Chemical Terror Threats—Are You Prepared?

NYSNA Continuing Education

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How to Take This Course

Please take a look at the steps below; these will help you to progress through the course material, complete the course examination and receive your certificate of completion.

1. **REVIEW THE OBJECTIVES**

   The objectives provide an overview of the entire course and identify what information will be focused on. Objectives are stated in terms of what you, the learner, will know or be able to do upon successful completion of the course. They let you know what you should expect to learn by taking a particular course and can help focus your study.

2. **STUDY EACH SECTION IN ORDER**

   Keep your learning "programmed" by reviewing the materials in order. This will help you understand the sections that follow.

3. **COMPLETE THE COURSE EXAM**

   After studying the course, click on the "Course Exam" option located on the course navigation toolbar. Answer each question by clicking on the button corresponding to the correct answer. All questions must be answered before the test can be graded; there is only one correct answer per question. You may refer back to the course material by minimizing the course exam window.

4. **GRADE THE TEST**

   Next, click on "Submit Test." You will know immediately whether you passed or failed. If you do not successfully complete the exam on the first attempt, you may take the exam again. If you do not pass the exam on your second attempt, you will need to purchase the course again.

5. **FILL OUT THE EVALUATION FORM**

   Upon passing the course exam you will be prompted to complete a course evaluation. You will have access to the certificate of completion **after you complete the evaluation**. At this point, you should print the certificate and keep it for your records.

Introduction

For Americans, the world changed on September 11, 2001. Our sense of security was shaken. After the terrorist attacks in New York City, Washington and rural Pennsylvania we were further assaulted by the biological attack of anthrax. We wondered at that time, "Where will it all end?" "What will come next?" We found ourselves not entirely prepared for what might lay ahead.

Healthcare providers have taken these threats seriously; we have had to educate ourselves about biological, chemical and nuclear threats. Nurses have actively participated learning about these threats; they have been instrumental in emergency planning and treatment in federal and state government emergency preparedness plans. Nurses have signed up for New York State's Nurse Response Program (http://www.nysna.org/rn_response/nr.htm). Nurses have worked on the local county and facility level to combat these threats. Nurses have volunteered for smallpox vaccination and indeed some of our colleagues have succumbed to the adverse effects of the vaccine.

As of July, 2005 no further attacks have occurred, although our threat level has episodically been raised and terrorist attacks have occurred in other locations throughout the world. Despite the current relative calm in relation to terrorism in the US, experts agree that it is not a matter of "if," but of "when."

This course provides an overview of common chemical weapons that may be used as terror threats, how to recognize a chemical attack, what precautions to take to protect oneself, and how to manage and treat exposures among our patients.

Objectives

- Identify the categories of chemical agents that can be used as weapons of terror.
- Describe select chemical agents in each category.
- Discuss decontamination procedures.
- Identify how to protect oneself from the possibility of exposure to chemical agents.
- Discuss the health effects of select chemical agents.
- Describe treatment options for these chemical threats.
- Identify where to obtain further resource material on the subject of chemical terrorism.

About the Author

This course is primarily based on information provided by the Center for Disease Control and Prevention (CDC). Information was accessed from www.cdc.gov; content from the CDC is in the public domain and is not copyrighted. Specific CDC information that was used is listed in the Reference section of this course. Additional references were used in the development of this course and are referenced throughout the course as well as in the Reference section of the course.

The History of Chemical Weapon Use

While we might consider chemical weapons a product of the industrial and technological ages, they have been used during wartime for centuries. As early as 1000 BC, the Chinese used arsenical smokes against their enemies (Smart, 1997). During the Peloponnesian War in 429 and 424 BC, the Spartans used noxious smoke and flame against the Athenians. During the 30 years war toxic smoke projectiles were used. The British used picric-acid filled shells during the Boer War; the Japanese threw arsenal rag torches into Russian trenches during the Russo-Japanese war (Smart, 1997).

Debates about the moral use of chemical weapons during warfare were ongoing throughout the centuries; many proposals for chemical weapons were made, but many governments opposed their use. Military strategists might view chemical warfare agents as simply one of the means to

immobilize or destroy an enemy force; others may view such weapons as abhorrent extensions of conventional warfare (Takafuji & Kok, 1997).

