ACT FAST

Agent Characteristics and Toxicology
First Aid and Special Treatment

STUDENT GUIDE

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Prepared for the

Chemical Stockpile Emergency Preparedness Program

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ACRONYMS AND ABBREVIATIONS

ABC	airway, breathing, circulation
ACT FAST	Agent Characteristics and Toxicology: First Aid and Special Treatment
AEGL	Acute Exposure Guideline Level
C	Centigrade
cm	centimeter
CSEPP	Chemical Stockpile Emergency Preparedness Program
EMS	emergency medical service
EMT	emergency medical technician
F	Fahrenheit
FAST	First Aid and Special Treatment
g	gram
hr	hour
IM	intramuscularly
IV	intravenously
kg	kilogram
lb	pound
m	meter
mg	milligram
mg/m^3	milligram per cubic meter
min	minute
ml	milliliter
MMT	Meridian Medical Technologies, Inc.
mo	month
MSDS	material safety data sheet
OSHA	Occupational Safety and Health Administration

2-PAM Cl	pralidoxime chloride
PPE	personal protective equipment
USAMRICD	U.S. Army Medical Research Institute of Chemical Defense
yr	year

PREFACE

Chances are there will never be a need to treat or decontaminate patients who have been exposed to the chemical warfare agents stored at the local Army installation. The situations presented in this guide are worst case scenarios; they are not intended to suggest that release of chemicals is likely or that injuries would occur in the event of a release. The evidence suggests otherwise. But, we believe in being prepared and want to help those who are responsible for providing initial emergency medical care to prepare for that low-probability event.

In the years since the earlier versions of the ACT FAST training materials were published, knowledge of the chemical warfare agents and understanding of their effects on the human body have advanced. In addition, procedures for treating civilian victims for exposure to the agents have been refined. This Third Edition of ACT FAST has been revised to incorporate the latest knowledge and treatment techniques.

MODULE 1 INTRODUCTION

Chemical warfare agents act quickly. The actions taken by emergency medical personnel in the first few minutes can mean the difference between life and death. Treatment must be immediate. There is no time to transport the patient and have a doctor or emergency department staff take the initial actions.

Because immediate treatment is so essential, this course takes as its title the acronym ACT FAST. It stands for Agent Characteristics & Toxicology: First Aid & Special Treatment.

1.1 Target Audience

This course is designed for all pre-hospital emergency medical personnel providing initial care and treatment of victims of a chemical warfare agent release. The training is primarily directed toward emergency medical technicians (EMTs) with basic life support credentials, but other emergency medical personnel will benefit from the information presented here.

1.2 Goal

The goal of ACT FAST is to prepare emergency medical personnel to respond to an off-post scene involving a release of chemical warfare agent and provide the appropriate treatment to people who have been exposed to the agent.

To achieve this goal, emergency medical personnel must learn to recognize the signs and symptoms of exposure to each type of chemical warfare agent, to assess the severity of exposure, and to take appropriate measures to protect themselves from exposure while providing treatment.

1.3 Training Objectives

At the end of this training program students should be prepared to perform the following two primary objectives:

1. Describe the initial first-aid treatment for victims of nerve agent exposure.

2. Describe the initial first-aid treatment for victims of blister agent exposure.

In order to master these two primary objectives, students must also be able to accomplish these secondary objectives:

- Describe the potential hazards of nerve agents: what they are, how they work, and potential routes of exposure.

- Describe the potential hazards of blister agents: what they are, how they work, and potential routes of exposure.

- Identify the signs and symptoms of nerve agent exposure.

- Identify the signs and symptoms of blister agent exposure.

1.4 Relation to Other CSEPP Training Materials

ACT FAST is interrelated with several other training courses that have been developed for CSEPP. Although these other courses contain information that is crucial to the ability to safely treat patients suffering from chemical agent exposure, their contents are not repeated in detail here. We recommend that, in addition to ACT FAST training, students complete the following CSEPP training courses:

- Chemical Awareness
- Decontamination of People Potentially Exposed to Chemical Agents,
- Personal Protective Equipment, and
- Use of Auto-Injectors by Civilian Emergency Medical Personnel.

The following CSEPP training videos may be viewed in conjunction with this class:

- Re-ACT FAST II,
- Don't Be a Victim: Medical Management of Patients Contaminated with Chemical Agent, and
- Exposure and Contamination.

State or local CSEPP offices may have identified other training courses or materials to prepare medical personnel to participate in the medical response to a chemical agent release. If so, these state or local CSEPP organizations will advise students of these additional training requirements.

1.5 Explanation of Modules

This Student Guide is divided into five (5) modules:

- Module 1—Introduction
- Module 2—Background Information
- Module 3—Chemical Agents: Characteristics and Effects
- Module 4—Signs and Symptoms
- Module 5—First Aid and Special Treatment (FAST)

Each module, except the Introduction, begins with a statement of the learning objectives for the module and concludes with a summary of the major information students should have learned from the module. The most important modules—numbers 3, 4, and 5—also include a self-check quiz so students can use to see how well they learned the key points of the module. Appendix A contains the correct answers to the self-check questions.

After completing ACT FAST, students can evaluate their mastery of the course material by taking the Final Exam in Appendix B. Answers to the exam questions are included in Appendix C.

1.6 How to Use this Guide

Classroom Training

It is recommended that this Student Guide be used as a reference for students attending the ACT FAST classroom training course. The class instructor's presentation and this guide have been designed to coincide so that note-taking can be minimized.

Self-Study

This guide can also be used for studying on one's own. For example, the guide may be used

- before attending the in-class session to do some pre-study,
- during the in-class session, as a guide for taking notes or to highlight information as the class progresses,
- after attending the in-class session, to keep as a reference and review resource,
- in place of attending the in-class session, as a self-study resource.

MODULE 2 BACKGROUND INFORMATION

Objectives

This module provides general information about the U.S. Army's chemical warfare agent stockpile. After completing this module students should be able to identify the chemical warfare agent(s) stored at their local Army installations.

Introduction

This module provides a brief description of the U.S. chemical agent stockpile. A more complete discussion of this topic can be found in the Chemical Awareness course materials. Students should pay particular attention to the names of the agent(s) stored at their local Army installation.

2.1 General Information on the Chemical Stockpile

What Is the "Chemical Stockpile"?

During the Cold War, the U.S. Army produced tons of chemicals designed for the sole purpose of warfare. They stored this "chemical stockpile" at eight locations in the continental U.S.

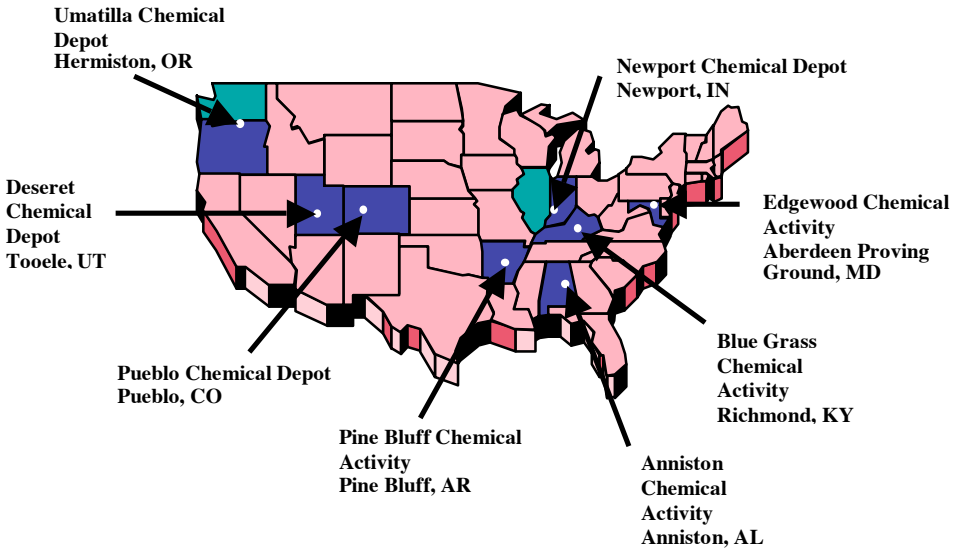


Figure 2.1. Location of CSEPP Sites

In August,1996, the Army began destroying or disposing of the chemical agents in the stockpile. As the disposal program progresses, the composition of the stockpile at each location continually changes. These changes are reflected in the following tables. Table 2.1 describes the stockpile that existed at the beginning of disposal activities; Table 2.2 describes the stockpile as it existed in September 2006. Notice that, by 2006, some

agents had been completely eliminated at some locations, and one location (Edgewood, MD) had destroyed all of the agent in its stockpile.

Table 2.1. U.S. Army Chemical Stockpile Inventory at Beginning of Disposal Activities (August 1996)[1]

Installation	Agent Stored				
	GA	GB	VX	Sulfur Mustard	Lewisite
Anniston		X	X	X	
Blue Grass		X	X	X	
Deseret	X	X	X	X	X
Edgewood				X	
Newport			X		
Pine Bluff		X	X	X	
Pueblo				X	
Umatilla		X	X	X	

Table 2.2. U.S. Army Chemical Stockpile Inventory in September 2006[2]

Installation	Agent Stored				
	GA	GB	VX	Sulfur Mustard	Lewisite
Anniston			X	X	
Blue Grass		X	X	X	
Deseret	X			X	
Edgewood					
Newport			X		
Pine Bluff		X	X	X	
Pueblo				X	
Umatilla		X	X	X	

The number of agents present at each stockpile location will continue to decline as the disposal program progresses. It is important to know which agents currently exist at the Army installation in each CSEPP community so that training for responders in that community can be tailored to prepare for responding to those specific hazards.

MODULE 3 CHEMICAL AGENTS: CHARACTERISTICS AND EFFECTS

Objectives

After completing Module 3, students should be able to:

1. Describe the potential hazards of nerve agents: what they are, how they work, and potential routes of exposure.
2. Describe the potential hazards of blister agents: what they are, how they work, and potential routes of exposure.
3. Identify the most likely routes of exposure.

Introduction

Before studying how to treat patients, it is necessary to understand a little about the agents themselves. Thus, Module 3 focuses on (1) what the agents are called, (2) their physical properties, and (3) how they affect the body. Understanding this information will help students learn how to treat patients exposed to blister and nerve agents while protecting themselves from secondary contamination.

The most important points to learn from this module are:

- Nerve agents
  - are stored as liquids (one is oily) that generate extremely toxic vapors
  - can be absorbed through direct contact with the skin or eyes, inhalation of contaminated air, and ingestion (swallowing) of contaminated food or drink

- Blister agents:
  - are stored as solids or liquids (thick and oily), but can generate toxic vapors
  - can be absorbed through direct contact with the skin or eyes, inhalation of contaminated air, and ingestion (swallowing) of contaminated food or drink

- The most likely route of exposure pre-hospital emergency medical personnel should be prepared for is inhalation of contaminated air

3.1 Nerve Agents: A Description

Toxic chemical warfare agents classified as “nerve agents” are so called because they are capable of attacking the body’s nervous system. The stockpiled nerve agents are organophosphates, a scientific classification that describes a group of compounds that inhibit cholinesterase, a naturally-occurring enzyme that is important to normal nervous and muscular system function. The organophosphate nerve agents are much more potent versions of organophosphate pesticides such as malathion and parathion.

Specific Names

While the term “nerve agent” refers to a general class of toxic chemical, specific common and chemical names are used for the individual chemical compounds within this group. They are: VX, GA, and GB. When the term “nerve agent” is used in this course it refers to all of the agents (VX, GA, and GB) unless a specific agent is mentioned. The chemical agent stockpile contains only a small amount of agent GA which is stored at Deseret Chemical Depot in Utah.

Abbreviation	Common Name	Referred To As
VX		VX
GA	Tabun	GA or G-agent
GB	Sarin	GB or G-agent

Students interested in learning more about the different nerve agents can refer to the Material Safety Data Sheets (MSDSs) in Appendix D. The U.S. Army’s Edgewood Chemical Biological Center developed these documents to provide technical details on the agents’ chemical formulas, structures, names, physical data, and hazard characteristics. Additional information may be found in volume 3 of the Textbook of Military Medicine[8]. However, this course does not require that students know about nerve agents to the level of detail described in these outside sources.

Physical Properties

Nerve agents are stored in the chemical stockpile in liquid form. These liquids are volatile—they generate vapors. It is the vapor form of agent that has the greatest potential of affecting people outside the Army stockpile facilities. There may also be some potential for agent being released as an aerosol. Once in a vapor or aerosol form and mixed with the outside air, the agent may move from the storage location.

The physical characteristics of nerve agents are described in some detail in the student guide for the Chemical Awareness course. The major properties of these agents are summarized in Table 3.1.

Table 3.1 Nerve Agent Comparative Data

	GA[3]	GB[4]	VX[5]
Appearance	Colorless to brown liquid	Colorless, watery liquid	Colorless, oily liquid (amber when impure)
Odor	Faintly fruity odor (odorless when pure)	Odorless when pure	Odorless when pure
Volatility	497 mg/m^3 at 77°F (25°C), 45.2 mg/m^3 at 32°F (0°C)	18,700 mg/m^3 at 77°F (25°C), 3,370 mg/m^3 at 32°F (0°C)	12.6 mg/m^3 at 68°F (20°C), 0.662 mg/m^3 at 32°F (0°C)
Vapor density (x air)	5.6	4.8	9.2
Solubility	Slightly soluble in water; Readily soluble in organic solvents	Completely miscible in water and common organic solvents	In water: 5% at 71°F, miscible at 49°F; Soluble in common organic solvents
Specific Gravity (H_20=1 g/mL at 77°F)	1.076	1.0919	1.0113
LCt_{50} (inhalation of vapor)*	70 mg-min/m^3	35 mg-min/m^3	15 mg-min/m^3
LD_{50} (direct contact with liquid)**	1.5 g/155-lb (70-kg) man	1.7 g/155-lb (70-kg) man	5 mg/155-lb (70-kg) man
Rate of action***	Very Rapid	Very rapid	Rapid

*LCt_{50}: (concentration x time of exposure) that is lethal to 50% of the exposed population
**LD_{50}: dose that is lethal to 50% of the exposed population

Relative Toxicity

The U.S. Army has estimated the human lethal doses of chemical warfare agents based on experiments using animals. These estimates apply to military personnel in combat deployment situations and should not be used for diverse civilian populations. The estimates are useful, however, as indicators of the toxicity of the agents in comparison to each other and to other hazardous compounds.

The estimated median lethal dose (LD_{50}) of liquid nerve agent VX is 5 milligrams on the skin of a 155-lb (70 kg) man. This is approximately equivalent to a small droplet being absorbed through the skin of a 155-pound person. By the inhalation route, VX vapor (considered the most potent nerve agent) (LCt_{50} of 15 mg-min/m^3) is estimated to be at least 100 times more toxic than cyanide gas. The estimated LD_{50} of GB agent is approximately 1.7g on the skin on a 155-lb man. The median lethal dose (LCt_{50}) from inhalation of GB vapor is estimated to be 35 mg-min/m^3.

Mechanisms of Action

In emergency first-aid training, emphasis is placed on learning the “why’s” in addition to the “how to’s.” EMTs and other pre-hospital emergency medical personnel are accustomed to learning not only specific procedures but also (to some degree) how the injury or trauma affects the normal physiological functions of the human body. In most instances, this knowledge is fundamental or prerequisite to the level of understanding required for making critical, life-dependent decisions. It is from this perspective that the following information is presented describing how the nerve agents act on the body.

