical protective gloves should

be worn when handling M9 detector paper. Placement of M9 is dictated by the dominant hand of the user. If the user is right-handed, M9 detector paper should be placed around the right upper arm, left wrist, and right ankle. If the user is left-handed, M9 detector paper should be placed around the left upper arm, right wrist, and left ankle. If a color change is indicated, proper masking, decontamination, and MOPP procedures must be followed.

NOTE: *M9 Chemical Agent Detector Paper will not detect cyanide.

Many substances are known to cause false positive responses on M9 paper. The following are common false positive indicators: antifreeze, liquid insecticide, or petroleum products. Attention to possible interfering substances on the battlefield can help in the later interpretation of a color change on the M9 paper in the absence of confirmation tests for agents. This does not relieve the service member of the obligation to mask and take other appropriate measures.

Paper, CM Agent Detector: M8
NSN 6665-00-050-8529

M8 Chemical Agent Detector Paper is used to detect the presence of liquid V type nerve, G type nerve, and H type blister agents. M8 paper

is issued in booklets containing 25 tan-colored sheets of chemically treated, dye-impregnated paper. Each page is perforated and staple-bound for easy removal. The reverse side of the front cover contains a color comparison bar chart for color comparison agent recognition.

If M8 paper is exposed to chemical agents, the dye impregnated paper will convert from tan to an agent-specific color, depending on the agent. The following agents will cause the dye to change to one of three colors.

- G: Nonpersistent Nerve: Yellow
- H: Blister: Red
- V: Persistent Nerve: Olive Green or Black

NOTE: *M8 Chemical Agent Detector Paper will not detect cyanide.

If indicated by M9 chemical agent detector paper or encountering a liquid suspected of being a chemical agent, service members must follow proper masking, decontamination, and MOPP procedures.

To prepare M8 paper to conduct agent identification, tear one half sheet from the booklet and affix the sheet to a stick or other object. Use the stick as a handle, blot the paper onto the unknown liquid, and wait for 30 seconds. Once 30 seconds has elapsed,

compare the tested M8 paper to the color comparison bar chart located on the inside cover of the booklet.

The following are common false positive indicators: antifreeze, liquid insecticide, or petroleum products. Attention to possible interfering substances on the battlefield can help in the later interpretation of a color change on the M8 paper

Improved Chemical Agent Detector Kit: M256A1
NSN 6665-01-133-4964
TM 3-6665-307-10

The M256A1 Chemical Agent Detection Kit is designed to detect and identify chemical agents in liquid or vapor and consists of the following:

- A booklet of M8 paper (previously described) to detect agents in liquid form; and
- 12 foil-wrapped detector tickets containing eel enzymes as reagents to detect very low concentrations of chemical vapors.

Instructions for the use of the detector tickets appear on the outside of each of the foil packets and in a separate instruction booklet in the kit. The chart below shows the agents detected by the M256A1 Kit.

Agent Detected	Symbol	Class
Hydrogen Cyanide	AC	"Blood" (cyanide)
Cyanogen Chloride	CK	"Blood" (cyanide)
Mustard	H	Blister
Nitrogen Mustard	HN	Blister
Distilled Mustard	HD	Blister
Phosgene Oxime	CX	Blister
Lewisite	L	Blister
Nerve Agents	V and G Series	Nerve

By following the directions on the foil packets or in the instruction booklet, service members can conduct a complete test with the liquid-sensitive M8 paper and the vapor-sensitive detector ticket in approximately 20 minutes. During the test, the sampler must be kept out of direct sunlight, which speeds evaporation of the reagents. Waving the detector sampler in the air also accelerates

evaporation, so the sampler should be held stationary during all parts of the test.

Simulator, Detector Tickets, Chemical Agents: Training, M256A1
NSN 6665-01-112-1644
TM 3-6665-320-10

The M256 trainer simulator was developed to provide realistic training while avoiding unnecessary exposure to potentially carcinogenic reagents in the M256A1 detector kit. The M256 trainer contains 36 pre-engineered detector tickets and an instruction booklet. The pre-engineered detector tickets show color changes comparable to those seen when the M256A1 detector kit is used in clean or contaminated environments.

Improved Chemical Agent Monitor (ICAM)
NSN 6665-01-199-4153
TM 3-6665-331-12&P

The ICAM, which is used to detect nerve and blister agents as vapors only, uses a 10-mCi nickel-63 (Ni^{63}) beta-particle radiation source to ionize airborne agent molecules that have been drawn into the unit by a pump. The resulting ion clusters vary in mass and charge and thus also travel at different rates in an applied electrical field. Comparison of the mobilities of the different ionic species to electronically stored standards

allows an on-board microcomputer to determine the type of agent and its relative concentration. A liquid crystal display (LCD) presents these data as a series of concentration-dependent bars in a G mode for G agents and VX and in an H mode for blister agents.

The ICAM detects agent vapor in that volume of air drawn by the pump into the sampling chamber of the instrument. It follows that the inlet port must not come into contact with a suspected area of evaporating agent on a surface but must nevertheless approach within a few inches of the site of suspected contamination. Because of the variation in agent concentration from one spot to another, depending upon wind velocity and other environmental factors, numerical displays of agent concentration in typical units would be impractical and unreliable. Accordingly, the display warns of a low vapor hazard (1 to 3 bars visible), a high vapor hazard (4 to 6 bars visible), or a very high vapor hazard (7 to 8 bars visible).

M22 Automatic Chemical Agent Alarm ACADA)
NSN 6665-01-438-6963
TM 3-6665-321-12&P

The M22 is an automatic agent alarm system capable of detecting and identifying standard blister and nerve agents. The

system is man-portable, operates independently after system start-up, and provides an audible and visual alarm. The M22 system also provides communications interface for automatic battlefield warning. The system consists of the M88 detector, as many as five M42 alarm units, a confidence sample, protective caps, square inlet, rain caps, a carrying case, and various power supplies.

The M22 ACADA samples the air for the presence of nerve agent vapors (GA, GB, GD, VX) and blister agent vapors (HD, L), and provides simultaneous detection and warning of these agents. It operates in cold and hot climates (-30^{o}F to +125^{o}F). Tactical operations of the M22 system are basically the same as the M8A1 Automatic Chemical Agent Alarm, except for some improvements over the M8A1. The M88 detectors normally are placed facing into the wind no more than 150 meters outside of the unit perimeter, with no more than 300 meters between detectors. They are connected to the alarm units with WD-1/TT telephone wire; whenever possible, the distance between the detector units and the alarm units should not exceed 400 meters. Improvements over the M8A1 system are as follows:

- Simultaneous detection and warning of nerve and blister agents
- Significantly more sensitive than the M8A1
- Much less response to interference

The following items can interfere with the normal operation of the M22 ACADA and will sound a false alarm:

- CS Tear Gas
- JP8 Fuel
- Brake Fluid
- Aqueous Fire Fighting Foam (AFFF)
- M18 Marking Grenade (Red and Violet)

Water Testing Kit, Chemical Agents: M272
NSN 6665-01-134-0885
TM 3-6665-319-10

The M272 Water Test Kit was designed and fielded to answer the need for a test to detect water contamination by nerve agent, blister agent, cyanide ("blood" agent), or Lewisite. The kit will operate between 32^{o}F and 125^{o}F. An enclosed instruction card enables the warfighter to conduct all the tests required to identify the threat agents. The kit will detect the chemical agents at the concentrations indicated on the following chart.

Chemical Agent	Symbol(s)	Concentration (mg/l)-*
Cyanide	AC	20.0 as CN^-
Mustard	HD	2.0 --
Lewisite	L	2.0 as As+++
Nerve	G/V	0.02 --

*Concentration reliably detected by kit tests. Water-containing agents in lesser concentrations are permissible for short-term use (up to 7 days) in both cold and warm regions as long as the daily consumption per person does not exceed 5 quarts. Each kit contains enough reagents for tests on 25 separate water samples. The operator can easily conduct the full range of tests in 20 minutes when the temperature is between 50-105°F; at lower temperatures, the water samples and the nerve agent ticket should both be warmed for 10 minutes before beginning testing. Water that is too hot may cause foaming in the detector tubes for Lewisite, mustard, and cyanide; therefore, water at temperatures between 105-125°F should be cooled for at least 5 minutes to reduce its temperature to 105° or cooler.

PATIENT PROTECTIVE EQUIPMENT

In this section, the following three items that have been fielded will be discussed:

- Patient Protective Wrap
- Decontaminable Litter
- Resuscitation Device Individual Chemical (RDIC)

Patient Protective Wrap

Wrap, Patient, Chemical Protective
NSN 6530-01-383-6260

Blower, Lightweight
NSN 4240-01-442-8415

Hose Assembly
NSN 4240-01-442-2314

Many protective garments have been developed for military personnel; however, protection for patients who are unable to wear standard chemical protective garments was lacking. The Chemical Warfare Patient Protective Wrap was developed to satisfy this requirement. The addition of the blower unit serves as a modification for the improvement of the performance of the Patient Wrap by increasing airflow to the patient. The Wrap resembles a lightweight sleeping bag. It is 107 cm wide x 249 cm long and weighs 2.7 kg. It is

constructed of a permeable sheet of carbon-impregnated fabric and an impermeable bottom sheet. The top sheet has an impermeable transparent window to permit observation of the patient during transit. A port to provide a protective entryway for the insertion of IV tubing is located at each side of the window.