It was during World War I that chemical weapons were used with regularity. The French used ethyl bromoacetate grenades against the Germans during their invasion of Belgium and France, apparently with little military benefit. In 1914 the Germans used respiratory irritant shells of dianisidine chlorosulfate against the British. Again, there was little military benefit, because when the shell exploded, the explosion destroyed the chemical effect of the weapon. Chlorine, a respiratory agent, was used against the Allies at Ypres, Belgium in 1915, with thousands of troops reportedly killed during the attacks. The Allies retaliated with further chemical agents (Smart, 1997). Throughout WWI, a variety of chemical agents were used, including mustard gas, chlorine, phosgene and cyanide.

Nerve agents and vesicants were used by the Iraqi government in the 1980s during the war with Iran; they also used it against their own Kurdish citizens in the town of Halabja in March of 1988. Sarin gas was used by the religious cult, Aum Shinrikyo in 1994 and 1995. During one of these occasions, the gas was used in the Tokyo subway (Smart, 1997). Currently the US military is searching for weapons of mass destruction, including chemical weapons, throughout Iraq.

Terrorist attacks, using chemical and other weapons, have increased throughout the world. Since 9/11/01, the US has been forced to deal with multiple threats; learning about chemical weapons and their effects helps healthcare providers to be prepared. Much of what is known about chemical agents is through experience with their use in wartime, manufacturing accidents, accidental releases such as when children find shells while playing and explosions occur, as well as with animal studies.

Readiness for the Possibility of Chemical Threats

While government agencies work toward the elimination of chemical terrorist threats, “being prepared” for healthcare providers, must focus on the health management of these threats. This requires gaining knowledge and competence in the recognition and treatment of chemical exposures, while safeguarding one’s own health.

The Centers for Disease Control and Prevention (CDC) has developed *Emergency Preparedness Competencies for all Public Health Workers*. While many in healthcare do not work in the public health field, these competencies have some application to other healthcare arenas. Included in the competencies is the ability to:

- Describe the agency chain of command in an emergency response;
- Identify and locate the agency emergency response plan, or in large agencies, the pertinent portion of the plan;
- Describe her/his functional role(s) and responsibilities in emergency responses;
- Demonstrate her/his role in regular emergency preparedness drills;
- Identify limits to her/his own knowledge, skills and authority;
- Identify key system resources for referring matters than exceed the above limits;
- Apply creative problem solving and flexible thinking to unusual challenges within her/his functional responsibilities;
- Evaluate effectiveness of all actions taken;
- Recognize deviations from the norm that might indicate an emergency;
- Describe appropriate action (e.g. communicate clearly within the chain of command).

Additionally, Public Health Professionals have the responsibility to be competent to:

- Demonstrate readiness to apply professional skills to a range of emergency situations during regular drills;

- Maintain regular communication with partner professionals in other agencies involved in emergency response;
- Participate in continuing education to maintain up-to-date knowledge in areas relevant to emergency response.

Effective January 1, 2003, The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires that a comprehensive, functional emergency preparedness plan be in place in healthcare organizations. This plan must include integration with community resources and the ability to be responsive to hazards. This plan must be developed, communicated and drilled at least twice per year.

According to the New York State Department of Health (NYSDOH) (August 2002), adequate planning and regular training are the keys to preparedness for terrorism-related events. Regularly updating one's information about chemical threats is an important aspect of readiness, as well as participating in local emergency preparedness drills, such as might be offered at a local health department, one's own workplace, or organizations such as The American Red Cross (www.redcross.org), the NYS Emergency Management Office (SEMO) (www.semo.state.ny.us/index.cfm) or the Federal Emergency Management Agency (FEMA) (www.fema.gov).

Healthcare providers should be alert to illness patterns and reports of chemical exposure that might signal an act of terrorism. Unlike biological terrorism threats, which may take weeks to detect, chemical weapons threats are likely to be rapidly apparent. For example, one category of chemical threats is nerve agents—their action occurs within minutes. Other chemical weapons threats may take hours to be detected.

NYSDOH (July, 2003) has identified the following clinical, epidemiological and circumstantial clues that may suggest a possible chemical terrorist event:

- An unusual increase in the number of people seeking care, especially with respiratory, neurological or gastrointestinal symptoms;
- Any clustering of symptoms or unusual age distribution (e.g., chemical exposure in children);
- Location of release not consistent with a chemical's use;
- Simultaneous impact to human, animal and plant populations;
- Any unusual clustering of patients in time or location (e.g., persons who attended the same public event).

NYSDOH recommends that any unusual symptoms, illnesses or clusters of these be reported immediately. EMS personnel should call their medical control facility and dispatching agency. The county health department and local Poison Control Center should also be notified (links are provided in the Resource section of this course).