How Nerve Agents Work

The nervous system controls body functions through the use of chemicals that act as “instructions” to nerves and to the muscles and glands. These “instructions” come in two forms: (1) stimulate (move or work), and (2) relax (stop or rest). When a nerve agent is present, it interferes with the normal chemical instructions that direct a stimulated muscle (or gland) to return to an un-stimulated state and relax or rest.

By interfering with the normal chemical checks and balances, the action of toxic nerve agents over-stimulates the nerve endings and central nervous system. Over-stimulation of the peripheral nervous system causes muscles and certain glands to over-react and various body organs to malfunction. Over-stimulation of the central nervous system causes a range of effects from giddiness to confusion, irritability, and, at higher doses, convulsions and depression of the central respiratory control center.

A More Technical Version of How Nerve Agents Work[6,7,8]

While the preceding explanation was greatly simplified, the following explanation is slightly more technical. It begins with an explanation of the reaction which normally occurs at a nerve ending and then describes how the nerve agent affects this process.

Nerves control muscles and organs; the nerve itself is made up of nerve cells that do not directly contact each other. Nerve impulses are relayed between nerve cells at a synapse (gap between the cells) by means of chemicals called neurotransmitters. When an impulse arrives at the synapse, a neurotransmitter, called acetylcholine, is released to carry the impulse across the synapse to a receptor site on the target cell. This is also how nerve impulses travel from nerves to a muscle or gland. When stimulated by acetylcholine, a skeletal muscle will contract, a smooth muscle will move rhythmically, and a gland will secrete.

Once the neurotransmitter has acted on the target cell receptor site, the enzyme acetylcholinesterase deactivates the acetylcholine. Stimulation of the target nerve, muscle, or gland cell stops with deactivation of the acetylcholine.

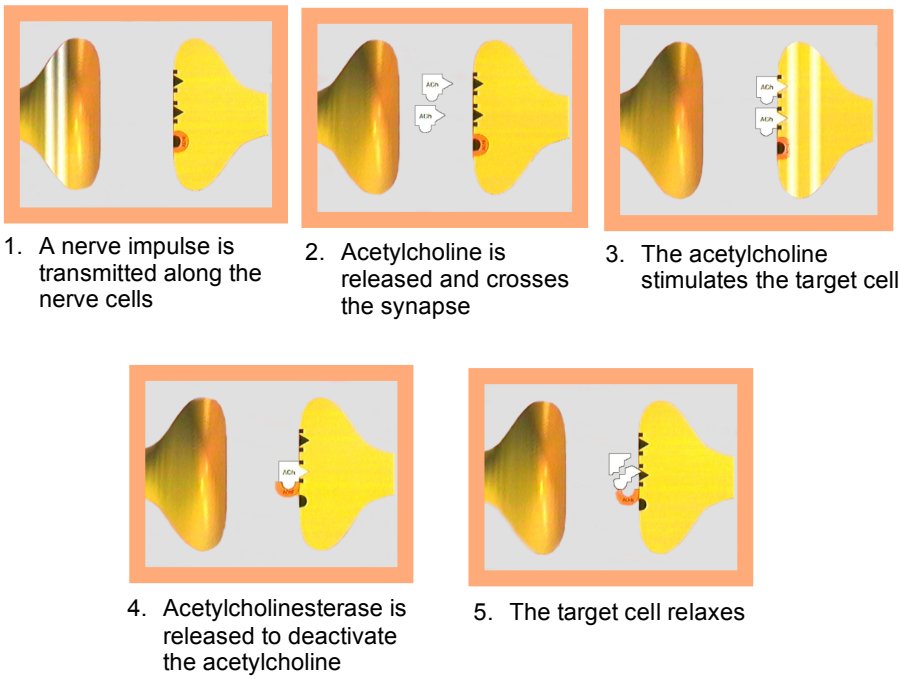


Figure 3.1. Normal Nervous System Function

However, nerve agents inactivate acetylcholinesterase, allowing excess acetylcholine neurotransmitter to accumulate and continue to stimulate receptor sites on the target nerve cell, muscle, or gland. If the affected nerve is stimulating a muscle, the muscle action becomes uncontrolled and a series of twitches, jerks, or spasms may be seen.

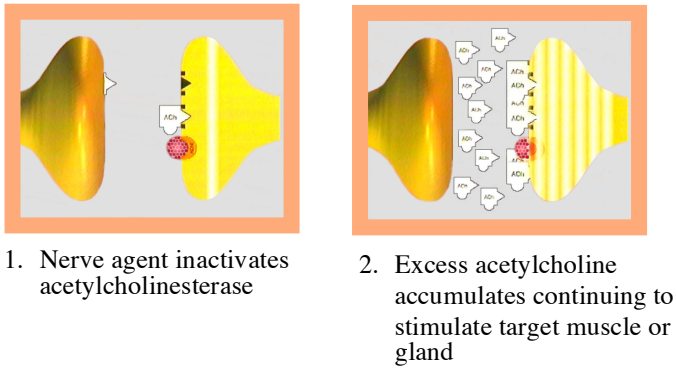


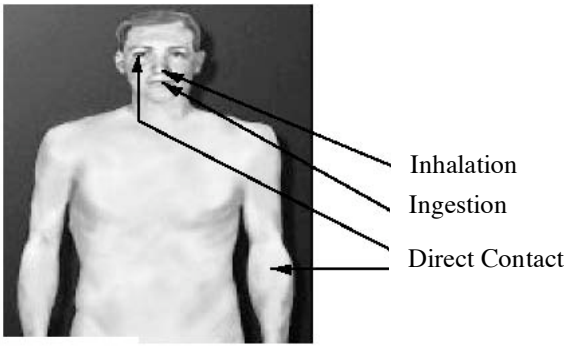
Figure 3.2. Nerve Agent Interference

Eventually, if the process is not interrupted, the muscle can go into a prolonged contraction and become fatigued, or even collapse. If the affected nerve is stimulating a gland, the result could be increased sweating, tearing, or mucus production in the respiratory passages. If the affected nerve cell is in the brain, the result could be giddiness, lack of ability to concentrate, mood changes, or loss of consciousness, convulsions, and inhibition of the respiratory control center.

Routes of Exposure

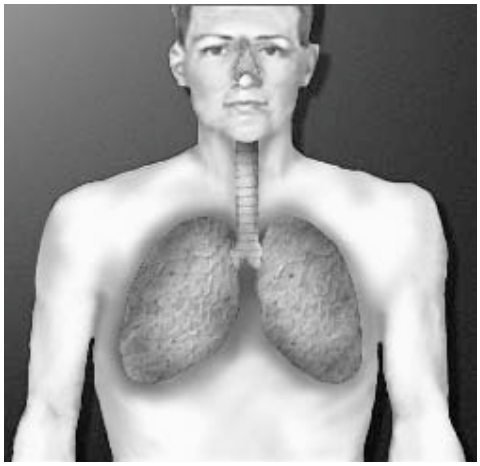
There are three main routes, or ways that a person can be exposed to a nerve agent. They are:

Inhalation—breathing air that has been contaminated with nerve agent vapors
Direct contact—absorption through the skin or eyes
Ingestion—swallowing contaminated food or drink



Inhalation

Although the nerve agents are stored as liquids, they are easily changed into vapors, especially agent GB. These vapors mix freely with the air. If a person breathes in the contaminated air, the toxic chemical enters the body through the respiratory system.



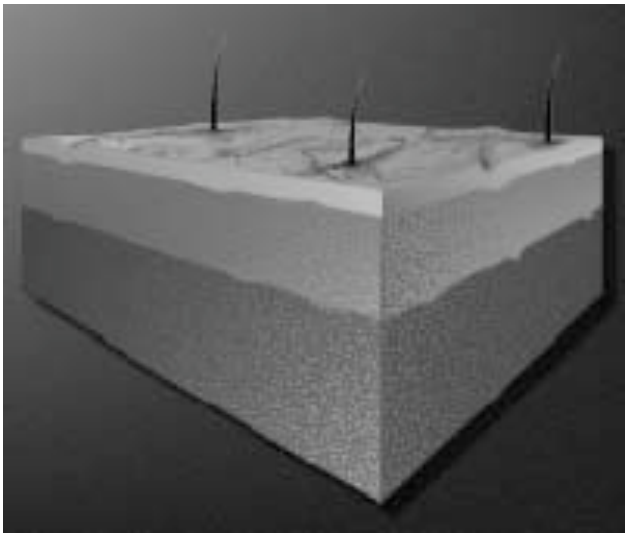
After the nerve agent has entered the lungs it is generally absorbed rapidly and effectively into the blood stream. This is because of the large surface area of the lung tissue and the high number of blood vessels in the lungs. Respiratory failure is the chief cause of death after a severe exposure to nerve agent.

Direct Contact

Direct contact occurs when the skin or eyes come into contact with agent vapor or liquid. It is through this route that contamination can spread from one patient to another or to health care providers. Nerve agents have no effect on the external layer of skin itself; rather, the nerve agent is absorbed through the skin. Once penetration has occurred, the nerve agent is circulated to the nervous system. Concentrated vapor or mist can also penetrate skin tissue in the same way the liquid does.

All of the nerve agents can be absorbed through the skin; however, agent VX tends to be absorbed much more completely because it does not evaporate as quickly. The persistence of VX causes it to remain on the skin, thus allowing more absorption time. Because it penetrates slowly compared to GB, immediate decontamination is important. VX is a “highly persistent” oily liquid that does not volatilize or degrade rapidly. VX persistence is weather-dependent. At 99°F, 90% of a VX droplet will evaporate in approximately 24 hours; at 50°F, the passage of 45 days would be required before 90% of a VX droplet would evaporate.

The more watery G-agents, on the other hand, have a tendency to evaporate quickly. Agent GB, in particular, tends to evaporate off the skin quickly rather than penetrate it. Cases of GB poisoning have occurred from skin exposure to liquid agent. Skin exposure to GB vapor can also result in poisoning, but only at concentrations higher than those that produce severe inhalation effects.



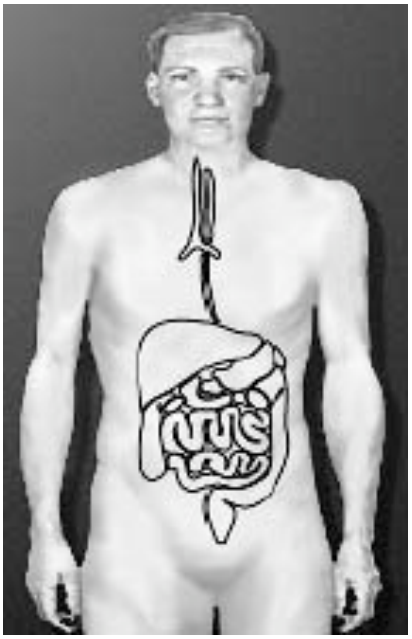
Once absorbed through the skin, the nerve agent begins to affect the normal chemical neurotransmission process that occurs between the nerves and muscles or glands. On normal, intact skin, nerve agent must first pass through a layer of outer dead cells and the epidermis before reaching the capillaries (bloodstream) and nerve cells found in deeper tissue layers. If a G-agent is involved, evaporation may be complete before a significant dose can be absorbed. However, scrapes cuts, or other skin damage present immediate entry points for the nerve agent and facilitate direct access to the bloodstream and nervous system.

Increased skin permeability is a concern for skin conditions that are not commonly thought of as “wounds”. For example, freshly shaven skin, sunburn, insect bites, and rashes are all examples of potential entry points for nerve agents.

Agent also enters the body very effectively through the eyes. It is important to protect the eyes from exposure to vapors or aerosols. The eye is the most sensitive organ for nerve agent effects. The threshold dose for pinpoint pupils (miosis) is the basis for both VX and GB exposure standards.

Ingestion

If an individual ingests contaminated food or drink, the nerve agent can enter the body through the digestive system. Incidental hand-to-mouth contact, smoking, and swallowing contaminated substances are all examples of potential sources of exposures by this route.



Once the nerve agent has entered the digestive system, it can easily reach the bloodstream. Although the likelihood of the agent contaminating food or drink is extremely slim, it is possible for someone to become exposed through ingestion. Common sense precautions dictate that:

- absolutely no food or beverages should be kept in or around medical treatment areas, and
- in treating or caring for patients, extreme caution should be exercised when giving anything by mouth.

Critical Routes of Exposure

A chemical agent release is most likely to affect surrounding communities if agent vapors or aerosols are transported in an airborne plume. Therefore, medical responders should be most concerned with the routes of exposure that can occur through air, especially (1) inhalation of agent vapor or aerosol and (2) eye and skin contact with vapor or aerosol. It is much less likely that emergency medical personnel will encounter casualties who have ingested chemical agent or come into direct contact with agent in liquid form (unless responding to the immediate scene of the incident, as in a terrorist event).

3.2 Blister Agents (Vesicants): A Description

Vesicants are poisons that destroy individual cells in target tissues. The most noticeable effect that these agents have is the "vesicles," or blisters, they cause. For this reason, these types of agents are also referred to as "blister agents". This student guide uses the term "blister" to describe this category of chemical agent.

Specific Names

While the term "blister agent" refers to a particular effect of chemical warfare agents, specific common names and abbreviations are associated with individual chemical compounds that produce this effect.

Common Name	Abbreviation
Sulfur Mustard	H, HD, HT
Lewisite	L

Sulfur mustard agent exists in three forms in the chemical stockpile. The original form, called H, contains up to 30% impurities. A refined form, HD, is much purer. The third form, HT, consists of a mixture of sulfur mustard and another agent that lowers the freezing point of the compound. Although Lewisite is in the blister agent family, all of this agent has been eliminated from the stockpile. Consequently, this section will discuss only sulfur mustard agent.

Students interested in reviewing more technical information about sulfur mustard agents can review the MSDSs in Appendix D. The U.S. Army's Edgewood Chemical Biological Center developed these documents to provide technical details on the agents' chemical formulas, structures, names, physical data, and hazard characteristics. Additional information may be found in volume 3 of the Textbook of Military Medicine[8]. However, this course does not require that students know about nerve agents to the level of detail described in these outside sources.

Physical Properties

Sulfur mustard agents are stored in the chemical stockpile as either solids or liquids. Agents H and HD solidify at fairly high temperatures (14°C or 57°F). Agent HT remains liquid until temperatures drop below 1.3°C (34°F). If they are heated, these liquids are

volatile—they generate vapors. Sulfur mustard agent is an oily liquid that burns well once ignited.

The physical characteristics of blister agents are described in some detail in the student guide for the Chemical Awareness course. The major physical properties of these agents are summarized in Table 3.2.

Table 3.2 Blister Agent Comparative Data

	H[9]	**HD**[9]	**HT**[10]
Appearance	Pale yellow to dark brown oily liquid	Pale yellow to dark brown liquid	Pale yellow to brown liquid
Odor	Garlic or horseradish	Garlic or horseradish	Garlic
Volatility	906 mg/m^3 at 25°C (77°F)	610 mg/m^3 at 20°C (68°F)	783 mg/m^3 at 25°C (77°F)
Vapor density (x air)	5.5	5.4	6.5
Freezing temperature	14°C (57°F)	14°C (57°F)	1.3^{o}C (34°F)
Solubility		Practically insoluble in water; freely soluble in oils and most organic solvents	Slightly soluble in water; soluble in most organic solvents
Specific gravity (H_20=1)		1.27 at 77°F	1.265 at 68°F
LCt_{50} (inhalation of vapor)*	1,000 mg-min/m^3	1,000 mg-min/m^3	1,000 mg-min/m^3
LD_{50} (direct contact with liquid)**	1,400 mg/155-lb (70-kg) man	1,400 mg/155-lb (70-kg) man	1,400 mg/155-lb (70-kg) man
Rate of action***	Moderately rapid	Moderately rapid	Moderately rapid

*LCt_{50}: (concentration x time of exposure) that is lethal to 50% of the exposed population
**LD_{50}: dose that is lethal to 50% of the exposed population
***Cellular damage from sulfur mustard exposure occurs within minutes even though pain, blisters, etc. may not manifest for two hours or more (up to 24 hours).