The blower unit is a small, lightweight unit that provides a continuous flow of clean, filtered air for breathing. The benefit of the addition of this item is a considerable reduction in the danger of heat stress on the casualty, and an increase in the operational effectiveness of the wrap while in hot climates.

The system consists of:

- Wrap, Patient, Chemical Protective
- Blower, Lightweight
- Hose Assembly

NOTE: Patients should not be left in the wrap for longer than six hours.

Decontaminable Litter
NSN 6530-01-290-9964

Contaminated casualties arriving at a medical treatment location will in most cases require decontamination prior to definitive treatment. This decontamination process will require the use of the limited supplies of equipment organic to the treatment unit. Ideally, equipment in limited supply should be capable of complete decontamination using field-available methods. However, in tests conducted by the U.S. Army Research, Development, and Engineering Command, canvas litters exposed to liquid blister agents and then decontaminated still desorbed vapors for 72 hours after all surface contaminants were removed.

The decontaminable litter was developed to replace the canvas litters. The new litter is made from a monofilament polypropylene that has high tensile strength and low elasticity. The fabric does not absorb liquid chemical agents and is not degraded by decontaminating solutions. The fabric is flame retardant, highly rip resistant, and treated to withstand exposure to weather and sunlight. The fabric has a honeycomb weave that results in a rough, non-slip surface, and liquids easily pass through the 40% of surface area that is open. The carrying handles retract into the metal pole frame for a closed total length of 83.5 inches (212.1 cm) to

allow for loading the litter onto the UH-60 helicopter. The handles have TWO open positions, 90.0 inches (228.1 cm) and 91.6 inches (232.7 cm). The first position is a NATO standard, and litter bearers provided the second position to allow increased gripping comfort. The aluminum poles have been designed to provide direct gripping surfaces for litter stanchions. All metal parts have been painted with Chemical Agent Resistant Coating (CARC) paint.

Resuscitation Device, Individual Chemical
NSN 6515-01-338-6602

The RDIC is a ventilatory system consisting of a compressible butyl rubber bag, a NATO standard C2 canister filter, a nonrebreathing valve, a cricothyroid cannula adapter, and a flexible hose connected to an oropharyngeal mask. The mask is removable from the distal end of the flexible hose for connection of the hose to the cannula adapter. The butyl rubber bag resists the penetration of liquid chemical agent that may be on the chemical protective gloves of the operator and is easily decontaminated. The elasticity of the outer cover limits airway pressure to a maximal value of 70 cm H_2O (70 mbar). The device will deliver up to 600 ml of filtered air per cycle at a rate of 30 cycles per minute.

APPENDIX A
PATIENT DECONTAMINATION

OVERVIEW

Patient decontamination is personnel, time, and equipment intensive. Nevertheless, with a little ingenuity and attention to just a few basic principles, an effective patient decontamination procedure can be accomplished with minimal cost. The first part of this appendix briefly discusses considerations in establishing a patient decontamination station (PDS), followed by step-by-step procedures.

The PDS is part of the MTF, and the same considerations for establishing the treatment facility apply to the decontamination area. The decontamination area is located about 50 meters downwind from the treatment area (i.e., wind blowing from the clean treatment area to the dirty decontamination area).

KEY PRINCIPLES

- Wind direction
- Security of the decontamination station
- Area control of the decontamination station
- Litter patient decontamination

- Ambulatory patient decontamination

The important considerations of personnel and equipment requirements are discussed in other publications.

Wind Direction

Wind direction is important because a vapor hazard may be present downwind from a liquid contaminated area (i.e., patient arrival/triage area). Patient decontamination is always performed upwind, or at least not downwind, from the patient arrival area.

The decontamination station will initially be set up to take advantage of the prevailing wind; however, setup should be adaptable to allow for quick rearrangement when the wind comes from another direction.

If the wind changes direction by more than 30^{o}, the decontamination station will need to be adjusted accordingly. A wait of 10 to 15 minutes to determine if the change is permanent should precede the move. When the station is moved, it must be moved at least 75 meters upwind from any contaminated area.

Security of Decontamination Site

When choosing a decontamination site, the same security considerations must be given as for any other site chosen for medical operations. The decontamination site is at the same potential risk from attack as is the actual MTF.

Area Control of Decontamination Site

An entry control point (ECP) can be established to control movement of clean and contaminated vehicles to the MTF or the Decontamination Station. The ECP should be located at a distance far enough from the MTF to keep vapor hazard from contaminated vehicles to the minimum.

Traffic control at the decontamination site involves routing a clearly marked, one-way course from the ECP to the decontamination station.

Control of personnel movement is necessary to ensure that contaminated walking personnel do not accidentally contaminate clean areas. The hot line must be secured. Concertina wire works well to keep personnel in the desired areas, and a clearly marked, one-way route helps to ensure that correct entry and exit points are used.

Litter Patient Decontamination

Personnel. Two people are required per litter patient. These two augmentees will link up with one litter patient in the triage area and work with that same litter patient until hand-off at the "hot line." These two people conduct both clothing removal and any required skin decontamination. To assist these two augmentees, two other augmentees will be needed, one to assist the first two augmentees in picking up the patient from the clothing removal litter, and the second to remove the contaminated clothing and litter and replace it with a clean litter. These four augmentees will conduct all patient decontamination and movement of the patient while in MOPP Level IV and the Toxicological Agent Protective (TAP) apron.

Personnel working in the patient decontamination area will be at MOPP level IV plus the Toxicological Agent Protective (TAP) apron. At least two people from this area will move to the triage area and carry the patient from this area to the first decontamination station.

Hypochlorite Solutions. Hypochlorite solution is used as an alternative decontaminant in patient decontamination; however, the

hypochlorite solution must be prepared. Two concentrations of the hypochlorite solution are required. A 5% solution is used to decontaminate gloves, aprons, litters, cutting devices, the patient's mask hood, and other non-skin contact areas. A 0.5% solution is used to decontaminate the patient's mask, skin, splints, and tourniquets and to irrigate their soft tissue wounds.

The chlorine solutions are placed in buckets for use in their particular area. The buckets should be distinctly marked because it is very difficult to tell the difference between the 5% and 0.5% chlorine solutions. These solutions may be made using the 6-ounce Calcium Hypochlorite (HTH) containers that come with the Chemical Agent Decon Set. The 0.5% solution can be made by adding one 6-ounce container of calcium hypochlorite to 5 gallons of water. The 5% CL solution is made by adding eight 6-ounce containers of calcium hypochlorite to 5 gallons of water. These solutions evaporate quickly at high temperatures, so if they are made in advance, they should be stored in closed containers.

Procedure

1. Decontaminate the hood and mask. Wipe/sponge down the voicemitter, eyelets and outserts with the M291 SDK, or 5% bleach solution. While wiping around the

filter, cover the inlet of the C2A1 filter canister with a hand or gauze momentarily to keep liquid out of the inside of the canister where it could wet the charcoal, reduce filter efficiency, and clog the filter. To decon the hood (quick doff hood or integrated hood/hooded overgarment), wipe down the hood using M291 SDK or 5% bleach solution (starting at the top of the head and wiping down towards the litter and shoulders).

2. Remove hood.

a. Remove the quick doff hood of the M40 mask
   (1) Dip the cutting device in a bucket of 5% bleach solution or decon/scrub-cutting tool with the M295 or M291.
   (2) Cut the hood shoulder straps.
   (3) Cut the quick-doff hood from the front bottom center to the chin through the elastic band under the chin.
   (4) Cut from the center of the forehead and over the top of the head toward the litter so that the hood will lay flat on the litter.

b. Remove the hood of the JSLIST.
   (1) Dip the cutting device in a bucket of 5% bleach solution or decon/scrub-cutting tool with the M295 or M291.

(2) Cut the hood starting at the front center and continue cutting across the top of the head toward the litter.
(3) Fold the left and right sides of the hood away from the head onto the litter.

3. Decontaminate head. After the hood is laying flat on the litter under the patient's head, decontaminate any exposed areas of the patient's head, hair, back of the ears, and neck that were not protected by the hood. The mask remains on the patient. This exposed skin is decontaminated using either the M291, soap and water, or 0.5% bleach solution. Do not use 5% bleach solution on skin.

4. Remove the Field Medical Card (FMC).

a. The medic at the litter patient decontamination station should view the FMC prior to removal.
b. Cut the patient's FMC tie wire, allowing the FMC to fall into a plastic, "zip-lock" bag. Seal the plastic bag and decontaminate the outside of the bag with the M291, M295, or rinse the outside of the bag with 0.5% bleach solution. Place the plastic bag with the FMC under the back of the protective mask head harness straps. The FMC will remain with the patient until he reaches the

hot line, where it will be transcribed onto a clean FMC.

5. Remove gross contamination from the patient's protective overgarment. Wipe all evident contamination spots with M291 SDK or 5% bleach solution.