The US Department of Homeland Security (DHS) has provided information that helps all in the population to be ready for an emergency situation. Readiness for a terrorist attack includes knowledge and preparation of:

- A kit of emergency supplies (this includes water and food, clean air, first aid kit, supply checklists, special needs items);
- Information on biological, chemical and radiation threats; and
- Making a plan (this includes a family plan, deciding whether to shelter in place or evacuate, being prepared at work or school, being prepared in a high-rise building and being prepared in a moving vehicle.

This information regarding general and specific preparedness activities can be accessed at: http://www.ready.gov/index.html.

In addition to the being professionally prepared for a chemical attack, healthcare providers also need to have information about how to manage in an emergency at home, in relation to their families and communities.

It is highly recommended that nurses prepare for a potential chemical emergency at home and for their families, as the role conflict (i.e. whether to go to work and help those who have been exposed, or stay home because of being unprepared and fearful about arrangements for family members and pets) could become a major issue and impact on the readiness of the entire healthcare system to care for and treat the large number of casualties that may be possible. Besides the preparation of the emergency supplies, planning for such an emergency is critical. Important to the understanding of emergency planning are the concepts of sheltering in place and evacuation. This information will be provided later in this course, however, the reader may choose to obtain this information directly from the CDC when preparing for their own home and family emergency response preparedness.

Information regarding sheltering in place can be accessed at http://www.bt.cdc.gov/planning/shelteringfacts.asp.

Information related to evacuation can be accessed at http://www.bt.cdc.gov/planning/evacuationfacts.asp.

Information about personal cleaning and disposal of contaminated clothing can be accessed at http://www.bt.cdc.gov/planning/personalcleaningfacts.asp.

What Happens After a Chemical Weapons Attack

In the event of a chemical weapons attack, certain public health and emergency preparedness actions will occur. An incident command center will be established; first responders, dressed in proper personal protective equipment, will enter the hot zone. The **hot zone** is the area where the chemical attack occurred and the area of greatest exposure to the chemical. First responders will evacuate those who have been exposed from the hot zone to the **decontamination zone**. Either a basic decontamination occurs, or one more specific, if the chemical agent is known. After decontamination, if needed, exposed persons may be taken to the emergency department of a local hospital. It is the function of public health officials, first responders and other emergency preparedness agencies to manage the initial attack scene. Much will occur prior to patients arriving in the emergency department of local hospitals or trauma centers.

Personal Protective Equipment

Vapors, gases, and particulates from hazardous substance response activities place response personnel at risk. For this reason, response personnel must wear appropriate personal protective clothing and equipment whenever they are near the site. The more that is known about the hazards at a release site, the easier it becomes to select personal protective equipment. There are basically four levels of personal protective equipment (EPA, 2004):

- Level A protection is required when the greatest potential for exposure to hazards exists, and when the greatest level of skin, respiratory, and eye protection is required. Examples of Level A clothing and equipment include positive-pressure, full face-piece self contained breathing apparatus (SCBA) or positive pressure supplied air respirator with escape SCBA, totally encapsulated chemical- and vapor-protective suit, inner and outer chemical-resistant gloves, and disposable protective suit, gloves, and boots.

- Level B protection is required under circumstances requiring the highest level of respiratory protection, with lesser level of skin protection. At most abandoned outdoor hazardous waste sites, ambient atmospheric vapors or gas levels have not approached sufficiently high concentrations to warrant level A protection -- Level B protection is often adequate. Examples of Level B protection include positive-pressure, full face-piece self

contained breathing apparatus (SCBA) or positive pressure supplied air respirator with escape SCBA, inner and outer chemical-resistant gloves, face shield, hooded chemical resistant clothing, coveralls, and outer chemical-resistant boots.

- Level C protection is required when the concentration and type of airborne substances is known and the criteria for using air purifying respirators is met. Typical Level C equipment includes full-face air purifying respirators, inner and outer chemical-resistant gloves, hard hat, escape mask, and disposable chemical-resistant outer boots. The difference between Level C and Level B protection is the type of equipment used to protect the respiratory system, assuming the same type of chemical-resistant clothing is used. The main criterion for Level C is that atmospheric concentrations and other selection criteria permit wearing an air-purifying respirator.

- Level D protection is the minimum protection required. Level D protection may be sufficient when no contaminants are present or work operations preclude splashes, immersion, or the potential for unexpected inhalation or contact with hazardous levels of chemicals. Appropriate Level D protective equipment may include gloves, coveralls, safety glasses, face shield, and chemical-resistant, steel-toe boots or shoes.