Relative Toxicity

The U.S. Army has estimated the human lethal doses of the chemical warfare agents based on experiments using animals. These estimates apply to military personnel in combat deployment situations and should not be used for diverse civilian populations. The estimates are useful, however, as indicators of the toxicity of the agents in comparison to each other and to other hazardous compounds.

The estimated median lethal skin dose (LD_{50}) of sulfur mustard agent is 1.4 grams on the skin of a 155-lb (70-kg) man. Between 4 and 32 micrograms are estimated to be enough to cause skin reddening and blistering. By the inhalation route, sulfur mustard vapor is estimated to be three times more toxic than cyanide gas (LCt_{50} at 1,000 mg-min/m^3).

How Blister Agents Work[6, 7, 11]

Blister agents can affect any skin tissue, but are especially harsh to warm, moist surfaces of the body and to delicate tissues such as the soft membranes surrounding the eyes, the eyeball, lung tissue, and tissues of the mouth and throat. Both the liquid itself and the vapors generated from sulfur mustard create an extreme hazard. The greater the absorbed dose of either, the greater the damage.

Sulfur mustard agent is a cellular poison; cell membranes are damaged within minutes after exposure. Severity of skin and tissue damage is highly dose-dependent. There is little or no pain at the time of exposure to the agent. The development of clinical signs such as burning, stinging, redness or blisters is usually delayed between 2 and 24 hours, occasionally even up to 36 hours.

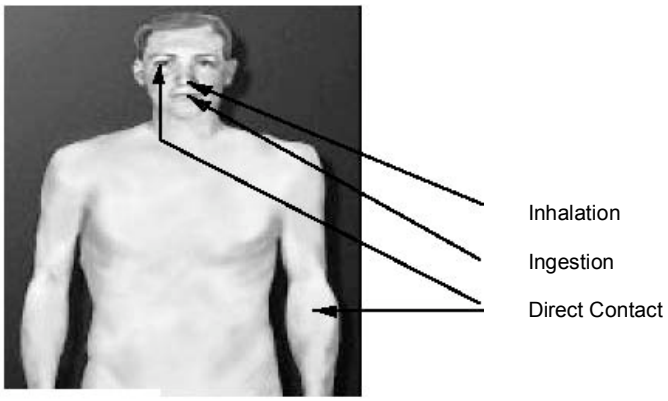
Routes of Exposure

A critical step in the treatment of patients of sulfur mustard agent exposure is to decontaminate the victim. Since sulfur mustard is also carcinogenic (capable of causing cancer), it is doubly critical that the patient be decontaminated quickly and thoroughly. In order to do this most effectively, responders must understand how the blister agent is capable of entering the body. This knowledge will enable emergency medical personnel to treat the patient effectively while protecting themselves and others from exposure.

The warfare function of the blister agents is to decrease the opponent's ability to fight by producing chemical burns on tissues that come into contact with either vapors or liquid droplets/aerosols. Exposed skin surfaces, eyes, the respiratory tract, and upper gastrointestinal tract are all at risk. The moist surfaces of perspiring skin, linings of the eyes, and airway mucous membranes preferentially absorb sulfur mustard agent and distribute it over a larger area. Under hot, humid conditions, when large areas of skin are likely to be wet with perspiration, much lower sulfur mustard concentrations generate debilitating effects.

There are three main routes, or ways, a person can be exposed to a blister agent:

Inhalation—breathing air that has been contaminated with nerve agent vapors
Direct contact—absorption through the skin or eyes
Ingestion—swallowing contaminated food or drink



Inhalation

Although the blister agents may be stored as liquids, they are easily changed into vapors, particularly at warm temperatures. These vapors mix freely with the air. If a person inhales the contaminated air, the toxic chemical enters the body through the respiratory system. Once inhaled, the blister agents also have direct access to the linings of the nose, throat, and bronchial tubes. These warm, moist membranes are particularly vulnerable to the effects of sulfur mustard agent. With prolonged exposure, the agent destroys the mucous membrane lining—just as skin blisters damage the outer layer of skin—causing internal inflammation and hemorrhage and allowing the airways and lungs to become infected. Blister agents do most damage to the upper airways, but with a severe exposure, the air sacs in the lungs can be injured and filled with fluids.

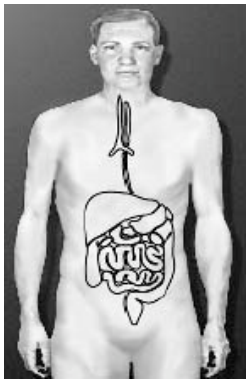
Direct Contact

Direct contact occurs when any skin surface or the eye comes in contact with liquid agent or touches a surface on which the agent has been deposited. It is through this route that contamination can spread from one patient to another or to health care providers.

Unlike nerve agents, blister agents are highly damaging to the skin. Warm, moist membranes are very susceptible to the effects of blister agents. These membranes include the lining around the eyelids and the insides of the nose, mouth, and throat. Since warmth and moisture increase the blister agent's effect, other body areas are particularly susceptible to severe blistering. These areas include between the toes, behind the knees, the buttocks, the groin, elbows, the arm pits, folds of the neck, behind the ears, and under the breasts (female).

Ingestion

If blister agent has been deposited on or in food items, drink, or anything that a person may place in the mouth (e.g., cigarettes), the agent can injure the warm, moist tissues of the mouth, throat, and esophagus. Incidental hand-to-mouth contact, smoking, and swallowing airborne contaminants are examples of this exposure route.



Although the likelihood of the agent contaminating food or drink is small, it is possible for someone to become exposed through ingestion. Common sense dictates that:

- absolutely no food or beverages should be kept in or around medical treatment areas, and
- in treating or caring for patients, extreme caution should be exercised when giving anything by mouth.

Critical Routes of Exposure

A chemical agent release is most likely to affect surrounding communities if agent vapors or aerosols are transported in an airborne plume. Therefore, emergency medical personnel should be most concerned with the routes of exposure that can occur through air, especially (1) inhalation of agent vapor or aerosol, and (2) eye and skin contact with vapor or aerosol. It is much less likely that responders will encounter casualties who have ingested chemical agent or come into direct contact with agent in liquid form (unless responding to the immediate scene of the incident, as in a terrorist event).

Self-Check

Students can assess their understanding of the material presented in this module by completing the following Self-Check.

Multiple Choice

3.1 Toxic chemicals that are categorized as “nerve agents” are so called because

a. they disrupt the body’s nervous system
b. the agent is essential to the nervous system
c. they send “electrical” shooting pains throughout the nervous system
d. the blisters they cause are extremely painful to the nerve endings in the skin tissue

3.2 The specific nerve agents currently in the U.S. Army’s chemical stockpile inventory are:

a. VX and sulfur mustard
b. VX, sulfur mustard, and Lewisite
c. VX, GA, and GB
d. Lewisite and GB

3.3 Nerve agents are stored in the chemical stockpile as:

a. gases
b. solids
c. vapors
d. liquids

3.4 Nerve agents affect the nervous system by

a. killing off the nerve cells
b. making the central nervous system shut down
c. blocking the messages sent to the nerve, muscle and gland cells
d. inactivating acetylcholinesterase, thus causing the nerve endings to over-stimulate the nerve/muscle/gland

3.5 A person can be exposed to a nerve agent through

a. touching a surface that has the nerve agent on it
b. inhaling air that has been contaminated by a nerve agent
c. eating or drinking anything that has been contaminated by a nerve agent
d. any of the above

3.6 The nerve agent VX is described as “highly persistent.” This means that

a. the agent is only very slightly degraded by the environment over a long period of time
b. even if an exposed person is treated immediately the effects never go away
c. it is so watery that it washes away easily with the correct treatment
d. a spill is more potent after several days than the first day

3.7 Which of the following skin conditions is most likely to increase the initial effects of nerve agent exposure?

a. hairy chest
b. eyes behind goggles
c. hand wearing a glove
d. forearm with cuts and abrasions

3.8 Toxic chemicals that are categorized as "blister agents" are so called because

a. an inevitable side-effect caused by the drug antidote for this agent has been known to cause blisters in most people
b. the most noticeable effects are blisters and the destruction of cells in target tissues
c. they cause the body to breakout in painless sores that resemble tiny chicken pox-like blisters
d. they were named after the chemist who discovered their potential wartime use

3.9 The specific blister agents currently in the U.S. Army's chemical stockpile inventory are:

a. VX and sulfur mustard
b. VX, GA, and GB
c. sulfur mustard
d. VX, sulfur mustard, and Lewisite

3.10 Blister agents are stored in the stockpile as:

a. either liquids or solids
b. aerosols
c. vapors
d. gases

3.11 It is often difficult to detect an exposure to a sulfur mustard agent immediately because

a. the signs and symptoms are delayed
b. the blister does not form for several weeks after exposure
c. pain is felt immediately but it is difficult to know what the cause of it is
d. once the blister agent has contacted the skin, the patient no longer has any sensation in that area

3.12 A person can be exposed to a blister agent through all of the following routes EXCEPT:

a. touching a surface that has the blister agent on it
b. inhalation of air that has been contaminated by a blister agent
c. eating or drinking anything that has been contaminated by a blister agent
d. touching the fluid that seeps from a blister of a person who has been exposed to a blister agent

3.13 Although public exposure during the chemical stockpile destruction process is unlikely, the route of exposure that EMTs should be most familiar with is

a. ingestion through contaminated food
b. skin exposure through direct contact
c. spill caused by accident while in transportation
d. air contamination, where the plume moves off the storage installation

Check responses using Appendix A, Self-Check Answers.

Summary

The most important points covered in this module are:

- Nerve agents:
  - are stored as liquids (VX is thick and oily), but can generate extremely toxic vapors as they evaporate
  - can be absorbed through direct contact with the skin or eyes, inhalation of contaminated air, and ingestion (swallowing) of contaminated food or drink

- Blister agents:
  - are stored as solids or liquids (thick and oily), but can generate toxic vapors as they evaporate
  - can be absorbed through direct contact with the skin or eyes, inhalation of contaminated air, and ingestion (swallowing) of contaminated food or drink

- Most likely routes of exposure that pre-hospital emergency medical personnel should be prepared for are
  - inhalation of contaminated air
  - eye and skin contact with agent vapor or aerosol

MODULE 4 SIGNS AND SYMPTOMS OF EXPOSURE

Objectives

After completing Module 4, students should be able to:
1. Identify the signs and symptoms of nerve agent exposure.
2. Identify the signs and symptoms of blister agent exposure.

Introduction

The most important part of medical treatment given to patients of nerve and blister agents is first aid and special treatment. EMTs must be prepared to act fast. The decisions made during the first few seconds—especially in cases of severe nerve agent exposure—are extremely critical and can make the difference between life and death.

In the case of patients suffering from chemical agent exposure, where immediate treatment is critical, emergency medical personnel must be able to size up the situation based on:

- information that has been provided about the chemical agent release, and
- recognition of chemical agent exposure based on the signs and symptoms of the victims.

While standard procedure calls for full disclosure and widespread notification in the event of a chemical agent release, there could be times when a release was not detected or when the communication of important information to first responders was delayed. For this reason, medical responders must be prepared to act based on the signs and symptoms produced by exposure to nerve and blister agents.

In addition, as with any first aid rendered for poisoning, the specific treatment and/or antidote required varies depending on the chemical inflicting the illness or injury. Emergency medical personnel must be able to identify the agent involved before beginning treatment. This chapter describes the signs and symptoms of nerve agent poisoning and the signs and symptoms of blister agent poisoning.

The most important points to learn from this key module are:

- The most prominent signs and symptoms of nerve agent vapor/aerosol exposure
  - miosis (pinpointing of the pupils)
  - increased secretions from mouth, nose and bronchi
  - respiratory difficulty

- The most prominent signs and symptoms of blister agent vapor/aerosol exposure
  - irritation, reddening and swelling of eye tissues (conjunctivitis)
  - reddening of the skin (erythema)
  - blisters
  - respiratory irritation and distress

- Not all signs and symptoms may appear; dose, duration, and route of entry (among other factors) can make a difference.

4.1 Signs and Symptoms of Nerve Agent Exposure

For anything less than severe exposures, the signs and symptoms of nerve agent intoxication and the speed with which they appear differ markedly depending on the route of exposure and the dose of agent received. It is especially important to note that the effects of mild to moderate exposure to nerve agent *vapor* are quite distinct from the effects of similar levels of exposure to *liquid* agent on the skin. For severe, life-threatening exposures, however, the effects are similar regardless of the route of exposure.

Material Safety Data Sheets (MSDSs) for agents GA, GB, and VX are included as Appendices D.1, D.2, and D.3. These sheets, released by the U.S. Army's Edgewood Chemical Biological Center. provide additional information about nerve agent signs and symptoms. Students may also wish to refer to the NIOSH (National Institute of Occupational Safety and Health) Emergency Response Cards produced by the Centers for Disease Control and Prevention.[12, 13, 14]

Note that not all of the listed signs and symptoms will necessarily appear in an individual exposed to nerve agent. The amount of agent, duration of exposure, exposure route, and differences in individual susceptibility influence the appearance of effects.

Signs and Symptoms of Exposure to Nerve Agent Vapor

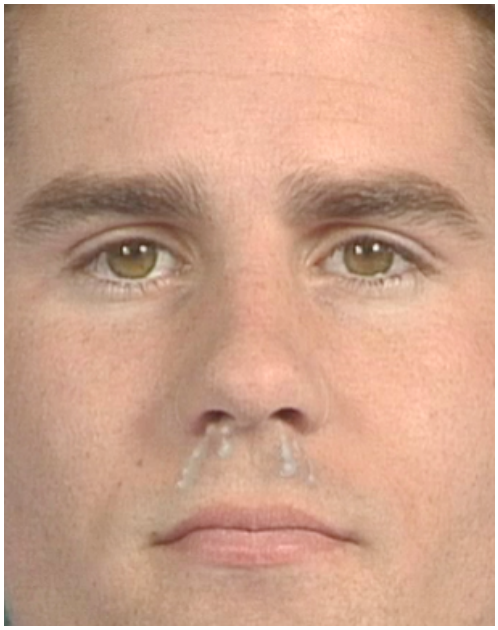
The critical route of exposure to nerve agent in CSEPP communities is through inhalation and contact with agent vapors carried in an airborne plume. Signs and symptoms of exposure to nerve agent vapor include both those that occur due to the vapor's direct effect on organs it contacts and those that occur as the agent is absorbed into the body's systems. Table 4.1 lists the common effects of mild, moderate, and severe exposures to nerve agent vapor. It is important to note that, regardless of the degree of exposure to agent vapor, signs and symptoms will appear within seconds to minutes after the beginning of exposure. Signs and symptoms normally peak within 15 to 25 minutes after exposure stops.