6. Remove patient's personal effects from overgarment. Remove all items from the protective overgarment pockets and place them in a plastic "zip-lock" bag. Label the bag with the patient's identification and seal the bag. If the articles are not contaminated, place them in a separate bag from suspected contaminated items. Wipe down the outside of the bag with the M291 or dip it in a bucket of 5% hypochlorite solution. The bags are sent on the litter with the patient and checked for contamination at the contamination check area.

NOTE: The patient's identification tags stay around their neck throughout the decontamination process. They are decontaminated with soap and water, M291, or 0.5% bleach.

7. Cut the patient's overgarment.

a. The overgarment jacket and trousers may be cut simultaneously. Two persons may be cutting clothing at the same time.
b. Cutting is performed using sharp bandage scissors or long-handled seatbelt cutters (J knife).
c. Cut around bandages, tourniquets, and splints, leaving them in place. Only medical personnel remove bandages, tourniquets, and splints.

NOTE: Dip cutting device in a bucket of 5% bleach solution after each complete line of cut to avoid contaminating inner parts of the clothing or exposed skin. If bleach solution is not available, then cutting tools must be scrubbed using the M295 or M291.

8. Remove overgarment jacket.

a. Unfasten or cut the Velcro closures at the wrists.
b. Start cutting at the collar and make two cuts, one up each sleeve from the wrist to the shoulder and then to the collar. Keep the cuts close to the insides of the arms so that most of the sleeve material is folded outward. An alternative is to start at the collar and cut down the sleeve to the wrist.

c. Cut the jacket drawstring at the bottom of the jacket and unfasten Velcro closures, moving from the waist to the neck, and then unzip the jacket. If the jacket will not unzip, make a cut parallel to the zipper.
d. Carefully fold the sleeves of the overgarment away from the patient's arms, exposing only the black liner. Avoid aerosolizing any dust particles on the garment or allowing the outside of the garment to touch the patient.
e. Instruct the patient to keep his hands to the sides, away from the pieces of overgarment lying on his chest. If the casualty is unable to lift his arms, then one augmentee will hold the casualty's gloved hand and perform this action. Another augmentee then carefully folds the chest sections over the outside of the litter. The patient's arms are then lowered to the sides, keeping the arms away from the area where the overgarment has been removed.

9. Remove overgarment trousers.

a. Cut the trouser suspenders.
b. Cut the leg closure cord and hook and pile fasteners at the ankle cuff.
c. Cut from the ankle along the inseam of the left trouser leg until the crotch area is reached, and then cut across the zipper.

d. Cut along the inseam of the right trouser leg until the crotch area is reached, then go sideways into the first cut at the zipper.
e. Allow the trouser halves to drape over the sides of the litter. Carefully roll and tuck the remaining cloth (that at the crotch and on the inside of the legs) in on itself ensuring that only the black liner of the cloth is showing.

10. Remove outer gloves. Do not remove the inner gloves.

a. Decontaminate your own gloves with the M295, M291, or 5% bleach solution.
b. Decontaminate the casualty's gloves with the M295, M291, or 5% bleach solution.
c. Instruct the casualty to hold his arms away from the litter and upper body or, if he cannot comply with instructions, hold his gloves by the fingers.

NOTE: Always remove the gloves over the sides of the litter.

d. Grasp the cuff of the glove, turning the glove inside out, and remove it.
e. Carefully lower the patient’s arm(s) across his chest as each glove is removed. Avoid touching the patient’s cloth glove liner or arm with your rubber glove.

CAUTION: Do not allow the arms to contact the exterior (camouflage) side of the overgarment.

f. Dispose of the contaminated gloves by placing them in a trash bag.
g. Decontaminate your own gloves with the M295, M291, or 5% bleach solution.

11. Remove black vinyl overboots (BVO) or Multi-purpose overboot (MULO).

a. Unfasten the three elastic closures.
b. Gently pull the overboot by the heel until it is removed.
c. If the overboot will not come off, cut the boot from top to bottom along the centerline of the boot or along the inside of the boot. Fold the overboot down and gently pull the heel until it is removed.

12. Remove personal effects from BDU. Place personal effects in a plastic, zip-lock bag. This can be the same bag used for items taken from the overgarment pockets if they were not contaminated; otherwise, place these items in a separate bag. Seal the bag and decontaminate the outside of the bag. Keep the bag with the patient or take to a contaminated item holding area where they can be decontaminated.

13. Remove combat boots without touching body surfaces.

a. Cut the boot laces along the tongue.
b. Pull the boots downward and toward you until removed.
c. Place the boots in the plastic bag containing the chemical overboots and gloves.

14. Remove inner clothing.

a. Cut or unbuckle belt.
b. Cut the BDU pants following the same procedures as for the overgarment trousers.
c. Cut the BDU jacket following the same procedures as for the overgarment jacket.

15. Remove undergarments.

a. Remove the patient's T-shirt. Dip the cutting device in the 5% hypochlorite solution between each cut. Cut both sleeves from the inside, starting at the elbow, up to the armpit. Continue cutting across the shoulder to the collar. Cut around bandages or splints, leaving them in place. Next, peel the T-shirt away from the body to avoid spreading contamination.
b. If the patient is wearing a brassiere, cut it between the cups. Cut both shoulder straps where they attach to the cups and lay them back off of the shoulders.

c. Remove the patient's undershorts/panties by cutting from the lower side of the hip to the waist on both sides.
d. Remove the socks and cotton glove liners.
e. Do not remove the patient's identification tags.

16. Litter transfer and decontamination. Transfer the patient to a decontamination litter. After the patient's clothing has been cut away, he is transferred to a decontaminable litter. The decontamination team members decontaminate their gloves and apron with the 0.5% hypochlorite solution or M291. One member places his hands under the patient's legs and thigh, a second member places his arms under the patient's back and buttocks, and the third member places his arms under the patient's shoulders and supports the head and neck. They carefully lift the patient using their knees, not their backs, to minimize back strain. While the patient is elevated, another decontamination team member removes the litter from the litter stands and another member replaces it with a clean decontaminable litter. The patient is carefully lowered onto the clean litter. Two decontamination members carry the litter to the skin decontamination station. The contaminated clothing and overgarment are placed in bags and moved to the

decontaminated waste dump. The dirty litter is rinsed with the 5% bleach solution or M295 for reuse.

17. Skin and Wound Decontamination**.**

a. The casualty is now decontaminated with soap and water, the M291, or 0.5% bleach solution.
b. If the patient was in IPE, the best method is to decontaminate only those skin areas where there was a break in the IPE (e.g., around wounds, areas where the underlying uniform is wet with agent, or where there is a tear in the overgarment).
c. If the patient is not wearing IPE or had significant uniform tears or damaged underlying uniform, an alternate method is to decontaminate the entire skin surface by wiping the skin with a sponge and soapy water, or 0.5% bleach solution with a water rinse. Wash the casualty from the midline outward, constantly washing from clean to dirty and not placing a dirty sponge back on a clean area without first rinsing the sponge. The complete topside of the casualty is washed in this manner, paying particular attention to hairy areas of the body (groin and axillary regions) and sweaty areas (belt-line, just above the boots, the crease of the buttocks, and wrists). After log-rolling the patient onto his side, wash the backside of

the casualty. Then wash the casualty's back from the shoulders to over halfway down the backside, taking care not to miss any areas. The upper side of the litter is deconned prior to rolling the patient to their back again. Wash the opposite side of the casualty in exactly the same manner, and decontaminate the litter with soap and water.

d. After the patient is decontaminated, his dressings and tourniquet are changed. Superficial (not body cavities, eyes, or nervous tissue) wounds are flushed with the 0.5% CL solution and new dressings are applied as needed. Cover massive wounds with plastic or plastic bags. New tourniquets are placed 0.5 to 1 inch proximal to the original tourniquet, and then the old tourniquets are removed. Splints are not removed but saturated to the skin with 0.5% CL solution. If the splint cannot be saturated (air splint or canvas splint), it must be removed sufficiently so that everything below the splint can be saturated with the 0.5% CL solution. The patient, his wounds, and the decontaminable litter have now been completely decontaminated.

18. Final monitoring and movement to treatment area. The patient is monitored for contamination using the (ICAM) or M8 paper. Once the casualty is confirmed clean

of chemical agent, he is transferred via a shuffle pit over the hot line. The shuffle pit is composed of two parts Super Tropical Bleach (STB) and three parts earth or sand. The shuffle pit should be deep enough to cover the bottom of the protective overboots. The buddy system wash of the TAP apron and gloves in 0.5% hypochlorite solution precedes the transfer of the patient to a new, clean canvas litter if the decontaminable stretchers are in limited supply. A three-person patient lift is again used as the litter is switched. If the litter as well as the patient was checked, both patient and the same litter can be placed over the hot line.

AMBULATORY PATIENT DECON

Casualties that are decontaminated in an ambulatory area are those who require treatment that can be provided in the clean treatment area/MTF. These casualties will have their clothing removed during the decontamination process before crossing the hotline to the clean side.