Table 4.1 Signs and Symptoms of Exposure to Nerve Agent Vapor[6, 7, 8]

Severity of Exposure	Signs and Symptoms	Time to Onset of Effects
Mild	Pinpoint pupils (with or without red eye or pain) Dim or blurred vision Runny nose Slight chest tightness Slight difficulty breathing Secretions (tears, nasal fluids, saliva, sweat, phlegm) Nausea and vomiting	Seconds to minutes
Moderate	Pinpoint pupils (with or without red eye or pain) Dim or blurred vision Excessively runny nose Pronounced chest tightness Plentiful secretions (tears, nasal fluids, saliva, phlegm) Moderate to severe difficulty breathing (gasping for air) Nausea and vomiting Diarrhea General muscle weakness	Seconds to minutes
Severe	Same as for moderate exposure, plus Loss of consciousness Convulsions Generalized rippling of muscles under the skin (fasciculation) Flaccid paralysis Cessation of breathing Involuntary urination or defecation	Seconds to minutes

Mild Exposure

A mild exposure to nerve agent vapor usually affects the organs directly contacted by the vapor, primarily the eyes, nose, and airways. The eyes and nose are most sensitive and typically show the first effects. Miosis (pinpoint pupils) and a runny nose are the most common signs of a mild exposure. Miosis may be accompanied by red eye or by pain in or around the eyeball or in the front part of the head. Vision may be dim or blurred. The patient may also complain of tightness in the chest or experience a slight difficulty in breathing. There may also be an increase in the discharge of fluids (tears, nasal fluids, saliva, phlegm) from affected glands. Some patients may experience nausea and vomiting.



After being removed from the nerve agent environment and decontaminated, a patient who experienced very mild vapor exposure may quickly begin to recover and may feel normal within 15 to 30 minutes. However, the signs and symptoms of patients who experienced higher exposures will be relieved only with medical treatment.

Moderate Exposure

A moderate exposure to nerve agent vapor produces signs and symptoms that are similar in nature to those of a mild exposure, but are more pronounced and more detrimental to the patient's health. In the eyes, miosis (pinpoint pupils) may be more severe, and dim or blurred vision will be more likely. The effects of the runny nose may be much greater than normally experienced during a bad cold, and secretions from glands may be similarly increased. Abundant mucous secretions from the respiratory passages may make breathing more difficult, and the patient may be gasping for air. Gastrointestinal effects will be more common, with patients experiencing nausea, vomiting, and diarrhea. Some or all of these effects may appear within seconds or minutes of the beginning of exposure.

Severe Exposure

Severe exposure to nerve agent vapor will very quickly affect the body's nerve and skeletal muscle systems, resulting in "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".[7] Victims of severe exposure may display generalized fasciculations (rippling motion of muscles under the skin), convulsive jerking or flailing of limbs, or a rigidly arched torso. Soon, the affected muscles become fatigued and flaccid paralysis sets in. Loss of muscle control may lead to involuntary urination or defecation.

Because these effects progress so rapidly, victims of severe vapor exposure are likely to be unconscious, convulsing, and/or not breathing when first seen by first responders.

Signs and Symptoms of Skin Exposure to Liquid Nerve Agent

While even more unlikely than exposure to nerve agent vapor, emergency medical personnel in CSEPP communities could be called on to treat patients suffering the effects of liquid nerve agent on their skin. Non-severe exposures received by this route produce signs and symptoms that differ from those of vapor exposure both in the nature of the effects and in the time at which they appear.

Table 4.2 lists the effects of mild, moderate, and severe exposure to liquid nerve agent on the skin. The severity of signs and symptoms depends primarily on the amount of nerve agent absorbed through the skin. Higher temperatures and humidity also cause effects to be more severe.

Effects of mild to moderate exposure to nerve agent liquid on the skin may appear within 10 minutes or be delayed for up to 18 hours after exposure ends. The effects of severe exposure usually appear within thirty minutes. In general, the longer the delay in the onset of effects, the less severe the effects will be. Exposure to nerve agent liquid may produce delayed effects even in patients who have been thoroughly decontaminated.

Mild Exposure

Mild skin exposure to nerve agent liquid typically causes local effects at the site of exposure. Sweating at the exposure site is common, and fasciculations may also occur at the site. In some cases, nausea and vomiting may occur even in the absence of localized sweating and fasciculations. Miosis (pinpointing of pupils) does not occur unless the exposure site is near the eye.

Moderate Exposure

Following a moderate skin exposure to nerve agent liquid, the signs and symptoms of mild exposure are likely to be accompanied by nausea and vomiting and, less commonly, diarrhea. The patient may also exhibit generalized sweating and report feeling tired and generally unwell.

Remember, these signs and symptoms may not show up until hours after exposure. In the meantime, the patient will appear to be unaffected by the exposure.

Severe Exposure

Signs and symptoms will appear suddenly within minutes of a severe skin exposure to liquid nerve agent. The effects are similar to those associated with severe vapor exposure. Once the effects begin, they progress rapidly. Within a very few minutes, the victim will collapse, lose consciousness, experience convulsions and generalized fasciculations, become flaccidly paralyzed, and stop breathing. Since these effects occur so quickly, EMTs arriving on the scene of severe skin exposures will likely encounter victims who are unconscious and possibly not breathing.

Table 4.2. Signs and Symptoms of Skin Exposure to Nerve Agent Liquid[6, 7, 11]

Severity of Exposure	Signs and Symptoms	Time to Onset of Effects
Mild	Sweating at site of exposure Rippling of muscles under skin at site of exposure (fasciculation)	10 minutes to 18 hours (effects may develop rapidly following an asymptomatic period)
Moderate	Same as for mild exposure, plus: Nausea Vomiting Diarrhea Generalized sweating Generalized weakness, tiredness, or ill feeling	10 minutes to 18 hours (effects may develop rapidly following an asymptomatic period)
Severe	Same as for moderate exposure, plus: Collapse Loss of consciousness Convulsions Generalized rippling of muscles under skin (fasciculation) Generalized, copious secretions (tears, nasal fluids, saliva, phlegm) Severe respiratory difficulty Flaccid paralysis Cessation of breathing Involuntary urination or defecation	2-30 minutes (effects likely to develop very rapidly following an asymptomatic period)

Factors that Affect Nerve Agent Exposure Signs and Symptoms

Route of Exposure

If the exposure route is inhalation of air with a high concentration of agent, effects can occur after a single breath. This immediate response occurs within seconds. After the patient has been removed from the exposure, look for the effects to peak within 15-20 minutes. Generally, an EMT can be reasonably certain that if the exposure was through air (only) and the time lapse has been 15 to 20 minutes or more, the effects have maximized and will not worsen after this time.

If the exposure route is dermal (droplet or liquid or vapor that touches the skin), absorption may continue for hours. This is likely to continue even after the victim has been decontaminated since the absorption occurs within the skin layers and from fatty deposits. In contrast to the air route (where the worst can be expected within 15-20 minutes), the effects of direct skin contact may not occur for hours after exposure (range from 1-18 hours for mild and moderate exposures). Signs and symptoms that occur many hours after exposure are usually non-lethal.

Toxicity

The toxic effects a person experiences after inhaling air containing nerve agent depends on the type of agent involved, the concentration of agent in the air, and the length of time that the individual inhales the air. A National Advisory Committee, working under the jurisdiction of the National Research Council's Committee on Toxicology, has developed estimates of the combinations of concentrations and exposure times that could produce specific levels of health effects for a single exposure. These levels, called Acute Exposure Guideline Levels (AEGLs) are summarized in Table 4.3. Notice that, for a given exposure duration, minor effects (AEGL-1) begin to appear at much lower concentrations than those required to produce severe effects (AEGL-3). More detailed information on AEGLS is available on information sheets[15] released by the U.S. Army Center for Health Promotion and Preventive Medicine.

Table 4.3 Example Nerve Agent Acute Exposure Guideline Levels[16]

Acute Exposure Guideline Level	Duration of Exposure	Agent GA concentration (mg/m^3)	Agent GB concentration (mg/m^3)	Agent VX concentration (mg/m^3)
AEGL-3: Exposures above this level may cause *severe adverse effects* including vomiting, seizures and increasing possibility of death.	8 hr	0.10	0.051	0.0038
	1 hr	0.26	0.13	0.010
	10 min	0.76	0.38	0.029
AEGL-2: Exposures above this level may cause *significant adverse effects* (including impairment of vision in dim light, runny nose, sweating, and breathing difficulties) but no permanent effects.	8 hr	0.013	0.013	0.0010
	1 hr	0.035	0.035	0.0029
	10 min	0.087	0.087	0.0072
AEGL-1: Exposures above this level may begin to cause *minor* miosis and runny nose, but no significant or permanent effects.	8 hr	0.0010	0.0010	0.000071
	1 hr	0.0028	0.0028	0.00017
	10 min	0.0069	0.0069	0.00057

Lethality

The chief cause of death from nerve agent exposure is respiratory failure due to a combination of bronchoconstriction and secretions in the airways, weakness of the muscles that force the lungs to expand and contract, and inhibition of the respiratory center in the brain.

Differential Diagnosis - Nerve Agent

Many of the symptoms described above may be caused by health problems other than exposure to a nerve agent. Emergency medical personnel must determine that a person is suffering from actual nerve agent exposure before administering nerve agent antidotes.

In some cases, emergency responders will know the identity of the agent before arriving at the scene. If a chemical agent release is detected from a stockpile location, authorities pass information about the release—including the agent involved—to response personnel. In other cases, such as a terrorist event, first responders may have to evaluate the signs and symptoms of victims to determine that a nerve agent release has occurred. They make this determination using a technique called "differential diagnosis."

This is the process of distinguishing one disease from another when two or more diseases produce the same or similar effects.

Emergency medical personnel should suspect nerve agent poisoning whenever they encounter a patient exhibiting some of the signs and symptoms of nerve agent exposure and no definite alternative explanation of these effects is apparent. If many casualties displaying symptoms of nerve agent exposure are encountered within a brief time, responders should suspect terrorism.[6]

Individual signs and symptoms of nerve agent exposure may also be attributed to the following conditions[6, 7, 18, 19]:

- epilepsy
- gastroenteritis
- exposure to agricultural insecticides (organophosphates and carbamates)
- exposure to other toxins (e.g., cyanide, coniine)
- treatment with certain drugs (e.g., medicinal carbamates, cholinomimetic compounds, and neuromuscular blocking drugs)
- upper respiratory illness
- emphysema
- allergic reaction
- stroke
- head trauma
- drug overdose (e.g., muscarine-containing mushrooms, fentanyl)
- heat illness

A mild exposure to nerve agent vapor may produce symptoms (runny nose and chest tightness) similar to the effects of an upper respiratory illness or allergy. Examination of the patient's eyes in dim light can detect pinpoint pupils, a clear indication of nerve agent vapor exposure.[7]

There are no clear signs by which gastrointestinal symptoms (nausea, vomiting, diarrhea) of nerve agent exposure can be distinguished from other possible causes. If possible, the patient should be questioned about the possibility of exposure and about other medical conditions that could explain these symptoms.[7]

The diagnosis of severely intoxicated victims is less complicated. The following combination of severe nerve agent effects is not easily confused with any other cause: pinpoint pupils, plentiful secretions (tears, nasal fluids, sweat, phlegm), generalized muscular twitching, severe respiratory distress, and convulsions.[7]

4.2 Signs and Symptoms of Sulfur Mustard Agent Exposure

The signs and symptoms of sulfur mustard agent exposure are different from those produced by exposure to a nerve agent. This section presents information about the signs and symptoms of exposure to sulfur mustard agent. The MSDSs for sulfur mustard agents located in Appendix D contain additional information on this agent's signs and symptoms.

A critical characteristic of sulfur mustard agent exposure is that signs and symptoms seldom appear until hours after exposure has occurred. Consequently, emergency

medical personnel may find themselves providing treatment to patients who are not yet showing the effects of exposure. In spite of the delay in the appearance of signs and symptoms, the agent becomes irreversibly fixed to human tissue within a very few minutes. People who are suspected of exposure to sulfur mustard agent should immediately be thoroughly decontaminated and observed for the possible onset of signs and symptoms.

How soon signs and symptoms of sulfur mustard agent exposure appear and the severity of the effects depend on the degree of exposure as well as weather conditions. The larger the dosage of agent the victim receives, the more severe the effects will be and the sooner they will appear. Hot, humid weather also increases the severity of effects and the speed of their appearance.

Signs and Symptoms of Exposure to Sulfur Mustard Agent Vapor

In the unlikely event that a release of sulfur mustard agent reaches civilian populations, it would probably occur in the form of agent vapors or aerosols transported in an airborne plume. People in the path of the plume could be exposed in two ways: first, by agent vapors contacting the skin and eyes and being absorbed into the body; second, by the agent being inhaled into the respiratory tract, contacting the mucous membrane linings and being absorbed into the body. Exposure to sulfur mustard agent vapor could produce signs and symptoms in several bodily organs. These effects are described in the following sections and are summarized in Table 4.4.

It is important to note that an exposed person may not exhibit all of the signs and symptoms of sulfur mustard agent poisoning. Some of the signs and symptoms described in this section depend on the dose of agent received, the sensitivity of the individual exposed, and the route of exposure.

Table 4.4 Signs and Symptoms of Exposure to Sulfur Mustard Agent Vapor[6, 7, 11]

Organ Affected	Severity of Exposure	Signs and Symptoms	Time to Onset of Effects
Eye	Mild	Tearing, itching, burning, gritty feeling, sensitivity to light	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
Skin	Mild	Erythema (redness) with itching, burning, and stinging	2-24 hours
	Severe	Above, plus blisters	2-24 hours
Airways	Mild	Runny nose; pain in the throat, nostrils, or sinus; sneezing; nosebleed; hoarseness; hacking cough	6-24 hours
	Moderate	Above, plus loss of voice, productive cough	
	Severe	Above, plus moderate-to-severe breathing difficulty	2-6 hours

Effects of Sulfur Mustard Vapor on the Eyes

The eye is the organ that is most sensitive to sulfur mustard agent and is generally the first organ to show signs and symptoms. The severity of the effects and the speed with which they appear depends on the degree of exposure. Following a severe vapor exposure, signs and symptoms may appear in as little as 1 hour. After a mild exposure, 12 or more hours may pass before effects become apparent.

Patients receiving a low dosage of sulfur mustard agent may experience only slight irritation and reddening of the eye and eyelid. Effects may include tearing, itching, blinking, and a sensation of “grit” in the eye. Because the eye is so extremely susceptible to sulfur mustard agent, this particular set of signs is the most sensitive indicator of exposure.

Higher dosages of agent result in more severe inflammation of the eyelids, severe pain, and possible damage to the cornea. The eyelids may swell to the extent that the eyes are completely closed. Patients may be alarmed and think they are blind. They should be reassured that it is only the swelling that is affecting their vision.

Regardless of the level of exposure, the patient is likely to experience photophobia - eye pain or discomfort caused by light.

Effects of Sulfur Mustard Vapor on the Skin

The skin is vulnerable to damage by sulfur mustard agent vapor, but it is considerably less sensitive than the eye. This means that a more intense exposure is necessary to produce signs and symptoms on the skin than on the eye. Skin injuries caused by exposure to sulfur mustard agent vapors are generally comparable to first- or second-degree thermal burns. Sulfur mustard vapor rapidly penetrates the skin and reacts irreversibly with the underlying tissue. Warm, moist areas of the body are especially sensitive to the effects of the agent. Such body areas include the anal-genital area, armpits, insides of elbows and knees, and the neck.

Generally the first sign that appears following skin exposure to sulfur mustard agent vapor is erythema, or redness resembling sunburn, at the site of exposure. This effect normally appears between 2 and 24 hours after exposure. Erythema is usually accompanied by itching, burning, or stinging. If the exposure was mild, this may be the only sign and symptom evident on the skin.

Following a more severe exposure, blisters begin to form 2 to 18 hours after the onset of erythema. At first the blisters are small, but they may later combine to form large, dome-shaped, thin-walled, translucent, yellowish blisters surrounded by erythema. The fluid in the blisters is clear and does not contain mustard agent.

The blisters, like serious chemical burns, may not be painful initially. However, pain and itching may occur not long after the blisters develop, then subside, then reoccur a few days later as healing progresses.

Effects of Sulfur Mustard Vapor on the Airways

Inhalation of sulfur mustard agent vapor results in damage to the mucous membranes of the respiratory tract. The extent and severity of the damage depends on the amount of agent inhaled. Mild exposures may affect only the upper airways (nose and throat), while more severe exposures can also injure the lower airways.

Signs and symptoms of mild exposure generally appear between 6 and 24 hours after exposure and include runny nose, a burning sensation in the nostrils, sinus pain, sneezing, nose bleed, and sore throat. Almost all patients are hoarse, and many have a hacking or barking cough. Following a larger exposure, these effects may appear in as little as 2 hours and gradually progress to loss of voice, productive cough, and breathing difficulty.

Effects of Sulfur Mustard Vapor on the Gastrointestinal Tract

Patients often experience nausea and vomiting about the same time that eye or skin damage becomes apparent. For vapor exposure, no additional gastrointestinal effects are likely except for rare cases involving severe exposure.

Signs and Symptoms of Exposure to Sulfur Mustard Agent Liquid

Although much less likely than exposure to sulfur mustard agent vapor, in some circumstances exposure could occur due to direct contact of agent with the eyes or skin.

In these situations, the signs and symptoms are similar in nature to those resulting from vapor exposure, but they tend to be more severe and to appear more quickly.

Effects of Liquid Sulfur Mustard on the Eyes

Exposure to liquid sulfur mustard agent is much more damaging to the eye than exposure to agent vapor. If the exposure is severe, symptoms may be produced within minutes and can result eventually in severe damage to the cornea, loss of vision, and possibly loss of the eye. Decontamination immediately after exposure is necessary because the liquid agent is absorbed into the eye very rapidly.

Effects of Liquid Sulfur Mustard on the Skin

Signs and symptoms produced by direct skin contact with liquid sulfur mustard agent are similar in type to those resulting from exposure to agent vapor, although they are more severe and appear more quickly. Liquid sulfur mustard exposure may produce deeper damage, including the death of tissue at the site of exposure. The injury caused by skin exposure to liquid sulfur mustard agent may be comparable to a third-degree thermal burn.

Signs and Symptoms due to Ingestion of Sulfur Mustard Agent

Ingestion of food or water contaminated by liquid sulfur mustard injures the mucous membranes lining the gastrointestinal tract. This damage may result in nausea and vomiting, pain, and sometimes diarrhea or constipation. More severe effects on the gastrointestinal tract are uncommon.

Factors that Affect Sulfur Mustard Agent Signs and Symptoms

Time Factor (onset)

Although the signs and symptoms of sulfur mustard agent exposure are characteristically delayed, they may appear quickly if the person has been exposed to a large quantity. Just as with nerve agent exposure, the time to onset of symptoms depends on:

- type of agent
- amount of agent to which the patient has been exposed
- dose (how much patient has absorbed)
- duration of exposure
- route of exposure (inhalation, contact, ingestion)
- sensitivity of the patient's system (depends on general state of health, age, gender, etc.)

Peak Effect

If the exposure route is inhalation, the effects can begin to occur after a few hours of latency. The onset is usually accompanied by sneezing, coughing, and tracheobronchitis.

If the exposure route is dermal (droplet or liquid or vapor that comes into contact with the skin), the effects are usually delayed and absorption may continue for hours. This is likely to continue even after decontamination since the absorption may continue deep within the skin layer.

Toxicity

The toxic effects a person experiences after inhaling air containing nerve agent depends on the type of agent involved, the concentration of agent in the air, and the length of time that the individual inhales the air. A National Advisory Committee, working under the jurisdiction of the National Research Council's Committee on Toxicology, has developed estimates of the combinations of concentrations and exposure times that could produce specific levels of health effects for a single exposure. These levels, called Acute Exposure Guideline Levels (AEGLs) are summarized in Table 4.5. Notice that, for a given exposure duration, minor effects (AEGL-1) begin to appear at much lower concentrations than those required to produce severe effects (AEGL-3). More detailed information on AEGLS is available on information sheets[15] released by the U.S. Army Center for Health Promotion and Preventive Medicine.

Table 4.5 Example Sulfur Mustard Agent Acute Exposure Guideline Levels[17]

Acute Exposure Guideline Level	Duration of Exposure	Agent HD concentration (mg/m^3)
AEGL-3: Exposures above this level may cause increasingly severe delayed effects and incapacitation, including (delayed) blistering and lung effects; eventually potential fatalities.	8 hr 1 hr 10 min	0.27 2.1 3.9
AEGL-2: At exposures above this level there is an increasing chance of significant eye irritation with possible delayed impairment to vision; possible breathing difficulties approaching the AEGL-3; effects will be delayed but not permanent.	8 hr 1 hr 10 min	0.013 0.10 0.60
AEGL-1: Exposures above this level may begin to cause minor eye discomfort, irritation, redness. Effects will be delayed but not permanent.	8 hr 1 hr 10 min	0.0083 0.067 0.40

Lethality

The chief causes of death from blister agent exposure are usually

- respiratory failure due to damage caused to lungs and airways
- infections due to the combination of airway damage and suppressed immune response mechanisms (e.g., pneumonia, etc.).

However, sulfur mustard exposures are rarely lethal. For example, a massive number of soldiers were exposed to the agent in World War I, but only approximately 3% died.

Differential Diagnosis – Sulfur Mustard Agent

Many of the symptoms described above may be caused by health problems other than exposure to sulfur mustard agent. Emergency medical personnel must determine whether or not a person is suffering from actual sulfur mustard agent exposure in order to provide the appropriate treatment.

In some cases, emergency responders will know the identity of the agent before arriving at the scene. If a chemical agent release is detected from a stockpile location, authorities pass information about the release—including the agent involved—to response personnel. In other cases, such as a terrorist event, first responders may have to evaluate the signs and symptoms of victims to determine that a sulfur mustard agent release has occurred. They make this determination using a technique called "differential diagnosis." This is the process of distinguishing one disease from another when two or more diseases produce the same or similar effects.

Emergency medical personnel should suspect sulfur mustard agent poisoning whenever they encounter a patient exhibiting some of the signs and symptoms of blister agent exposure and no definite alternative explanation of these effects is apparent. If many casualties displaying symptoms of sulfur mustard agent exposure are encountered within a brief time, responders should suspect terrorism.

Individual signs and symptoms of sulfur mustard agent exposure may also be attributed to the following conditions[7, 11, 20, 21]:

- hay fever (red eyes)
- burns—thermal, sun, or chemical (reddened skin and blisters)
- exposure to other vesicant agents, such as nitrogen mustard, Lewisite, or phosgene (all signs and symptoms)
- severe exposure to tear gas (all signs and symptoms)
- exposure to poison ivy, poison oak, or other contact allergens (reddened skin and blisters)
- allergic reaction to drugs or other substances (reddened skin and blisters)
- insect bites or stings (reddened skin and blisters)

Differential diagnosis of sulfur mustard agent poisoning relies heavily on information obtained from the patient. Emergency medical personnel should ask the victim about the likelihood of exposure to sulfur mustard or to other chemicals or toxic plants as well as about other possible explanations for the victim's signs and symptoms.[11]

In particular, patients who may have been exposed to an unknown chemical should be asked about the length of time that passed between the exposure and the appearance of signs and symptoms. Pain and other effects occur immediately for many chemicals, but are delayed in the case of mustard agent exposure.[7]

Skin lesions caused by contact with toxic plants (e.g., poison ivy or poison oak) are usually linear in configuration and tend to occur on the most exposed areas of skin. Lesions caused by sulfur mustard exposure, on the other hand, are not linear in distribution and are usually most prominent in warm, moist areas of the body.[21]

Self-Check

Students can assess their understanding of the material presented in this module by completing the following Self-Check.

Matching

Identify the probable cause of the following signs and symptoms by matching the Sign/Symptom in Column A with the correct Cause in Column B. Notice that the choices in Column B also include: either and neither.

Column A
Signs/Symptoms

_____4-1. miosis
_____4-2. difficulty in breathing
_____4-3. increased oral/nasal secretions
_____4-4. sudden hair loss
_____4-5. twitching spasms
_____4-6. blisters
_____4-7. fever
_____4-8. a delayed, severe rash
_____4-9. localized sweating
_____4-10. overall weakness
_____4-11. intense itching in body crevices
_____4-12. extreme sore throat, irritated eyes, nose, and mouth
_____4-13. convulsions
_____4-14. temporary hearing loss
_____4-15. burning sensation in mucous membrane around eyes, in nose and mouth
_____4-16. giddiness
_____4-17. high blood pressure
_____4-18. extremely dilated pupils
_____4-19. severe burn-like blisters
_____4-20. irregular heart beat

Column B
Cause

a. nerve agent

b. blister agent

c. either; may be caused by both nerve and blister agents

d. neither; is not a sign/ symptom of nerve nor blister agent

Multiple Choice

4-21. As a general rule, the reaction time (symptom onset) to a nerve agent is immediate if

a. very small amounts are involved.
b. moderate to large amounts are inhaled.
c. a minute amount is absorbed through the skin in a localized area.
d. if moderate to large amounts are combined with a blister agent.

4-22. As a general rule, the reaction time (symptom onset) to a sulfur mustard agent is

a. delayed for severe cases
b. delayed for mild to moderate cases
c. immediate for mild to moderate cases
d. immediate regardless of the severity

4-23. Reaction to a nerve or blister agent, and whether or not a particular sign/symptom shows up at all, depends on several factors which include:

a. route of exposure; dosage concentration
b. the duration of the exposure; the common name of the agent
c. common name of the agent; altitude of the terrain at the time of exposure
d. the duration of the exposure; whether the agent is classified as unitary or binary

4-24. The chief cause of death due to nerve agent exposure is

a. cerebral hemorrhage
b. respiratory failure
c. severe bilateral miosis
d. severe gastroenteritis

4-25. "Differential diagnosis" means

a. a different care giver arriving at the same diagnosis
b. diagnosing multiple causes in order to give different treatments at the same time
c. arriving at the same diagnosis for different patients who display the same or similar effects
d. distinguishing one disease from another when two or more diseases produce the same or similar effects

True/False

4-26. After being exposed to a nerve agent, a patient will not necessarily display all of the signs/symptoms described in this student guide.

a. true
b. false

Explain:
_____
_____
_____
_____

4-27. After being exposed to a sulfur mustard agent, a patient will not necessarily display all of the signs/symptoms described in this student guide.

a. true
b. false

Explain:

_____
_____
_____
_____

Short Answer

4-28. List at least two factors that affect the onset of symptoms for a victim of nerve or blister agent exposure.

a. _____
b. _____

4-29. List at least two other causes that produce some of the same effects as exposure to nerve agents.

a. _____
b. _____

4-30. List at least two other causes that produce some of the same effects as exposure to sulfur mustard agents.

a. _____
b. _____

Check responses using Appendix A, Self-Check Answers.

Summary

The most important points covered in this module are:

- The specific signs and symptoms of nerve agent vapor/aerosol exposure
  - miosis (pinpointing of the pupils)
  - increased secretions
  - respiratory difficulty or collapse

- The specific signs and symptoms of blister agent vapor/aerosol exposure
  - eye irritation and inflammation
  - erythema (reddening of the skin)
  - blisters
  - respiratory irritation and distress

- Not all signs and symptoms may appear; dose, duration, and route of entry (among other factors) can make a difference.

MODULE 5 FIRST AID AND SPECIAL TREATMENT (FAST)

Objectives

After completing Module 5, students should be able to:

1. Describe the need for self-protection when providing treatment for nerve or blister agent exposure.
2. Describe first aid treatment for patients of nerve agent exposure.
3. Describe first aid treatment for patients of sulfur mustard agent exposure.

Introduction

The most important part of medical treatment given to patients of nerve and blister agents is first aid and special treatment. First responders must be prepared to act fast. The actions taken during the first few minutes, especially in cases of severe nerve agent exposure, are extremely critical and can make the difference between life and death.

Basic EMT training usually teaches the "ABCs of emergency care":

Airway (establish airway),
Breathing (artificial respiration),
Circulation.

The initial treatment for patients exposed to nerve and blister agent ranks just as high on this priority list. Just like the ABCs, there is no time to transport or wait for help. Emergency medical personnel must be prepared to act decisively and immediately.

The most important points to learn from this key module are:

- The Number 1 Rule in providing treatment for nerve or blister agent exposure is: PROTECT YOURSELF.

- The initial treatment (first-aid) for nerve agent patients is:

  - Give atropine, followed by 2-PAM Cl (authority to administer antidote compounds by non-physicians varies among states)
  - As necessary, support airway management

- The initial treatment (first-aid) for blister agent patients is:

  - Ensure that patient is decontaminated
  - If necessary, take precautions for sterile technique and support airway management as necessary.

- Atropine overdose can occur if given when there has been no exposure to nerve agent.

5.1 Protecting Yourself

The first rule in treating any patient of a toxic agent exposure is to protect yourself. Emergency responders should always remember that the chemical warfare agents were designed to spread and cause multiple injuries. In the midst of an emergency, when the adrenaline level is high and the responder is focused on helping the patient, it is easy to put aside concern for one's own welfare. This urge must be resisted. Responders who allow themselves to be exposed to the agent lose their ability to treat victims, add to the number of patients who must be treated, and undergo the pain and trauma resulting from the exposure.

Emergency medical personnel can protect themselves by

- recognizing the area with exposure potential and the zones set up for safe operations during the emergency,
- wearing the level of PPE appropriate for the zone in which they are working and for the level of contamination that may be present on the victim.

If a chemical agent release with potential off-post consequences should occur at one of the disposal locations, an initial hazard assessment will identify the expected pattern and path of off-post exposure. The area affected by the release will be restricted, as in other hazardous materials emergency response situations. Typically, three zones are established to promote safe response operations: (1) a hot zone adjacent to the spill or release, where there is a potential for exposure or contamination, (2) a warm zone where personnel and equipment are decontaminated upon leaving the hot zone, and (3) a cold zone which is assumed to be free from significant risk of exposure or contamination.

Depending on the zone in which they are working, it may be necessary for civilian responders, including EMTs, to wear protective clothing and respirators to ensure that they are not exposed to chemical agent. The CSEPP training course, *Personal Protective Equipment*, identifies the specific clothing and breathing equipment approved for use in CSEPP along with associated training, medical, and maintenance policies.

Decontamination: An Essential Part of Treatment

An essential part of any treatment provided for a patient of a nerve or blister agent exposure is decontamination; that is, neutralization or removal of the agent from the victim. Patients being treated in the cold zone will have been decontaminated before being brought to the treatment area. Medical responders treating patients in the hot or warm zone should be prepared to perform decontamination as part of the medical treatment protocol unless other responders have already done so.

Decontamination not only prevents the agent from doing any further damage to the specific patient, but also, if done correctly, prevents the agent from spreading to others and thereby producing other patients. Decontamination is needed within 2 minutes after exposure, preferably using soap and warm water. Specific instructions for step-by-step contamination reduction procedures, such as what materials to use and how to use them, are covered in the training course *Decontamination of People Potentially Exposed to Chemical Agents*.