Casualties who require only minimal care will undergo spot decon of their overgarment. They will be treated in the contaminated EMT area and return to duty.

Personnel

Personnel from the decontamination station might assist the casualty, or the casualties might assist each other during this process under close supervision.

Procedure

Decontamination of ambulatory casualties closely follows the methods described in FM 4-02.7 Health Service Support in an NBC Environment and FM 3-5, NBC Decontamination.

The step-by-step procedure outlined below is the prescribed doctrine for decontaminating an ambulatory patient, but it is by no means the only method. Knowing this method, however, ensures that correct and essential steps are not omitted, and when they are, other measures are taken to preclude a hazardous outcome.

The M291 Skin Decontamination Kit or a soap solution and water are used for chemical contamination on the skin. (The least desired alternative for skin decontamination is bleach [hypochlorite solution]). A 0.5% hypochlorite solution with water rinse is useful if water is limited and the M291 kits are not available. Only a 0.5% hypochlorite solution is used for skin decontamination; higher concentrations will

irritate and burn the skin, allowing agents to enter the skin more rapidly.

The M295 Decontamination Kit Individual Equipment is used to remove obvious contamination from the patient and help to control the spread of contamination on IPE (MOPP ensemble) and other equipment. If it is not available, then either soap solution or a field-expedient adsorbent material, such as clean dry earth or flour, can be substituted.

The first five steps are the same as in litter patient decontamination and are not described in detail.

1. Decontaminate mask and hood.

2. Remove hood.

3. Decontaminate head.

4. Remove the FMC.

5. Remove gross contamination from the outergarment.

6. Remove casualty's overgarment.

a. Cut overgarment around tourniquets, bandages, and splints. One augmentee medic or an augmentee will supervise the patients to cut one another's overgarments off if there are not adequate numbers of augmentees to assist.

NOTE: Dip cutting device in a bucket of 5% bleach solution after each complete line of cut to avoid contaminating inner parts of the clothing or exposed skin. If bleach solution is not available, cutting tools must be scrubbed using the M295 or M291.

b. Remove overgarment jacket by cutting.

(1) The casualty is standing and can hold on to a support such as a chair or litter stand.
(2) First, cut around all bandages and tourniquets.
(3) Cut the Velcro wrist closures.
(4) Cut the BDO jacket drawstring or the JSLIST drawcord at the jacket bottom. On the BDO, unzip the three snaps that connect the back of the BDO jacket and pants.
(5) Cut the BDO/JSLIST jacket starting at the waist and cut toward the collar in a line parallel to the zipper.
(6) Instruct the casualty to clench his fists; stand with arms held down and extended backward at about a 30-degree angle if the jacket was unzipped or cut in the front. If the jacket was cut along the rear have the patient extend the arms forward at about a 30-degree angle.
(7) Grasp the jacket collar at the sides of the neck and peel the jacket off the shoulders in a down and away motion, smoothly pulling the jacket inside out over the casualty's fists.
(8) Place the jacket in a plastic trash bag.

NOTE: The jacket may need to be cut along the sleeve if bandages are in the way and sleeves cannot be rolled over the bandaged area.

c. Remove the overgarment trousers by cutting.

(1) The casualty should have an object to help steady him in standing, such as a chair or litter stand.

NOTE: Do not cut the trouser suspenders until the end of the process; you do not want the trousers to fall and interfere with the cutter.

(2) Keep the pants zipped. Unfasten Velcro ankle fasteners and begin cutting at the ankle. Cut along the inseam and move up toward the waist of the trousers. After cutting both trouser legs from ankle to waist, cut each suspender and allow the trousers to fall to the ground. Take the trousers and lay them on the ground, black side up, next to the patient. Later the patient will step onto this as he removes his overboots.

NOTE: After each long cut, dip the cutting device in a bucket of 5% bleach solution or decon the scrub-cutting tool with the M295 or M291.

7. Remove the overboots.

a. Unfasten all boot closures.
b. Step on the heel of the boot and have the patient step out of the overboot and onto the black side of the cut trousers that are lying on the ground. Repeat this process for both boots. The overboots can be decontaminated and issued to other individuals.
c. If the overboot will not come off, cut the boot from top to bottom along the centerline of the boot until the boot is loose enough to remove.

8. Remove outer gloves. Do not remove the inner glove liners.
a. Decontaminate your own gloves with the M295, M291, or 5% bleach solution.
b. Decontaminate the casualty's gloves with the M295, M291, or 5% bleach solution.
c. Instruct the casualty to hold his arms up, if possible, and away from his upper body. If the patient cannot do this, hold his gloves at the fingers.
d. Grasp the cuff of the glove.
e. Pull the cuff over the fingers, turning the glove inside out.
f. Dispose of the contaminated gloves by placing them in a trash bag.
g. Decontaminate your own gloves again with the M295, M291, or 5% bleach solution.

9. Remove inner gloves. The patient should remove the liners to reduce the possibility of spreading contamination. The augmentee instructs the casualty to remove the white glove inner liner using the following guidance:

a. Grasp heel of glove liner without touching exposed skin.
b. Peel liner downward and off.
c. Drop it into the plastic trash bag.
d. Remove the remaining liner in the same manner.
e. Drop it into the plastic trash bag.
f. The patient then moves to the monitoring station.

NOTE: Waste material from two ambulatory patients, including the cut trousers, are placed into one 35-gallon trash bag along with the 5% bleach and soapy water that was used on the two patients. Tie the bag shut and transport it to the dirty dump.

10. Monitor xx.

a. Monitor with ICAM or M8 detection paper.
b. Check all areas of the casualty's clothing and combat boots. Pay particular attention to:
   (1) Combat boots
   (2) Protective mask
   (3) Hair and neck area
   (4) Discolored areas
   (5) Damp spots
   (6) Wrist closure area
   (7) Areas under tears in BDO
   (8) Around dressings and splints
c. If clean, send the casualty to the hot line.
d. If contaminated areas are found, decontaminate the areas using the M291 or soap and water. If the BDOs are contaminated they must be removed (see following). After decontamination or BDO removal, recheck the area with the ICAM or the M8 detection paper.

11. Remove the BDU.

a. Remove personal effects from BDU
   (1) Have the casualty remove all items from his BDU and deposit them into a zip-lock bag.
   (2) Check for contamination. If not contaminated, they remain with the

patient. If contaminated, they are moved to a contaminated item holding area.

b. Remove inner clothing (if contaminated).

(1) Cut or unbuckle belt.
(2) Cut the BDU pants following the same procedures as for the overgarment trousers.
(3) Cut the BDU jacket following the same procedures as for the overgarment jacket.

c. Remove undergarments (if contaminated).

(1) Remove the patient's T-shirt.
(a) Dip cutting devices in 5% bleach solution or scrub them with the M295 or the M291 between each cut.
(b) Cut around bandages or splints, leaving them in place.
(c) Cut up the front (back) of the patient's T-shirt from the waist up to the collar.
(d) Cut both sleeves from the elbow to the shoulder and then to the collar.
(e) Next, peel the T-shirt away from the body to avoid spreading contamination.

(2) Remove the patient's brassiere.

(a) Cut it between the cups.
(b) Cut both shoulder straps where they attach to the cups and remove the brassiere.

(3) Remove the patient's undershorts/panties.
(a) Cut from the lower side of the hip to the waist on both sides.
(b) Place the undergarments into the plastic garbage bag containing the other contaminated items.

12. Check Patient for Contamination.

a. After the patient's BDU and underwear have been removed, check the skin, hair, and boots for contamination by using M8 detector paper or the ICAM.
b. Carefully survey all areas of the patient's skin, paying particular attention to areas around the neck, wrist, ears, dressings, and splints.

13. Final Decontamination. At the contamination check area, use the M291, soap and water, or a 0.5% hypochlorite solution followed by a water rinse on any areas of the patient that still indicate contamination.

14. Remove any contaminated bandages and tourniquets (the medic does this procedure).

a. Place new tourniquets 1/2 to 1 inch above the old tourniquets.
b. Remove old tourniquets.
c. Decontaminate the exposed skin area.
d. Cut away bandages.
e. Decontaminate the exposed skin area.
f. Replace bandages only to control bleeding.
g. Decontaminate exposed skin.

15. Conduct final check for completeness of decontamination with the ICAM or M8 detection paper.

16. Move to the hot line. The augmentee instructs the patient to move 10 to 30 meters to the shuffle pit/hot line.

The Hot-Line and Clean Side Actions for the Ambulatory Patient

NOTE: Straddling the hot line is the casualty pass-over point, which is in a shuffle pit. The shuffle pit is composed of two parts super tropical bleach (STB) and three parts earth (by volume). The ambulatory patient shuffle pit should be wide enough for the ambulatory patient and two assistants.

1. At the shuffle pit, an augmentee from the clean side meets the patient and opens a blanket or other covering for the patient that is appropriate for the environmental conditions.

2. The patient shuffles through the shuffle pit wearing combat boots.

3. Once across the vapor control line, the ambulatory patient can remove his mask.

4. In the **clean treatment area**, the patient is now retriaged, treated, and evacuated.

a. In a hot climate, the patient will probably be significantly dehydrated; the rehydration process must begin immediately.
b. Overhead cover should be provided for casualties in the holding area. It is here that the mask may be removed for treatment unless circumstances dictate that the casualty remains closer to the Hot line.