5.2 Nerve Agent Exposure: Initial First Aid Treatment

Treatment for a severe nerve agent exposure must be immediate. Depending on the severity of the exposure, seconds can make the difference between life and death. Patients should be treated if they have at least two signs and symptoms of nerve agent poisoning. Minimal exposure may result only in mild miosis (slightly reduced pupil size) and a runny nose, requiring only decontamination and observation.

The first aid treatment for symptomatic patients of nerve agents includes three steps:

- Decontamination,
- Antidote administration, and
- Airway management.

The Antidote: A Combination

The initial treatment for nerve agent exposure comes as a two-part antidote: (1) atropine to counteract the cholinergic effect of the nerve agent, and (2) 2-PAM Cl to remove agent from cholinesterase and restore normal control of skeletal muscles.

Atropine is the first drug used for treating nerve agent exposure. It stops the effect of the nerve agent by blocking the effects of over-stimulation. This antidote relieves the smooth muscle constriction in the lungs and GI tract and dries up respiratory tract secretions. It cannot reverse respiratory muscle paralysis. Atropine is a drug that has long been used in medicine as a pre-anesthetizing agent.

After the initial dose, the need for additional atropine is re-evaluated every few minutes as indicated in Tables 5.1, 5.2, and 5.4. In cases of severe exposure, additional doses of atropine may be required during the entire trip to the emergency room.

The follow-up drug to atropine is pralidoxime chloride (also called 2-PAM Cl). This drug complements, or completes, the action of atropine by restoring normal functions at the nerve synapse by removing organophosphate from cholinesterase. This antidote is

effective at re-establishing normal skeletal muscle contraction (relieves twitching and paralysis of respiratory muscles). 2-PAM Cl has side effects if administered too much too rapidly. It should be administered slowly and carefully. Side effects include hypertension, blurred vision, and vomiting.

Laws Regarding Drug Administration

The laws concerning drug administration vary from state to state. Some states require that any administration of atropine or 2-PAM Cl be under the direct orders of a physician. Other states permit EMTs to administer these drugs in emergency situations. Each emergency medical care provider should be familiar with the state laws governing drug administration in emergencies.

How the Nerve Agent Antidote Works

In a patient suffering from nerve agent exposure, the target muscle or gland is over-stimulated because of the agent's interference with the normal chemical "stop and go" process between the nerve cell and the target cell. Affected muscles and glands over-react and affected organs malfunction. If the over-stimulation is not interrupted, musclescan eventually go into a prolonged contraction and become fatigued, or go limp.

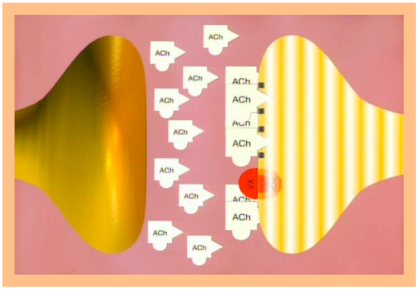


Figure 5.1. Nerve Agent Over-stimulates the Nervous System

When atropine is administered, it blocks the action of the accumulated acetylcholine from stimulating the target muscle or gland cell. As a result constriction of smooth muscles is relieved and respiratory secretions dry up.

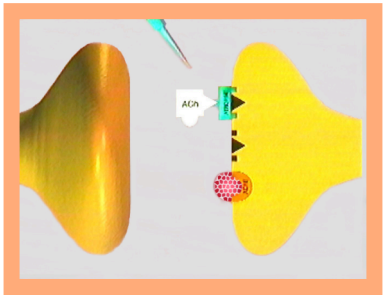
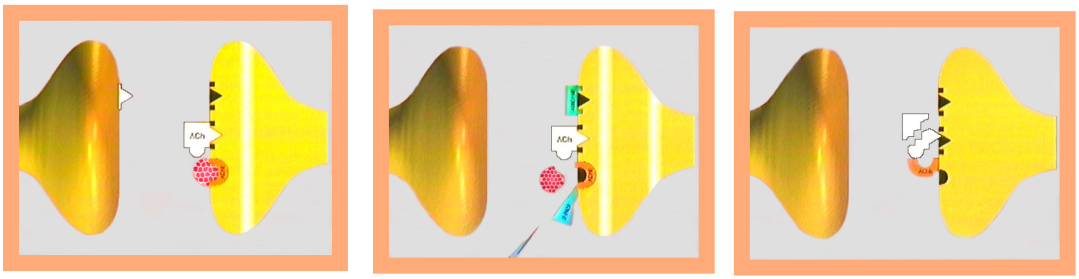


Figure 5.2. Atropine Blocks the Effects of Over-stimulation

Note that the atropine prevents part of the nerve agent action that had over-stimulated the muscle cell. What atropine does not do is free up acetylcholinesterase to restore normal control of target muscle cells.

That is why 2-PAM Cl is given in addition to atropine - it restores the activity of the inhibiting enzyme, acetylcholinesterase. This restores normal skeletal muscle contraction and relieves twitching and paralysis of the respiratory muscles, thus reducing the need for assisted ventilation.



Nerve agent is bonded to acetycholinesterase

2-PAM Cl removes agent from acetylcholinesterase

Acetylcholinesterase deactivates acetylcholine; normal skeletal muscle function is restored

Figure 5.3. 2-PAM Cl Restores Normal Control of Muscles

Recommended Dosages of Atropine and 2-PAM Cl

The appropriate dosages of atropine and 2-PAM Cl to administer after an exposure to nerve agent vary depending on the age and body weight of the patient and the severity of the signs and symptoms.

The recommended doses listed in this section may be repeated as clinically indicated. Atropine treatment should be repeated until the patient is “atropinized” (discussed later in this module). Incremental 2-PAM Cl dosages may be repeated until the maximum dose based on body weight has been given. The treatment lists in this module sometimes indicate that 2-PAM Cl should be given by a “slow IV.” This means that the recommended dosage should be administered over a 20- to 30-minute period in 250 ml of normal saline or 250 ml of 5% dextrose/water solution.

Experience has revealed two common problems with the administration of nerve agent antidotes. The most common problem is underdosage, when too little antidote is administered to relieve the effects of the nerve agent. The second most common problem is the administration of antidotes to patients who have not actually been exposed to nerve agent. Atropine, one of the key antidotes, can produce adverse effects when administered to someone who has not been exposed to organophosphates. However, the most serious problem would be failing to administer atropine when it was needed.

While nerve agent antidotes can be administered intravenously (IV), during emergency situations in the field, they are more likely to be administered intramuscularly (IM). IM injections can be given with a syringe or with an auto-injector—a device containing a

pre-measured dose of antidote that is automatically activated when pressed firmly against the patient's outer thigh. The Food and Drug Administration has approved auto-injectors for civilian use in instances involving nerve agents and organophosphorus insecticides. Meridian Medical Technologies, Inc. (MMT), manufactures and markets auto-injectors containing atropine and 2-PAM Cl.

The U.S. Army and the CSEPP communities use the Mark I Nerve Agent Antidote Kit, a MMT product containing one 2-mg atropine auto-injector and one 600-mg 2-PAM Cl auto-injector. This kit is useful in treating people over 10 years of age. 2-PAM Cl injectors are not available for children weighing less than 50 pounds.

The manufacturer, MMT, also offers a line of atropine auto-injectors—called AtroPen®—for civilian use. These devices come in four dosage levels for administering atropine to individuals of various weights and ages, ranging from adults to infants. Since the Mark I kit is currently used in CSEPP, this section concentrates primarily on medical treatment using this device. However, individual communities may want to consider looking into AtroPen® injectors for treating non-adults exposed to nerve agents.

Detailed and practical information on the administration of antidotes via auto-injectors is available in the CSEPP training course, *Use of Auto-injectors by Civilian Emergency Medical Personnel*.

In addition to the two–drug antidote, diazepam should be administered as an anticonvulsant to patients who are experiencing convulsions and to all patients who display signs of severe nerve agent exposure. The recommended dosages of diazepam vary depending on the patient's age. Auto-injectors containing adult dosages of diazepam are available from MMT.

Adult Dosages

When treating adults who have been exposed to a nerve agent, the appropriate dosages of nerve agent antidotes are based on the severity of signs and symptoms. Table 5.1 lists the recommended antidote treatment for adults and larger adolescents (over 90 pounds) exposed to nerve agent.

Table 5.1. Recommended Treatment of Adults Exposed to Nerve Agent[7, 8]

Severity of Signs/Symptoms*	Initial Treatment	Short-term Follow-on Treatment
Minimal—*Vapor Exposure:* pinpoint pupils with or without runny nose, reflex nausea and vomiting	Observation only (1 hr for inhalation exposure; 18 hrs for liquid contact exposure)	
Mild—*Vapor Exposure:* pinpoint pupils, dim or blurred vision, runny nose, slight breathing difficulty, secretion of tears/ saliva/phlegm, reflex nausea and vomiting; *Liquid Exposure:* localized sweating, localized muscular twitching	• Atropine: 2 mg IM or IV • 2-PAM Cl: 600 mg IM, or 1 g via slow IV	If no improvement within 5 min, give additional 2 mg atropine, 600 mg 2-PAM-Cl. Repeat 5 to 10 min later if condition worsens.
Moderate—*Vapor Exposure:* pinpoint pupils, dim or blurred vision, excessively runny nose, moderate to severe breathing difficulty, plentiful secretion of tears/saliva/phlegm, reflex nausea and vomiting, diarrhea, weakness; *Liquid Exposure:* same as for Mild exposure, plus nausea and vomiting, diarrhea, generalized sweating, generalized weakness/tiredness/ill feeling	• Atropine: 2-4 mg IM or IV • 2-PAM Cl: 600-1200 mg IM, or 1 g vial slow IV**	If symptoms continue after 5 to 10 minutes, repeat initial treatment up to two times. If symptoms still unrelieved, give 2-mg atropine doses at 5- to 10-min intervals until atropinized. (No more than three 600 mg doses of 2-PAM Cl should be given.)
Severe—*Vapor or Liquid Exposure:* same as for Moderate exposure, plus loss of consciousness, convulsions, generalized muscular twitching, flaccid paralysis, cessation of breathing, involuntary urination/ defecation	• Atropine: 6 mg IM • 2-PAM Cl: 1800 mg IM, or 1 g via slow IV • Ventilation support with oxygen, and suctioning of secretions • Diazepam: 10 mg IM or 5 mg IV	Once ventilation support is started, immediately give additional 2 to 4 mg atropine IV (if capability is available) or IM. Continue giving additional 2 mg doses of atropine at intervals of several minutes until patient is getting adequate oxygen (likely requiring ventilation assistance). Up to two additional doses (10 mg) of diazepam may be given at 10-min intervals to convulsing patients.

* Individual patients may not exhibit all of the signs and symptoms listed.

** If exposure was through contact with liquid agent and less than about 1 hr has passed since exposure, administer 4 mg atropine and 1,200 mg 2-PAM Cl (two Mark I Kits). If several hours have passed, administer 2 mg atropine and 600 mg 2-PAM Cl (one Mark I kit).

Dosages for Adolescents, Children, and Infants

A different antidote treatment regimen is followed for non-adults suffering from nerve agent intoxication. Here the appropriate dosages of the antidotes depends on the patient's age or weight. The recommended initial and follow-on dosages of atropine, 2-PAM Cl, and diazepam are listed in Table 5.2. As was the case with adults, severely exposed children will likely require ventilation support with oxygen and removal of secretions.

Table 5.2 Nerve Agent Antidote Dosages for Adolescents, Children, and Infants[6]

Antidote	Patient's Age/Weight	Initial Treatment	Short-term Follow-on Treatment
Atropine	> 10 yrs	2 mg IM or 0.02 mg/kg body weight IV	Repeat every 5-10 min until atropinized
	2 – 10 yrs	1 mg IM or 0.02 mg/kg body weight IV	Repeat every 5-10 min until atropinized
	< 2 yrs	0.5 mg IM or 0.02 mg/kg body weight IV	Repeat every 5-10 min until atropinized
2-PAM Cl	> 50 lbs (22 kg)	600 mg IM or 15 mg/kg body weight via slow IV*	IM dose can be repeated twice at 5- to 10-min intervals; IV dose can be repeated twice at intervals of 1 hr
	< 50 lbs (22 kg)	15 mg/kg body weight via slow IV*	Dose can be repeated twice at intervals of 1 hr
Diazepam	> 5 yrs	1 mg IV	Repeat, if needed, every 2 to 5 min to a maximum of 10 mg
	30 days to 5 yrs	0.2 to 0.5 mg/kg body weight IV	Repeat, if needed, every 2 to 5 min to a maximum of 5 mg

* Slow IV dosages are expressed as milligrams of antidote per kilogram of the patient's body weight. They are to be delivered over a 20- to 30-minute period in 250 ml of normal saline or 250 ml of 5% dextrose/water solution.

Upon recognizing that an infant, child, or adolescent is exhibiting at least two symptoms of mild-to-moderate exposure to nerve agent or any symptom of severe exposure, emergency medical personnel should determine the correct antidote dosages for the patient's age or weight. The appropriate atropine dosage should be administered immediately, followed by an initial dose of 2-PAM Cl. Diazepam should then be administered to patients who are convulsing or displaying signs of severe exposure. Dosages of the drugs should be repeated, if needed, as described in Table 5.2.

How Much Atropine Is Enough?

Frequently mentioned in this student guide is the fact that atropine tends to be **underdosed** rather than overdosed. While there is an optimum dosage, the precise amount needed is often difficult to determine because the exact amount of nerve agent exposure is often impossible, or difficult at best, to determine. To help answer the question, "How much antidote is enough?," use the following guidelines for atropine administration:

1. Make sure atropine is warranted before it is given. The second most common error is giving atropine when the only sign is miosis.

2. Administer the dosage of atropine recommended in Table 5.1 or 5.2. Follow up with the recommended dosage of 2-PAM Cl.

3. Continue administering the prescribed dosages of atropine as recommended in Tables 5.1 or 5.2 until signs of “atropinization” appear.

4. If needed, repeat dosage of 2-PAM Cl, as recommended in Tables 5.1 or 5.2, until the maximum total dose has been given.

“Atropinization” is the term used to describe the noticeable signs that a nerve agent patient has received ample amounts of atropine—the effect of treating with sufficient atropine to decrease respiratory symptoms, relieve bronchospasm, stop sweating, and relieve diarrhea and abdominal cramping. Recognition of “atropinization” helps to determine that the patient is not underdosed (a common mistake made by care providers when treating nerve agent patients).

Watch for these signs and symptoms to determine that enough atropine has been given:

- secretions are dry (eyes, nose, bronchi, and mouth are no longer runny)
- breathing is less labored

A severely affected patient may need up to 20 mg of atropine the first day. A single patient may require four injections, or more if symptoms dictate, on the way to the hospital.

Atropine Overdose

Adverse side effects can result if atropine is given to a person who has not been exposed to a nerve agent. Although the most common mistake in providing atropine treatment (when nerve agent exposure is certain) is to underdose rather than overdose, an overdose of atropine can result in undesirable effects. Most likely, however, atropine poisoning is caused when it is given but was not needed at all. In comparing the consequences of both mistakes regarding atropine administration, overdosing is far less serious. Atropine overdose is not usually life-threatening.

Signs and Symptoms of Atropine Overdose

The following signs and symptoms help to identify atropine effects.

Dilated (large) Pupils

The patient’s pupils are dilated. Notice that this effect is directly the opposite effect of the nerve agent. The nerve agent causes miosis, or pinpointing of the pupils. When atropine is given in the absence of nerve agent intoxication, the pupils are opened almost completely.

Dry Mouth and Skin

The patient's mouth and skin are dry. Notice that this effect is also the direct opposite of the nerve agent effect. While the nerve agent induces sweating and copious salivation, atropine overdose causes dryness. Other indications of an atropine overdose are:

- Rapid pulse
- Red (flushed) skin
- Difficulty urinating
- Confusion; visual hallucinations; delirium
- Temperature control is diminished, resulting in high fever and hot skin
- Intense thirst
- Restlessness

Differential Diagnosis of Atropine Overdose

Other causes have been known to produce similar signs:

- Heat stroke
- Locoweed
- Atropine-like medicines used in treating ulcers or other conditions

Treatment for Atropine Overdose

- Keep patient cool.
- Protect patient from irrational actions.
- Transport patient to hospital as soon as possible.

Respiratory Support

Remember, the chief cause of death due to severe nerve agent exposure is respiratory failure. If the patient has been exposed to a moderate to heavy dose of nerve agent, it is quite likely that respiratory support will be necessary. This support may range from administering oxygen (if breathing is difficult) to providing ventilation, airway management and suctioning secretions as needed.

In severe nerve agent poisonings, it may not be possible to correct respiratory trouble until enough atropine has been given. This is because of the intense bronchoconstriction and heavy secretions that are characteristic of severe poisoning. In treating this condition, **administer atropine first**, then begin efforts to ventilate the patient and establish a patent airway.

Ventilation

In some cases, ventilation support may be required. If the patient must be ventilated, the insertion of an endotracheal tube by a qualified person is recommended. High pressure is required to overcome the resistance of the narrowed airways and increased secretions.

Some respirating devices, including some that are common on ambulances, “pop-off” at about 40–45 cm H_2O and will not deliver the pressure needed to treat a severely poisoned adult who is “un-atropinized.” Emergency medical personnel should check the pressure relief rating of their manual or powered respirator. Pressures of 50 to 70 cm H_2O or greater may be needed to ventilate severely poisoned patients.

Resuscitation

If breathing has stopped, give artificial respiration using an approved mask-bag oxygen delivery system.

5.3 Blister Agent Exposure: Initial First Aid Treatment

Treatment for a blister agent exposure must be immediate. There is no available antidote for blister agent poisoning. The best first aid treatment that can be offered consists of immediate removal from the exposure source and decontamination, use of sterile technique, and airway management as necessary.

Treatment for Eye Contact

If the agent has contacted the eyes, speed in decontamination is especially critical. Irreversible damage may occur to the eyes and skin very quickly, even though the effects of this damage may not begin to appear for one to three hours.

Flush the eyes immediately with water by tilting the head to the side, pulling the eyelids apart with the fingers and pouring water slowly into the eyes. Do not cover eyes with bandages. Make sure that hands and fingers (or gloves, if they are being worn) are free of contamination before performing this procedure.

Decontamination of the eyes becomes less effective with the passage of time as the agent rapidly bonds to the tissue. Once signs and symptoms begin to appear, not much benefit is gained by painfully prying swollen eyelids apart to flush with water.

If the eyes have been exposed to a blister agent the person may experience photophobia (sensitivity to light). Dark or opaque glasses help shield the eyes from the light and provide relief from photophobia.

If eye injuries are severe, homatropine or other pupil dilators should be applied topically to prevent scarring. Eye pain should be relieved using oral analgesics as topical analgesics may delay healing and allow damage to the cornea. Topical antibiotics should be topically several times a day, and petroleum jelly applied to the lid edges will prevent them from sticking together.

Treatment for Skin Contact

When performing decontamination procedures for blister agents, pay special attention to skin creases (groin, armpits, behind ears, between fingers, under breasts, etc.). These are the areas where victims of sulfur mustard exposure are most likely to experience severe blistering.

Once at the health care facility, additional treatment for the blisters may be given, as determined by the attending physician. This may include administration of antibiotics (including application of topical antibiotics) and other treatments common for burn injuries. Calamine or other soothing lotions to relieve burning may be all that is needed if the only effect is a rash.

If the patient is suffering from severe blistering, sterile techniques must be observed to avoid introducing infection into the blister wounds. Because of the damage to the skin tissue and the immunosuppressive action of sulfur mustard, the patient's ability to fight infection may be diminished. This same precaution is used for any type of massive surface injury (such as thermal burns). Fluid from ruptured blisters does not contain sulfur mustard and is not itself a vesicant.

Treatment for Inhalation

Respiratory support may be required if the patient has inhaled significant amounts of blister agent vapor or aerosols. If blisters have formed in the respiratory tract, breathing may be difficult. Administer oxygen as needed. Providing humidified air or oxygen and cough suppressants may relieve sore throat, hoarseness, and coughing.

Intubation may be needed if airway injury is severe. In this case it should be performed before it is made difficult by constriction of the larynx and swelling of affected tissues. Bronchodilators may help relieve the constriction of the air passages of the lungs, and, if these are not effective, steroids may be given.

If breathing has stopped, resuscitate the person. Additional intravenous fluids may be needed, but fluid replacement is not as serious a problem as it would be for thermal burns affecting the same area. Do not overload with intravenous fluids.

Self-Check

Students can assess their understanding of the material presented in this module by completing the following Self-Check.

Multiple Choice

5-1. The FIRST step in providing treatment to a patient of nerve or blister agent exposure is:

a. protect yourself
b. stop the bleeding
c. decontaminate the patient
d. transport to the hospital

5-2. The FAST steps for nerve agent exposure patients must be done:

a. immediately
b. within 48 hours of the time of exposure
c. by licensed EMTs under official supervision of the U.S. Army

5-3. The authority to administer atropine and pradiloxime chloride under emergency conditions is granted by

a. the Department of Homeland Security
b. the U.S. Army
c. the CSEPP Program
d. local or state statutes

5-4. The antidote for patients of nerve agent exposure consists of the following drug or drug combination

a. atropine only
b. 2-PAM Cl only
c. both atropine and 2-PAM Cl
d. either atropine or 2-PAM Cl

5-5. Nerve agent antidotes may be administered

a. intramuscularly (IM)
b. intravenously (IV)
c. with auto-injectors
d. any of the above

5-6. The best indicator used to ensure that adequate amounts of atropine are given is

a. signs of atropinization
b. the disappearance of miosis
c. blood pressure of 110/90 or lower
d. the disappearance of the skin rash

5-7. The most common mistake made when administering atropine is

a. overdosing
b. underdosing

5-8. Atropine overdose may occur by administering atropine

a. in doses that are too small
b. without administering 2-PAM Cl
c. when the person has not been exposed to a nerve agent
d. when the person has not been exposed to a blister agent

5-9. By far, the MOST serious of the following conditions are

a. atropine underdosing
b. atropine overdosing
c. not giving atropine when it is needed
d. giving atropine without giving 2-PAM Cl

5-10. In order to determine if additional atropine is needed after the initial dose, an adult patient with severe signs and symptoms of nerve agent exposure should be re-evaluated

a. continually, every 30 minutes
b. continually, every 3-5 minutes
c. at the scene, before transport, at the receiving hospital
d. once at the scene and then again, if necessary, at the receiving hospital

5-11. When treating a blister agent exposure patient, sterile precautions may be necessary because

a. the fluid in the blisters can spread the agent
b. sterile technique accomplishes the same results as decontamination
c. damage done to the skin can permit infection if the blisters are severe
d. treatment provided later, at the hospital, requires that the blisters be as sterile as possible for testing

Short Answer

5-12. List 2 main FAST (first aid & special treatment) steps required for nerve agent exposure patients.

a. _____
b. _____

5-13. List 2 main FAST (first aid & special treatment) steps that may be necessary for blister agent exposure patients.

a. _____
b. _____

5-14. List 2 specific signs that signify that sufficient atropine has been administered

a. _____
b. _____

Summary

The most important points covered in this key module are:

- The Number 1 Rule in providing treatment for nerve or blister agent exposure is: PROTECT YOURSELF.

  Emergency responders should always remember that the chemical warfare agents were designed to spread and cause multiple injuries. Responders who allow themselves to be exposed to the agent lose their ability to treat victims, add to the number of patients who must be treated, and undergo the pain and trauma resulting from the exposure. Emergency medical personnel can protect themselves by

  - recognizing the area with exposure potential and the zones set up for safe operations during the emergency,
  - wearing the level of PPE appropriate for the zone in which they are working and for the level of contamination that may be present on the victim.

- The initial treatment (first-aid) for nerve agent patients is:

  - Give atropine, followed by 2-PAM Cl (authority to administer antidote compounds by non-physicians varies among states)
  - Support airway management as needed; resuscitate if breathing has stopped

- The initial treatment (first-aid) for blister agent patients:

  - Ensure that patient is decontaminated
  - If necessary, take precautions for sterile technique and support airway management as necessary.

- Atropine underdose is the most common mistake in cases of severe nerve agent exposure.

- Atropine overdose can occur if given when there has been no exposure to nerve agent. This is less serious than atropine underdose.

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Useful Web-based Information Sources

Additional useful information regarding the topics addressed in this training course is available on the worldwide web. In particular, the websites listed below contain information that is pertinent and authoritative. While the listed web addresses were accurate when this Student Guide was published, please note that, due to the fluid nature of the web, they may have changed since then. If you find that any of these addresses is no longer valid, we recommend that you look up the web address of the sponsoring agency or organization and search for the desired document or topic.

Information on the composition of the chemical agent stockpile, including the agents currently stored at each stockpile location, can be obtained by e-mailing the public affairs office of the U.S. Army's Chemical Materials Agency at public_affairs@cma.army.mil

LIST OF JOB AIDS

The following job aids have been prepared to assist in applying the knowledge learned in this course.

- Nerve Agents—a poster outlining the signs and symptoms of nerve agent exposure and the appropriate medical treatment)

- Blister Agents—a poster outlining the signs and symptoms of blister agent exposure and the appropriate medical treatment)

- Classification of Events—a poster describing (1) the four notification levels an Army installation would use to describe an incident involving the chemical stockpile to off-post officials, and (2) the appropriate community response for each notification level.

These job aids can be obtained in two ways:

- They can be downloaded from the CSEPP Technical Training Website at the following url: http://emc.ornl.gov/CSEPPweb/TRAINDocumentation.html

- They can be requested from the following source:

  Robert W. Norville
  Training Manager: CSEPP
  U.S. Department of Homeland Security: Preparedness
  (703) 605-1209
  robert.norville@dhs.gov

GLOSSARY

Acetylcholine	A chemical compound formed from acetic acid and choline which causes muscles to contract. It is a neurotransmitter found in various organs and tissues of the body. It is rapidly broken down by an enzyme, cholinesterase. Excessive accumulation of acetylcholine at the synapse (such as found in nerve agent poisoning) may prolong the transmission of impulses between nerve cells and muscles, between nerve cells and glands and cause excitatory effects in the central nervous system.
Acetylcholinesterase	An enzyme which normally stops the action of acetylcholine. This occurs as soon as acetylcholine has produced a muscle contraction or glandular stimulation. Nerve agents combine with acetylcholinesterase to prevent it from inactivating acetylcholine.
Allergen	Any substance which is able to cause an allergic condition.
Antidote	A substance used to counteract adverse effects resulting from toxic agent exposure. Nerve agent antidotes (atropine and 2-PAM chloride) block the effects of agent-induced skeletal and smooth muscle contraction (relieve loss of breathing control) and reduce glandular paralysis (dry up the excess respiratory secretions that make normal breathing difficult). Diazepam relieves agent-induced convulsions. There are no specific antidotes for mustard agent poisoning because the agent reacts too quickly with body tissue.
Atropine	A medication used as an antidote to organophosphate poisoning such as might result from exposure to nerve agents or some insecticides. Atropine counteracts the excess acetylcholine that accumulates at nerve endings thus relieving the bronchoconstriction and bronchial secretions that interfere with breathing. Atropine cannot reverse respiratory muscle paralysis. It is best to use 2-PAM Cl with atropine.
Atropinization	The presence of noticeable signs that a nerve agent patient has received ample amounts of atropine. Secretions will be dry and ventilation easy; conscious patients will be breathing comfortably.

Blister (vesicant) agent	A chemical warfare agent which produces local irritation and damage to the skin and mucous membranes, pain and injury to the eyes, reddening and blistering of the skin, and, when inhaled, damage to the respiratory tract. Blister agents include mustards (HD, H, and HT), and arsenicals (L). The mustards are also human carcinogens.
Bronchoconstriction	Constriction of the bronchial tubes which tends to trap air in the lungs.
Bronchodilator	A drug that relaxes bronchial muscle resulting in expansion of the bronchial air passages; used to relieve bronchospasm.
Bronchospasm	Constriction of the air passages of the lungs by spasmodic contraction of the bronchial muscles.
Carbamate	A group of cholinesterase-inhibiting insecticides resembling organophosphates; common examples include carbaril and sevin.
Carcinogen	Any cancer-causing substance.
Chemical event	A term used by the military that includes (1) chemical accidents and leaks resulting from nondeliberate events where safety is of primary concern, or (2) chemical incidents resulting from deliberate acts or chemical acts where security is a concern.
Chemical Stockpile Disposal Program (CSDP)	The Congressionally-mandated program that requires the Army to dispose of all its unitary chemical agents.
Chemical Stockpile Emergency Preparedness Program (CSEPP)	A joint program of the Department of the Army and the Department of Homeland Security that oversees and assists in the development of emergency response plans and capabilities for all jurisdictions that might be affected by a chemical agent release associated with stockpile storage of CSDP activities.
Chemical warfare agent	A chemical substance which, because of its physiological, psychological, or pharmacological effects, is intended for use in military operations to kill, seriously injure, or incapacitate humans (or animals) through its toxicological effects.
Cholinergic	Referring to acetylcholine or nerve endings which liberate acetylcholine. Acetylcholine transmits the nerve impulse across the neuromuscular junction.

Cholinesterase (Ache)	Acetylcholinesterase is the enzyme which normally breaks down the neurotransmitter acetylcholine after it has transmitted a signal from a nerve ending to another nerve, muscle, or gland. Organophosphate pesticides and military nerve agents block the normal activity of Ache which results in the accumulation of excess acetylcholine at nerve endings.
Cholinomimetic	Having an action similar to that of acetylcholine.
Coniine	A poisonous alkaloid prepared from the poisonous hemlock plant that causes gastrointestinal irritation and paralysis with respiratory failure.
Conjunctivitis	Inflammation of the delicate membrane that lines the eyelids and covers the exposed surface of the white of the eye.
Contamination	The deposition of chemical agents on personnel, clothing, equipment, structures, or areas. Chemical contamination may consist of liquid, solid particles, or vapor hazards. Vapor hazards are probably the most prevalent means of contaminating the environment.
Cornea	The clear, transparent front portion of the eye, comprising about one-sixth of its surface through which light passes to transmit images to the retina.
Cyanide	A rapidly acting, potentially deadly chemical that can exist in gaseous or crystal form. Cyanide kills cells by preventing them from obtaining oxygen. Mild exposure results, within minutes, in rapid breathing, restlessness, dizziness, weakness, headache, nausea and vomiting, and rapid heart rate. Severe exposure may also cause convulsions, low blood pressure, slow heart rate, loss of consciousness, lung injury, and fatal respiratory failure.
Decontamination	The process of reducing or removing hazardous substance, including chemical warfare agents, by physical means or by chemical neutralization (detoxification) so they are no longer hazards. Decontamination is routinely used to remove potentially harmful substances from personnel, equipment, laboratory areas, workplaces, outdoor areas, clothes, food, water and sewage.
Emphysema	A condition in which air spaces in the lungs are enlarged; usually accompanied by labored breathing, a husky couth, and frequently by impaired heart action.
Endotracheal tube	A tube inserted into the trachea to maintain an open and unobstructed airway and prevent aspiration.
Erythema	Red area of the skin, caused by heat or cold injury, trauma, or inflammation.