Comments

The clean area is the resupply point for the patient decontamination station. Water is needed for rehydration of persons working in the decontamination area. The resupply section should have an adequate stock of canteens with the M1 cap.

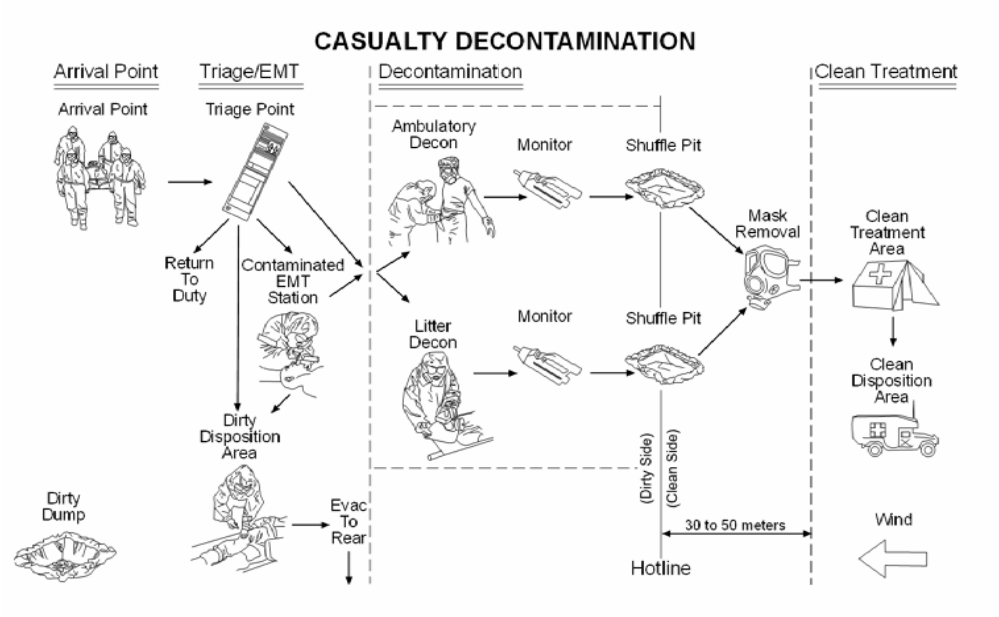
An area should be established 50 meters perpendicular to the litter casualty decontamination line and approximately 5 meters from the hot line for workers to use as a rest and rehydration point. Prior to using this point, workers must decontaminate the TAP aprons they are wearing using a 5% bleach solution and doff the apron near the decontamination line. Mask quick doff hoods, if worn, should also be decontaminated. After completion of this decontamination process, the warfighters are checked for contamination; then moved to the rest/rehydration point. If it is not safe to remove the mask, warfighters can rehydrate using their mask drink tubes.

APPENDIX B

PATIENT DECONTAMINATION STATION

The diagram shows a set-up for casualty management in a contaminated environment. The chapter on casualty management describes the various areas.

The actual set-up of this station may vary depending on the assets and circumstances.


CASUALTY DECONTAMINATION
Arrival Point
Triage/EMT
Decontamination
Clean Treatment
Arrival Point
Triage Point
Ambulatory Decon
Monitor
Shuffle Pit
Mask Removal
Clean Treatment Area
Return To Duty
Contaminated EMT Station
Litter Decon
Monitor
Shuffle Pit
Clean Disposition Area
Dirty Disposition Area
(Dirty Side)
(Clean Side)
Dirty Dump
Evac To Rear
30 to 50 meters
Wind
Hotline

APPENDIX C

TOXICITY DATA

The following tables provide estimated human toxicity data on the agents discussed in this handbook.

Agent	Effect	Ct_{50} (mg-min/m^3)	Liquid on skin
GA	Miosis	~2-3	
	Death	200-400	
GB.	Miosis	~3	
	Death	100-200	
GD.	Miosis	~2-3	
	Death	50-70	
VX	Death	10-50	
HD	Eye	12-200	
HD	Pulmonary	100-200	
	Erythema	200-1000	10 µg
	Death	1500 inhalation 10,000 skin	100 mg/kg
L	Erythema	>1500	10-15 µg
	Death	~1500 inhalation	40-50 mg/kg

Agent	Effect	Ct_{50} (mg-min/m^3)	Liquid on skin
CX	Eye	200?	
	Erythema	2500?	
	Death	3200?	
CG	Pulmonary effects	>1600	
	Death	3200	
AC	Death	2500-5000	
CK	Death	11,000	
CN	Irritation	10-20	
	Death	14,000	
CS	Irritation	5-10	
	Death	>50,000	

APPENDIX D
PHYSIOCHEMICAL DATA

The following tables provide physiochemical data on the agents discussed in this handbook.

	GA *Tabun*	**GB** *Sarin*	**GF**	**GD** *Soman*	**VX**
Molecular Weight	162	140	180	182	267
Vapor Density (compared to air)	5.63	4.86	6.2	6.33	9.2
Liquid Density (g/cc)	1.07@25°C	1.102@20°C	1.17@20°C	1.02@25°C	1.01@20°C
Freezing/Melting Point (°C)	-50	-56	-30	-42	-39
	GA *Tabun*	**GB** *Sarin*	**GF**	**GD** *Soman*	**VX**
Boiling Point (°C) (@ 750mm HG)	247	147	239 (@20ºC)	167	298
Vapor Pressure (mm HG @25°C)	0.07	2.9	0.04(@20º C)	0.4	0.0007
Volatility (mg/m^3 at 25°C)	610	17,000	581	3,900	10

	HD *(Distilled Mustard)*	**L** *(Lewisite)*	**CX** *(Phosgene Oxime)*
Molecular Weight	159	207	114
Vapor Density (compared to air)	5.4	7.1	3.9
Liquid Density(g/cc)	1.27@20^{o}C	1.89@20^{o}C	--
Freezing/Melting Point (^{o}C)	14.5	-18	35-40
Boiling Point (^{o}C) (@750mm HG)	(227.8)	190	128
Vapor Pressure (mm HG@ ^{o}C)	0.07@20^{o}C	0.2239@20^{o}C	11.2@25^{o}C
Volatility (mg/m^{3})	600@20^{o}C	4480@20^{o}C	1800@20^{o}C

	AC *(Hydrogen Cyanide)*	**CK** *(Cyanogen Choride)*	**CG** *(Phosgene)*
Molecular Weight	27	61.5	99
Vapor Density (compared to air)	0.94	2.1	3.4
Liquid Density(g/cc)	0.69	1.18	1.37
Freezing/Melting Point (^{o}C)	-13.3	-6.9	-128
Boiling Point (^{o}C) @750mm HG)	25.7	12.8	7.6
Vapor Pressure (mg/m^{3})	630@20^{o}C	1230@25^{o}C	1.17@20^{o}C
Volatility (mg/m^{3})	1,080,000@25^{o}C	2,600,000@12.8^{o}C	4,300,000@7.6^{o}C

	CN *(Mace)*	**CS**
Molecular Weight	154.59	189
Vapor Density (compared to air)	5.3	--
Liquid Density(g/cc)	1.32 (solid) @20ºC	1.04@20ºC
Freezing/Melting Point (^{o}C)	58	-94
Boiling Point (^{o}C) (@750mm HG)	248	315
Vapor Pressure (mm HG)	0.0041@20ºC	0.00034@20ºC
Volatility (mg/m^{3})	115@20ºC	0.71@20ºC

APPENDIX E

EQUIPMENT LIST

MEDICAL EQUIPMENT SET
CHEMICAL AGENT PATIENT TREATMENT
(30 patients)
6545-01-5187565, MES CHEM AG TRMT-2003

4240014422314	HOSE ASSEMBLY, AIR B	EA	12
4240014428415	BLOWER, LIGHTWEIGHT	EA	12
4720014061637	HOSE METALLIC	EA	12
6130015009675	BATTERY CHARGER 4POSI	EA	1
6140015009672	RECHARGEABLE BATTERY	EA	24
6505009269083	ATROPINE INJ 0.7ML	EA	500
6505011253248	PRALIDOXIM CHL INJ2ML	EA	50
6505013321281	ATROPINE SULF INHAL6S	PG	1
6505014542525	ATROPINE SULFATE OPHT	TU	12
6505014578901	ANTIDOTE TREATMENT	EA	5
6505015053476	DIAZEPAM INJ 2ML 10S	PG	10

6515006878052	AIRWAY PHARY 100MM10S	PG	1
6515007540412	SYRINGE HYPODERMI100S	PG	1
6515007542834	NEEDLE HYPO 18GA 100S	PG	2
6515009582232	AIRWAY PHARYN 80MM10S	PG	1
6515013386602	RESUSCITATOR HAND OPR	EA	4
6515014123099	SYRINGE HYPODERMIC20S	PG	1
6515014355350	SUCTION APPAR OROPHAR	EA	2
6515015195876	ADMINISTRATION SET60S	PG	1
6530013836260	WRAP AND LITTER PAT	EA	12
6530015196886	BAG STERILIZATION 10S	PG	1
6545009143490	CHEST NO4 30X18X12 EM	EA	3
6640015007717	CARTRIDGE RESPIRATOR	EA	48
6640015007721	INDICATOR AIRFLOW	EA	1
7520009357135	PEN BALL-POINT BLACK	DZ	1
8415011382503	GLOVES CHEM PROT LGE PR	PR	4

MEDICAL EQUIPMENT SET
CHEMICAL AGENT DECONTAMINATION
(60 patients)
6564-01-15187568, CHEM AGT PA-2003

6515009357138	SCISSORS BANDAGE7.25"	EA	15
6515011688108	SYRINGE HYPO 10ML100S	PG	1
6515014123099	SYRINGE HYPODERMIC20S	PG	1
6530006600034	SUPPORT LITTER FOLDNG	PR	8
6530013807309	LITTER FOLDING 91.60"	EA	4
6545009143490	CHEST NO4 30X18X12 EM	EA	3
6545009143510	CHEST MED INS SUP NO6	EA	1
6665000508529	PAPER CHEM AGENT 25S	BK	6
6665012265589	PAPER CHEMICAL AGEN	RO	1
6840002550471	DISINFECTANT CALC 6OZ	BT	49
6850012761905	DECONTAMINATING KIT	BX	2
7240002461097	PAIL UTILITY PLAS3GAL	EA	10
7520009357135	PEN BALL-POINT BLACK	DZ	1
7540014608995	FORM PRINTED	BK	1

7920008841115	SPONGE CELLULOSE RECT	EA	60
8105001913902	BAG PLASTIC	RO	2
8135006181783	PLASTIC SHEET RO POLY	RO	1
8415002817813	APRON TAP SMALL	EA	2
8415002817814	APRON TAP MEDIUM	EA	4
8415002817815	APRON TAP LARGE	EA	2
8415010333517	GLOVE SET CHEM PROTEC	SE	2
8415010333518	GLOVE SET CHEM PROTEC	SE	4
8415010333519	GLOVE SET CHEM PROTEC	SE	4
8415011382494	GLOVE INSERTS CHEM SM	PR	25
8415011382495	GLOVE INSERTS MEDIUM	PR	25

MCU-2A/P Protective Mask

Item	Size	NSN
MCU-2A/P Protective Mask	S	4240-01-327-4148
MCU-2A/P Protective Mask	M	4240-01-327-4149

MCU-2A/P Protective Mask	L	4240-01-327-4150

M40-A1 Protective Mask

Item	Size	NSN
M40-A1, Protective Mask	S	4240-01-258-0061
M40-A1, Protective Mask	M	4240-01-258-0062
M40-A1, Protective Mask	L	4240-01-258-0063

M42-A2 Protective Mask

Item	Size	NSN
M42-A2, Protective Mask	S	4240-01-413-4100
M42-A2, Protective Mask	M	4240-01-413-4101
M42-A2, Protective Mask	L	4240-01-413-4102

M45 Protective Mask

Item	Size	NSN
M45 Protective Mask	XS	4240-01-414-4034
M45 Protective Mask	S	4240-01-414-4035
M45 Protective Mask	M	4240-01-414-4051
M45 Protective Mask	L	4240-01-414-4052

DESERT JSLIST COAT

Item	Size	NSN
JSLIST COAT DESERT	XLL	8415-01-505-1616
JSLIST COAT DESERT	XLR	8415-01-509-8314
JSLIST COAT DESERT	2XLL	8415-01-505-1622
JSLIST COAT DESERT	3XLL	8415-01-506-7710

JSLIST COAT DESERT	LL	8415-01-444-6131
JSLIST COAT DESERT	LR	8415-01-444-6138
JSLIST COAT DESERT	ML	8415-01-444-6131
JSLIST COAT DESERT	MR	8415-01-444-5926
JSLIST COAT DESERT	MS	8415-01-444-5913
JSLIST COAT DESERT	SS	8415-01-444-5905
JSLIST COAT DESERT	SXS	8415-01-444-5902

DESERT JSLIST TROUSERS

Item	Size	NSN
JSLIST TROUSERS DESERT	XLL	8415-01-505-1567
JSLIST TROUSERS DESERT	XLR	8415-01-509-8269

JSLIST TROUSERS DESERT	2XLL	8415-01-505-1591
JSLIST TROUSERS DESERT	3XLL	8415-01-506-7713
JSLIST TROUSERS DESERT	LL	8415-01-444-5900
JSLIST TROUSERS DESERT	LR	8415-01-444-5898
JSLIST TROUSERS DESERT	ML	8415-01-444-5892
JSLIST TROUSERS DESERT	MR	8415-01-444-5893
JSLIST TROUSERS DESERT	MS	8415-01-444-5506
JSLIST TROUSERS DESERT	SS	8415-01-444-5504

JSLIST TROUSERS DESERT	SXS	8415-01-444-5417

WOODLAND JSLIST COAT

Item	Size	NSN
JSLIST COAT WOODLAND	XLL	8415-01-444-1241
JSLIST COAT WOODLAND	XLR	8415-01-509-8296
JSLIST COAT WOODLAND	2XLL	8415-01-505-1591
JSLIST COAT WOODLAND	3XLL	8415-01-506-7546
JSLIST COAT WOODLAND	LL	8415-01-444-1270
JSLIST COAT WOODLAND	LR	8415-01-444-1265
JSLIST COAT WOODLAND	ML	8415-01-444-1249
JSLIST COAT WOODLAND	MR	8415-01-444-1238

JSLIST COAT WOODLAND	MS	8415-01-444-1200
JSLIST COAT WOODLAND	SS	8415-01-444-1169
JSLIST COAT WOODLAND	SXS	8415-01-444-1163

WOODLAND JSLIST TROUSERS

Item	Size	NSN
JSLIST TROUSERS WOODLAND	XLL	8415-01-505-1274
JSLIST TROUSERS WOODLAND	XLR	8415-01-509-8265
JSLIST TROUSERS WOODLAND	2XLL	8415-01-505-1591
JSLIST TROUSERS WOODLAND	3XLL	8415-01-506-7698
JSLIST TROUSERS WOODLAND	LL	8415-01-444-2338

JSLIST TROUSERS WOODLAND	LR	8415-01-444-2325
JSLIST TROUSERS WOODLAND	ML	8415-01-444-2308
JSLIST TROUSERS WOODLAND	MR	8415-01-444-2310
JSLIST TROUSERS WOODLAND	MS	8415-01-444-1613
JSLIST TROUSERS WOODLAND	SS	8415-01-444-1439
JSLIST TROUSERS WOODLAND	SXS	8415-01-444-1435

BDO (Four Color Woodland Camouflage Pattern)

Item	Size	NSN
BDO	XXL	8415-01-137-1707
BDO	XL	8415-01-137-1706
BDO	LR	8415-01-137-1705

BDO	MD	8415-01-137-1704
BDO	SM	8415-01-137-1703
BDO	XS	8415-01-137-1702
BDO	XXS	8415-01-137-1701
BDO	XXXS	8415-01-137-1700

DBDO (Three Color Desert Camouflage Pattern)

Item	Size	NSN
DBDO	XXL	8415-01-327-5353
DBDO	XL	8415-01-327-5352
DBDO	LR	8415-01-327-5351
DBDO	MD	8415-01-327-5350
DBDO	SM	8415-01-327-5349
DBDO	XS	8415-01-327-5348
DBDO	XXS	8415-01-327-5347
DBDO	XXXS	8415-01-327-5346

Suit Contamination Avoidance and Liquid Protective (SCALP)

Item	Size	NSN
Footwear Covers, Liquid Contamination	SM	8430-01-364-3458
Footwear Covers, Liquid Contamination	M/L	8430-01-364-3459
Footwear Covers, Liquid Contamination	XL/XXL	8430-01-364-3460
Clothing Outfit, Liquid Contamination, Trousers, Poncho Attached Hood (Green)	SM	8415-01-364-3320
Clothing Outfit, Liquid Contamination, Trousers, Poncho Attached Hood (Green)	M/L	8415-01-364-3321

Clothing Outfit, Liquid Contamination, Trousers, Poncho Attached Hood (Green)	XL/XXL	8415-01-364-3322
Clothing Outfit, Liquid Contamination, Trousers, Poncho Attached Hood (Tan)	SM	8415-01-333-0987
Clothing Outfit, Liquid Contamination, Trousers, Poncho Attached Hood (Tan)	M/L	8415-01-333-0988
Clothing Outfit, Liquid Contamination, Trousers, Poncho Attached Hood (Tan)	XL/XXL	8415-01-333-0989

Chemical Protective Glove Sets

Item	Size	NSN
25-Mil Gloves with liners Butyl	XS	8415-01-144-1862
25-Mil Gloves with liners Butyl	SM	8415-01-033-3517
25-Mil Gloves with liners Butyl	MD	8415-01-033-3518
25-Mil Gloves with liners Butyl	LG	8415-01-033-3519
25-Mil Gloves with liners Butyl	XL	8415-01-033-3520
14-Mil Gloves with liners Butyl	SM	8415-01-138-2497
14-Mil Gloves with liners Butyl	MD	8415-01-138-2498