Fasciculation	Localized contraction or twitching of muscle fibers, usually visible through the skin.
Fentanyl	A highly potent, morphine-like drug used in the treatment of pain, often in conjunction with other drugs. Fentanyl is used illicitly as a substitute or enhancer of heroin. It may cause potentially fatal respiratory depression.
Flaccid paralysis	Loss of muscle tone and capability to function. Nerve agents cause this condition.
G-agent	A liquid nerve agent such as GA or GB.
GA	Also known as “Tabun.” See nerve agent.
GB	Also known as “Sarin.” See nerve agent.
H	See sulfur mustard agent.
HD	See sulfur mustard agent.
HT	See sulfur mustard agent.
Lewisite	A fast-acting vesicant, tear-producer, and lung irritant (chlorovinyldichloroarsine). All Lewisite in the U.S. Army’s chemical agent stockpile has been destroyed.
Microgram	One-millionth of a gram.
Miosis	Pinpoint or small pupils.
Muscarine	A naturally occurring poison found in certain mushrooms. It mimics the action of the neurotransmitter acetylcholine, producing the effects of increased salivation, sweating, and tearing within 15 to 30 minutes of ingestion. Larger doses may also cause abdominal pain, severe nausea, diarrhea, blurred vision, labored breathing, and (rarely) fatal cardiac or respiratory failure.
Mustard agent	See Sulfur mustard agent.
Neuromuscular blocking drug	A drug that blocks the transmission of impulses at the neuromuscular junction thus causing paralysis of the affected skeletal muscles. Such drugs are used medically to induce paralysis during surgery. These drugs may cause paralysis of the diaphragm, and some of them may result in low blood pressure, rapid heart rate, flushing, muscular weakness, mild breathless, and a general ill-feeling.
Nerve agent	Lethal organophosphate agent (forms include GA, GB, and VX) that is colorless and odorless. Can be fatal upon skin contact or when inhaled or ingested; attack the central nervous system and parts of the autonomic nervous system by complexing with and inactivating acetylcholinesterase.

Nitrogen mustard	Any of various toxic blistering compounds analogous to sulfur mustard but containing nitrogen instead of sulfur.
Organophosphate	Compounds with a specific phosphate group which inhibits acetylcholinesterase. Include substances used in chemical warfare and as insecticides.
OSHA	Occupational Safety and Health Administration: responsible for workplace safety regulation, including HazMat responder training standards.
2-PAM Cl	(Also called Pralidoxime chloride, or Protopam® chloride.) An antidote to organophosphate poisoning such as might result from exposure to nerve agents or some organic insecticides. The drug, which helps restore an enzyme called acetylcholinesterase, must be used in conjunction with atropine to be effective. Restores normal control of skeletal muscle contraction (relieves twitching and paralysis).
Persistent agent	A chemical agent that continues to present a hazard for considerable periods after delivery, by remaining as a contact hazard and/or by vaporizing slowly to produce a continuing hazard by inhalation. Generally, may be in a solid or liquid state.
Phosgene	A colorless gas that causes rapidly fatal pulmonary edema or pneumonia on inhalation; used in the synthesis of a number of organic compounds and formerly used as a chemical warfare agent.
Pneumonia	Inflammation of the lungs accompanied by fluid accumulation in the alveoli or air sacs, usually caused by an infective agent. May be secondary to chemical or physical injury to the lungs.
Rhinorrhea	Thin watery discharge from the nose.
Sarin	A nerve agent of organophosphate type that inhibits acetylcholinesterase. Is used in chemical warfare (GB).
Sulfur mustard agent	A vesicant chemical warfare agent (forms include H, HD, and HT) which has been used extensively in warfare. Creates destruction of epidermis, eye and pulmonary injury, and, in high doses, bone marrow depression. It is also a human carcinogen.
Tabun	A nerve agent member of the organophosphate type that inhibits acetylcholinesterase. Is used as a chemical warfare agent. Is the least toxic of the nerve agents but can cause death rapidly. (GA – only in stockpile at Deseret Chemical Depot)
Toxic chemical agent	Any chemical agent that is toxic, which creates a casualty, incapacitates, or kills.

Tracheobronchitis	Inflammation of the windpipe (trachea) and/or its two major divisions (bronchi); produces chronic cough.
V-Agent (VX)	A nerve agent of the organophosphate type that inhibits acetylcholinesterase.
Vesicant	A chemical blister agent that injures the eye and the lungs and burns or blisters the skin. Examples are H, HT, HD, and L.
Vesication	Blistering.

APPENDIX A SELF-CHECK ANSWERS

Module 3. Chemical Agents: Characteristics and Effects

3-1.	a
3-2.	c
3-3.	d
3-4.	d
3-5.	d
3-6.	a
3-7.	d
3-8.	b
3-9.	c
3-10.	a
3-11.	a
3-12.	d
3-13.	d

Module 4. Signs and Symptoms of Exposure

4-1.	a
4-2.	c
4-3.	a
4-4.	d
4-5.	a
4-6.	b
4-7.	d
4-8.	b
4-9.	a
4-10.	a
4-11.	d
4-12.	b
4-13.	a
4-14.	d
4-15.	b
4-16.	a
4-17.	d
4-18.	d
4-19.	b
4-20.	d
4-21.	b
4-22.	b
4-23.	a
4-24.	b
4-25.	d

4.26. a (Some patients may exhibit only one or two signs, others may experience several symptoms at the same time; factors such as dose received and individual sensitivity contribute to the onset of symptoms).

4-27. a (Some patients may exhibit only one or two signs, others may experience several symptoms at the same time; factors such as dose received and individual sensitivity contribute to the onset of symptoms).

4-28. amount of agent; dose concentration; duration of exposure; sensitivity of the patient's system; route of exposure

4-29. epilepsy; gastroenteritis; carbamates

4-30. sunburn; chemical burn

Module 5. First Aid Special Treatment (FAST)

5-1. a

5-2. a

5-3. d

5-4. c

5-5. d

5-6. a

5-7. b

5-8. c

5-9. c

5-10. b

5-11 c

5-12. Administer atropine/2-PAM chloride; support airway management as necessary

5-13. any 2 of: decontamination of patient; take sterile precautions; support airway management

5-14. secretions are dry; breathing is easy

APPENDIX B FINAL EXAM

Multiple Choice

1. The specific nerve agents currently in the U.S. Army's chemical stockpile inventory are:

   a. VX and mustard
   b. VX, mustard and Lewisite
   c. VX and GB
   d. Lewisite and GB

2. Toxic chemicals that are categorized as "nerve agents" are so called because

   a. they attack the body's nervous system
   b. the agent is essential to the nervous system
   c. they send "electrical" shooting pains throughout the nervous system
   d. the blisters they cause are extremely painful to the nerve endings in the skin tissue

3. Nerve agents affect the nervous system by

   a. killing off the nerve cells
   b. making the central nervous system shut down
   c. blocking the messages sent to the muscle and gland cells
   d. causing the nerve endings to stimulate the connected nerve/muscle/gland into a state of overstimulation

4. A person can be exposed to a nerve agent through all of the following routes EXCEPT:

   a. touching a surface that has the nerve agent on it
   b. inhalation of air that has been contaminated by a nerve agent
   c. eating or drinking anything that has been contaminated by a nerve agent
   d. touching the fluid that seeps from a blister of a person who has been exposed to a blister agent

5. Which of the following skin descriptions presents the most critical condition for nerve agent exposure?

   a. hairy chest
   b. sunburned calf
   c. hand wearing a glove
   d. forearm with cuts and abrasions

6. Toxic chemicals that are categorized as “blister agents” are so called because

   a. an inevitable side-effect caused by the drug antidote for this agent has been known to cause blisters in most people
   b. the most noticeable effects are blisters and the destruction of cells in target tissues
   c. they cause the body to break out in painless sores that resemble tiny chicken pox-like blisters
   d. it was named after the chemist who discovered its potential wartime use

7. It is often difficult to detect an exposure to a mustard agent immediately because

   a. the reaction is delayed
   b. the blister does not form for several weeks after exposure
   c. pain is felt immediately but it is difficult to know what the cause of it is
   d. once the blister agent has contacted the skin, the victim no longer has any sensation in that area

8. A person can be exposed to a blister agent through all of the following routes EXCEPT:

   a. touching a surface that has the blister agent on it
   b. inhalation of air that has been contaminated by a blister agent
   c. eating or drinking anything that has been contaminated by a blister agent
   d. touching the fluid that seeps from a blister of a person who has been exposed to a blister agent

9. Although any form of public exposure during the chemical stockpile destruction process is highly unlikely, the route of exposure that civilian EMTs are recommended to be most familiar with is

   a. ingestion through contaminated food
   b. skin exposure through direct contact
   c. spill caused by accident while in transportation
   d. inhalation of contaminated air

Matching

Identify the probable cause of the following signs and symptoms by matching the Sign/Symptom in Column A with the correct Cause in Column B. Notice that the choices in Column B also include: either and neither. This means that—in addition to the choices (a) nerve agent or (b) blister agent - some of the signs/symptoms may be caused by both agents, or are not caused by either agent.

Column A **Signs/Symptoms**	**Column B** **Cause**
___ 10. miosis	a. nerve agent
___ 11. difficulty in breathing	b. blister agent
___ 12. increased oral and nasal secretions	c. either; may be caused by both nerve and blister agents
___ 13. sudden hair loss	d. neither; is not a sign/symptom of nerve nor blister agent
___ 14. twitching spasms	
___ 15. blisters	
___ 16. localized sweating	
___ 17. convulsions	
___ 18. temporary hearing loss	
___ 19. burning sensation in mucous membranes around eyes, in nose and mouth	
___ 20. giddiness	
___ 21. high blood pressure	
___ 22. skin rash	
___ 23. severe burn-like blisters	
___ 24. involuntary urination/defecation	

25. Reaction to a nerve or blister agent, and whether or not a particular sign/symptom shows up at all, depends on several factors which include:

    a. route of exposure; dose concentration; duration of exposure
    b. the duration of the exposure; the common name of the agent
    c. common name of the agent; altitude of the terrain at the time of exposure
    d. the duration of the exposure; whether the agent is classified as unitary or binary

26. The chief cause of death due to nerve agent exposure is:

    a. cerebral hemorrhage
    b. respiratory failure
    c. severe bilateral miosis
    d. severe gastroenteritis

27. After being exposed to a nerve agent, a victim will not necessarily display all of the signs/symptoms described in this student guide:

a. true
b. false

28. The FIRST step in providing treatment to a victim of nerve or blister agent exposure is:

a. protect yourself
b. stop the bleeding
c. decontaminate the victim
d. transport to the hospital

29. The FAST steps for nerve agent exposure patients must be done:

a. immediately
b. within 48 hours of exposure
c. by licensed EMTs under official supervision of the U.S. Army

30. The antidote for victims of nerve agent exposure consists of the following drug or drug combination:

a. atropine only
b. 2-PAM Cl only
c. both atropine and 2-PAM Cl
d. either atropine or 2-PAM Cl

31. The standard initial dose of atropine for adults is:

a. 0.5 mg
b. 1 mg
c. 2 mg
d. 4 gm

32. The best indicator used to ensure that adequate amounts of atropine are given is:

a. signs of atropinization
b. the disappearance of miosis
c. blood pressure of 110/90 or lower
d. the disappearance of the skin rash

33. In order to determine if additional atropine is needed after the initial dose, an adult exposed to nerve agent vapor and exhibiting severe signs and symptoms is reevaluated:

a. continually, every 30 minutes
b. continually, every 3-5 minutes
c. at the scene, before transport, at the receiving hospital
d. once at the scene and then again, if necessary, at the receiving hospital

34. The signs and symptoms of atropinization include:

a. dry secretions; miosis
b. dry secretions; easier breathing
c. blood pressure of 110/90 or lower; miosis
d. blood pressure of 110/90 or lower; easy breathing

Questions 36 and 37 pertain to First Aid and Special Treatment. Use the treatment choices below to answer each question.

Treatment Choices

1. Ensure that patient is decontaminated
2. Administer atropine/2-PAM chloride
3. Support airway management as necessary

35. The First Aid and Special Treatment protocol for a victim of a nerve agent is:

a. 1 and 2
b. 1 and 3
c. 1, 2, and 3
d. 1

36. The First Aid and Special Treatment protocol for a victim of a blister agent is:

a. 1 and 2
b. 1 and 3
c. 1, 2, and 3
d. 1

37. The most common mistake made when administering atropine is:

a. overdosing
b. underdosing
c. giving atropine when the victim's symptoms are very mild
d. giving atropine when there has been no nerve agent exposure

38. By far, the most common mistake when treating nerve agent patients is:

a. atropine underdosing
b. atropine overdosing
c. not giving atropine when it is needed
d. giving atropine without giving 2 PAM chloride

39. It is critical that decontamination be done quickly and correctly in order to:

a. minimize adverse effects to the exposed person and prevent secondary contamination
b. prevent the agent molecules from multiplying and causing uncontrolled, widespread injuries
c. keep the contamination centralized on one person instead of spreading to several others
d. reverse the effects of the agent and prevent secondary contamination

40. The EMT should ensure that decontamination occurs:

a. at back door of the ambulance
b. before patient is brought to initial treatment area
c. hospital
d. during transport to hospital

41. When treating a blister agent exposure patient, sterile precautions may be necessary because:

a. the fluid in the blisters can spread the agent
b. sterile technique accomplishes the same results as decontamination
c. damage done to the skin can permit infection if the blisters are severe
d. treatment provided later, at the hospital, requires that the blisters be as sterile as possible for testing

Short Answer

42-43. List 2 main FAST (first aid and special treatment) steps required for nerve agent exposure patients.

a. _____

b. _____

44-45. List 2 main FAST (first aid and special treatment) steps that may be necessary for blister agent exposure patients.

a. _____

b._____

46-47. List 2 specific signs that sufficient atropine has been administered.

a. _____

b. _____

APPENDIX C FINAL EXAM ANSWER KEY

1. c
2. a
3. d
4. d
5. d
6. b
7. a
8. d
9. d
10. a
11. c
12. a
13. d
14. a
15. b
16. a
17. a
18. d
19. b
20. a
21. d
22. b
23. b
24. a
25. a
26. b
27. a
28. a
29. a
30. c
31. c
32. a
33. b
34. b
35. c
36. b
37. b
38. c
39. a
40. b
41. c
42. (1) administer atropine pradidoxime chloride or (2) support airway management as necessary
43. (1) administer atropine/ pradidoxime chloride or (2) support airway management as necessary
44. (1) take sterile precautions or (2) support airway or (3) decontaminate patient management as necessary
45. (1) take sterile precautions or (2) support airway or (3) decontaminate patient management as necessary
46. (1) secretions are dry or (2) breathing is easier
47. (1) secretions are dry or (2) breathing is easier

APPENDIX D MATERIAL SAFETY DATA SHEETS (MSDS)

Contents

D.1 MSDS Nerve Agent GA (Tabun)
D.2 MSDS Nerve Agent GB (Sarin)
D.3 MSDS Nerve Agent VX
D.4 MSDS Blister Agent Mustard (H, HD)
D.5 MSDS Blister Agent Mustard (HT)

D.1 MSDS Nerve Agent GA (Tabun)

D.2 MSDS Nerve Agent GB (Sarin)

D.3 MSDS Nerve Agent VX

D.4 MSDS Blister Agent Mustard (H, HD)

D.5. MSDS Blister Agent Mustard (HT)