14-Mil Gloves with liners Butyl	LG	8415-01-138-2499
14-Mil Gloves with liners Butyl	XL	8415-01-138-2500
7-Mil Gloves with liners Butyl	SM	8415-01-138-2501
7-Mil Gloves with liners Butyl	MD	8415-01-138-2502
7-Mil Gloves with liners Butyl	LG	8415-01-138-2503

Chemical Protective Overboots (MULO)

Item	Size	NSN
Overboot (Black) Multipurpose	3	8430-01-464-9453
Overboot (Black) Multipurpose	4	8430-01-464-9458

Overboot (Black) Multipurpose	5	8430-01-464-9459
Overboot (Black) Multipurpose	6	8430-01-464-9461
Overboot (Black) Multipurpose	7	8430-01-464-9462
Overboot (Black) Multipurpose	8	8430-01-464-9464
Overboot (Black) Multipurpose	9	8430-01-464-9474
Overboot (Black) Multipurpose	10	8430-01-464-9475
Overboot (Black) Multipurpose	11	8430-01-464-9477
Overboot (Black) Multipurpose	12	8430-01-464-9480

Overboot (Black) Multipurpose	13	8430-01-464-9479
Overboot (Black) Multipurpose	14	8430-01-464-9484

Chemical Protective Overboots (BVO)

Item	Size	NSN
Overboot (BVO)	3	8430-01-317-3374
Overboot (BVO)	4	8430-01-317-3375
Overboot (BVO)	5	8430-01-317-3376
Overboot (BVO)	6	8430-01-317-3377
Overboot (BVO)	7	8430-01-317-3378
Overboot (BVO)	8	8430-01-317-3379

Overboot (BVO)	9	8430-01-317-3380
Overboot (BVO)	10	8430-01-317-3381
Overboot (BVO)	11	8430-01-317-3382
Overboot (BVO)	12	8430-01-317-3383
Overboot (BVO)	13	8430-01-317-3384
Overboot (BVO)	14	8430-01-317-3385

Chemical Protective Overboots (GVO)

Item	Size	NSN
Overboot (GVO)	3	8430-01-084-6305
Overboot (GVO)	4	8430-01-084-6306
Overboot (GVO)	5	8430-01-049-0878

Overboot (GVO)	6	8430-01-049-0879
Overboot (GVO)	7	8430-01-049-0880
Overboot (GVO)	8	8430-01-049-0881
Overboot (GVO)	9	8430-01-049-0882
Overboot (GVO)	10	8430-01-049-0883
Overboot (GVO)	11	8430-01-049-0884
Overboot (GVO)	12	8430-01-049-0885
Overboot (GVO)	13	8430-01-049-0886
Overboot (GVO)	14	8430-01-049-0887

APPENDIX F

The following table is intended to serve as a reminder of the agents, their effects, first-aid measures, detection, and skin decontamination.

It is in no way complete, nor is it intended to be complete. Consult the appropriate chapter for further details.

Type of Agent	Effects	Onset	First-aid	Skin Decon	Field Detection
Pulmonary TIC: CG (PFIB, HC)	Dyspnea, coughing	Hours	None	None usually needed	None
Cyanide: AC, CK	Loss of consciousness, convulsions, apnea	Seconds	None (nitrite and thiosulfate)	None usually needed	M256A1 M18A2
Vesicants: H, HD, L	Erythema, blisters; irritation of eyes; cough, dyspnea Vapor: miosis, rhinorrhea, dyspnea	Hours (immediate pain after L) Vapor: seconds	None	M291, soap and water, 0.5% bleach	M256A1; M8 and M9 papers, CAM, ACADA, FOX, M90

Type of Agent	Effects	Onset	First-aid	Skin Decon	Field Detection
Nerve: GA, GB, GD, GF, VX	Vapor: miosis, rhinorrhea, dyspnea Liquid: sweating, vomiting Both: convulsions, apnea	Vapor: seconds Liquid: minutes to hours	MARK I (1 to 3); diazepam	M291, soap and water, 0.5% bleach	M256A1, M8 and M9 papers, CAM, M22 ACADA
Incapacitating: BZ, Agent 15	Mydriasis, increased body temperature; dry mouth and skin; confusion; visual hallucinations	Minutes to hours	Remove from harming themselves or others	Remove outer clothing; water, or soap and water	None
Riot-control: CS, CN	Burning, stinging of eyes, nose, airways, skin	Seconds	None	Water	None

APPENDIX G

Acronyms are helpful for remembering the toxicologically important aspects of a poisoned casualty. Choose the one that you find easiest to remember and commit it to memory. The logical progression of each acronym from agent through environment and to host should aid memorization.

ASBESTOS:

A: **A**gent(s): Type(s) and estimated doses

S: **S**tate(s): Solid, liquid, vapor, gas, aerosol

B: **B**ody sites: Where exposed (routes of entry) [exposure and absorption]

E: **E**ffects: Local vs. systemic

S: **S**everity: Of a) effects and b) exposure

T: **T**ime course: Past, present, and future [prognosis])

O: **O**ther diagnoses: (a) Instead of (DDx) and (b) in addition to (additional diagnoses)

S: **S**ynergism: Interaction among multiple co-existing diagnoses

TOXICANT:

T: **T**oxicant(s)/**T**oxidrome: Agent; does it fit with a specific toxidrome?
O: **O**utside the body: Form: Solid, liquid, vapor, gas, aerosol?
X: **X**ing into the body: Where did the agent cross into the body? [exposure and absorption]
I: **I**nside the body: Where did the agent go inside the body? [distribution]
C: **C**hronology: Time course of exposure (past, present, and future)
A: **A**dditional diagnoses: Possible co-existing diagnoses
N: **N**et effect of diagnoses: Interaction among all diagnoses; patient as a whole
T: **T**riage: Priority for treatment, decontamination, and transport

POISON:

P: **P**oison(s): Type(s) and estimated doses
O: **O**utside the body: Form: Solid, liquid, vapor, gas, aerosol?
I: **I**nto/**I**nside the body: Where did it get into the body and where did it go inside the body?
S: **S**equence of events: Time course [past, present, and future]

O: **O**ther diagnoses: a) Instead of (DDx) and b) in addition to (additional diagnoses)
N: **N**et effects of diagnoses: Interaction among all diagnoses; patient as a whole

The ABCDDs of Chemical Casualty Care

A: Airway attention should be focused on establishing an airway (by positioning or by procedures) if needed, and maintaining the airway. No matter what else is done to try to save a casualty, death will be guaranteed if the airway is lost.

B: Breathing may require intubation and ventilation of casualties. Keep in mind that once a casualty has been intubated, someone will be required to stay with the casualty to provide and monitor ventilations until the victim leaves the area, is capable of breathing spontaneously again, or expires or until the resources devoted to the casualty are critically required elsewhere to save other lives.

C: Circulation may require chest compressions to maintain circulation in the absence of effective cardiac contractions will probably not be feasible for more than a few patients in a mass-casualty event, and most casualties without a pulse will have to be triaged as expectant.

D: Decontamination: Immediate, along with thorough patient decontamination and technical decontamination constitutes one of the main types of personnel decontamination

D: Drugs, refers to specific antidotal treatment for selected agents and also to ancillary supportive medications.

Remembering and applying the ***ABCDDs*** is as crucial to organizing initial treatment as ***ASBESTOS***, ***POISON***, or ***TOXICANT*** is to a systematic evaluation of a chemical casualty.

APPENDIX H

GLOSSARY OF TERMS

ACAA. Automatic Chemical Agent Alarm.
ACADA. Automatic Chemical Agent Detector Alarm; this area monitoring detector sounds a warning when it senses the vapors of blister and nerve agents.
Acid. A substance with a pH less than 7.
Aerosol. A gaseous suspension of fine solid or liquid particles.
Alkali. A substance with a pH greater than 7.
Alveoli. Microscopic air sacs in the lungs where oxygen and carbon dioxide diffusion (movement) takes place through alveolar walls.
AMEDD. Army Medical Department.
Asphyxiation. Unconsciousness or death caused by lack of oxygen.
BAL. British Anti-Lewisite.
BDO. Battle Dress Overgarment.
BDU. Battle Dress Uniform.
Bronchi. The finer, smaller, divisions of the wind pipe into the lungs.
CAM. Chemical Agent Monitor.
CANA. Convulsive Antidote, Nerve Agent.
Capillaries. Small blood vessels.
CARC. Chemical Agent Resistant Coating.
C/B. Chemical/Biological.
CDC. Chemical Decontamination Center.

CBPS. Chemical and Biological Protective Shelter.
Central airway. The main airway that transports air from the nose and mouth to the lungs.
CPS. Chemical Protective Shelter.
C2A1 filter canister. The standard filter used on the military mask. Protects against historic chemical warfare agents.
DAAMS. Depot Area Air Monitoring System.
DBDO. Desert Battle Dress Overgarment.
DTD. Detailed Troop Decontamination.
ECP. Entry Control Point.
EMT. Emergency Medical Treatment.
FMC. Field Medical Card.
GREGG. Graves Registration.
HC smoke. Military tactical smoke.
HTH. High Test Hypochlorite.
KPH. Kilometer Per Hour.
ICAD. Individual Chemical Agent Monitor.
Intubation. The process of enhancing respiration by providing an artificial airway.
IDLH. Immediately-dangerous-to-life-and-health.
JSLIST. Joint Service Lightweight Integrated Suit Technology.
Laryngospasm. Spasmodic closure of the larynx (voice box at the top of the trachea/wind pipe).
Larynx. Voicebox and vocal cords.
LBE. Load Bearing Equipment.
LCL. Liquid Control Line.
MCW. Mass-casualty Weapon.

MES. Medical Equipment Set.
MOPP. Mission Oriented Protective Posture.
MTF. Medical Treatment Facility.
MTO&E. Modified Table of Organization and Equipment.
NAAK. Nerve Agent Antidote Kit.
Nasopharynx. The area of the nose and upper airway.
NATO. North Atlantic Treaty Organization.
NBC. Nuclear/Biological/Chemical.
NCO. Noncommissioned Officer.
NCOIC. Noncommissioned Officer-in-Charge.
NOx. Toxic smoke that can cause pulmonary edema. Produced by exploding munitions, industrial smoke, and in grain silos as a product of grain fermentation.
OIC. Officer-in-Charge.
Oropharynx. The mouth and upper airway.
PFIB. Toxic smoke produced by Teflon® burning at more than 700^{o}F.
Pulmonary edema. Fluid in the lungs, associated with an outpouring of fluids from the capillaries into the pulmonary spaces (air sacs or alveoli) producing severe shortness of breath. In later stages, produces expectoration of frothy, pink, fluid and blue lips (cyanosis).
SDK. Skin Decontamination Kit.
TAP. Toxicological Agent Protective, e.g., TAP apron.
TC. Training Circular.
TIC. Toxic Industrial Chemical; a chemical with a toxicity equal to or greater than ammonia and is

produced more than 30 times a year by an industrial facility.
TIM. Toxic Industrial Materiel.
Trachea. Wind pipe.
Vapor. Fumes given off by a liquid.
VCL. Vapor Control Line.
WMD. Weapon of Mass Destruction.

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LUNG-DAMAGING AGENTS: Toxic Industrial Chemicals

Cl, CG

SUMMARY

Signs and Symptoms: Central effects: Eye and airway irritation, dyspnea. Peripheral effects: Chest tightness, and **delayed** pulmonary edema.

Field Detection: The Chemical Agent Detector Kit and the M93A1 FOX RECONNAISSANCE System will detect small concentrations of CG; however, they will not detect Cl.

Decontamination: **vapor** - fresh air; **liquid** - copious water irrigation.

Management: termination of exposure, ABCs of resuscitation, enforced rest and observation, oxygen with or without positive airway pressure for signs of respiratory distress, other supportive therapy as needed.

CYANIDE

AC, CK

SUMMARY

Signs and Symptoms: few. After exposure to high concentrations, seizures, respiratory and cardiac arrest.

Field Detection: The M256A1 Sampler-detector, M18A2, and M90 Chemical agent detectors detect Hydrogen Cyanide (AC) as vapor or gas in the air, and the M272 chemical water testing kit detects AC in water.

Decontamination: Skin decontamination is usually not necessary because the agents are highly volatile. Wet, contaminated clothing should be removed and the underlying skin decontaminated with water or other standard decontaminants to prevent off-gassing as a hazard.

Management: Antidote: intravenous (IV) sodium nitrite and sodium thiosulfate. ***Supportive:*** oxygen, correct acidosis.

MUSTARD

HD, H

SUMMARY

Signs and Symptoms: asymptomatic latent period (hours). Erythema and blisters on the **skin, chemical burns**; irritation, conjunctivitis, corneal opacity, and damage in the **eyes**; mild upper respiratory signs to marked **airway** damage; also gastrointestinal (GI) effects and bone marrow stem cell suppression.

Field Detection: M256A1 kit, M18A2 chemical agent detector kits; Individual Chemical Agent Alarm (ICAM), M90 chemical agents detector, M8 and M9 chemical agent detector paper, M21 Remote Sensing Chemical Agents Alarm (RSCAAL), M93A1 FOX NBC RECONNAISSANCE System, M272 Chemical water testing kit, M22 Automatic Chemical Agent (ACADA) Detection Alarm.

Decontamination: M291, soap and water, 0.5% bleach solution.

Management: Decontamination immediately after exposure is the only way to prevent damage. Supportive care of patients - there is no specific therapy.

LEWISITE

L

SUMMARY

Signs and Symptoms: Lewisite causes immediate pain or irritation of skin and mucous membranes. Erythema and blisters on the skin and eye and airway damage similar to those seen after mustard exposures develop later.

Field Detection: M256A1 kit, M18A2 chemical agent detector kits; Individual Chemical Agent Alarm (ICAM), M90 chemical agents detector, M8 and M9 chemical agent detector paper, M21 Remote Sensing Chemical Agents Alarm (RSCAAL), M93A1 FOX NBC RECONNAISSANCE System, M272 Chemical water testing kit, M22 Automatic Chemical Agent (ACADA) Detection Alarm.

Decontamination: M291, soap and water, 0.5% bleach solution.

Management: Immediate decontamination; symptomatic management of lesions the same as for mustard lesions; a specific antidote (BAL) will decrease systemic effects.

PHOSGENE OXIME
CX

SUMMARY

Signs and Symptoms: Immediate burning and irritation followed by wheal-like skin lesions and eye and airway damage.

Field Detection: M256A1 and M18A2 chemical agent detector kits; M90 chemical agent detector, M93A1 FOX NBC RECONNAISSANCE SYSTEM.

Decontamination: M291, soap and water, 0.5% bleach solution.

Management: Immediate decontamination, symptomatic management of lesions.

NERVE AGENTS

SUMMARY

Signs and Symptoms:

Vapor:

Small exposure -- miosis, rhinorrhea, mild difficulty breathing.

Large exposure -- sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions, miosis.

Liquid on skin:
Small to moderate exposure -- localized sweating, nausea, vomiting, feeling of weakness.
Large exposure -- sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions.

Field Detection: M256A1 chemical agent detector kit, M8 chemical agent detector paper, M9 chemical agent detector paper; Improved Chemical Agent Alarm (ICAM), M93A1 FOX NBC RECONNAISSANCE System, M18A2 chemical agent detector kit, M21 Remote Sensing Chemical Agent Alarm (RSCAAL), M90 chemical agent detector, M22 Automatic Chemical Agents Detection Alarm (ACADA).

Decontamination: M291 SDK, soap and water, 0.5% Hypochlorate solution.

Immediate management: Administration of MARK I Kits (atropine and pralidoxime chloride); diazepam in addition if casualty is severe; ventilation and suction of airways for respiratory distress.

INCAPACITATING AGENTS
BZ, Fentanyl Derivatives

SUMMARY

Signs and Symptoms: **BZ:** mydriasis; dry mouth; hot, red, dry skin; increased DTRs; decreased level of concentration; disturbance in perception and interpretation (illusions and/or hallucinations); denial of illness; short attention span; impaired memory. **Fentanyl (carfentanil):** miosis, drowsiness, unconsciousness, vomiting, central apnea.

Detection: No field detector is available for BZ or fentanyl derivatives.

Decontamination: **BZ:** Remove clothing. Gentle, but thorough washing of skin and hair with water or soap and water is required. Bleach is not necessary. **Fentanyl (carfentanil):** no decontamination needed.

Management: **BZ Antidote:** physostigmine. **Supportive:** monitoring of vital signs, especially core temperature. **Fentanyl (carfentanil): Antidote:** Naloxone/naltrexone. **Supportive:** monitoring of vital signs, airway maintenance is critical until the effects of the agent wear off.

RIOT-CONTROL AGENTS
CS, CN, CR, and OC

SUMMARY

Signs and Symptoms: **CS, CN, CR, and OC:** feeling of intense burning and pain on exposed mucous membranes and skin, eye pain and tearing, burning in the nostrils, respiratory discomfort, and tingling of the exposed skin. With severe exposure: laryngospasm which can compromise breathing if not attended to. **CS, CN, CR,** with severe exposure: respiratory discomfort and skin blistering. **DM:** delayed skin irritation by several minutes, vomiting and malaise that can last for hours.

Field Detection: No field detector is available.

Decontamination: **Eyes:** thoroughly flush with water, saline, or similar substance. **Skin:** flush with copious amounts of water, alkaline soap and water, or a mildly alkaline solution (sodium bicarbonate or sodium carbonate). Generally, decontamination is not needed if the wind is brisk. Hypochlorite exacerbates the skin lesion and should not be used. **OC:** use of water for decontamination will increase pain. If water must be used it must be in copious amounts. OC is best decontaminated using baby shampoo, milk, or vegetable oil. Pain will diminish over time, once the individual's substance P is depleted.

Immediate management: Usually none is necessary; effects are self-limiting.