While these are general guidelines for typical equipment to be used in certain circumstances, other combinations of protective equipment may be more appropriate, depending upon specific site characteristics.

PPE to Prevent Inhalation Exposure

Protection from both vapors and particulates may be required when the chemical agent is being released. After release, protection from vapors is most important. Half-face and full-face respirators, with the appropriate canister, will provide good protection from vapors. These operate by negative pressure and must be fit tested for optimal protection. Powered, air-purifying respirators (PAPR) and self-contained breathing apparatus (SCBA) provide even greater protection and operate under positive pressure so that fit characteristics are less important. Surgical and N-95 masks will not protect against inhalation of vapors (NYSDOH, 2003).

Healthcare providers must obtain and utilize the appropriate respirator in order to maximize protection from exposure.

PPE to Prevent Dermal Exposure

Latex examination gloves provide very little protection from most chemical agents and can cause allergies. Gloves made of Viton, nitrile, butyl or neoprene provide more protection and, in some styles, allow adequate dexterity. However, the resistance of these materials to different chemicals varies and it is best to have a variety of gloves available. Double gloving may provide additional protection. Chemical-resistant aprons or suits can also prevent dermal exposure (NYSDOH, 2003).

PPE to Prevent Eye Exposure

Full-face respirators, PAPR and SCBA will provide protection from both splashes and vapors. Protective eyewear, such as goggles or a face shield, will not provide protection from chemical vapors. Protective eyewear is required during decontamination to prevent splashing into eyes (NYSDOH, 2003).

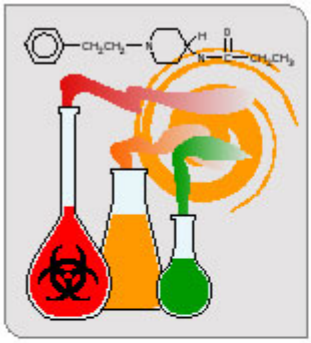
Although preventive measure such as PPE, sheltering in place (discussed later in this course) and other measures are used as means of avoiding exposure; sometimes the exposure cannot be prevented. In the event of exposure to chemical agents, immediate removal from the source and decontamination is highly correlated with better outcomes.

Don't Become a Casualty Yourself

It is absolutely critical that healthcare providers protect themselves from a direct attack, as well as through secondary exposure from exposed patients. Healthcare providers must safeguard against becoming patients themselves. Then there would be one less healthcare provider to care and treat exposed persons, and the healthcare provider would themselves add to the pool of patients. Although first responders face the greatest exposure potential, often to unidentified agents, healthcare providers are among the first to respond to an emergency. In order to protect yourself:

- Be alert
- Keep an appropriate distance
- Stay upwind
- Wait for assessment by a hazardous materials (HAZMAT) team before entering the release area

The DHS also has information relevant in the event of potential chemical attacks. It can be downloaded from http://ready.gov/readygov_chemical.pdf:



1. A chemical attack is the deliberate release of a toxic gas, liquid or solid that can poison people and the environment.

2. Watch for signs such as many people suffering from watery eyes, twitching, choking, having trouble breathing or losing coordination.

3. Many sick or dead birds, fish or small animals are also cause for suspicion.

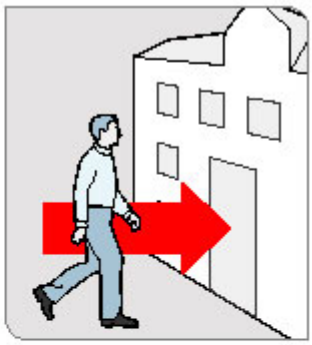
4. If you see signs of a chemical attack, quickly try to define the impacted area or where the chemical is coming from, if possible.

5. Take immediate action to get away from any sign of a chemical attack.

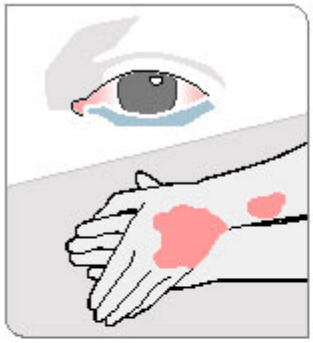
6. If the chemical is inside a building where you are, try to get out of the building without passing through the contaminated area, if possible.

7. Otherwise, it may be better to move as far away from where you suspect the chemical release is and "shelter-in-place."

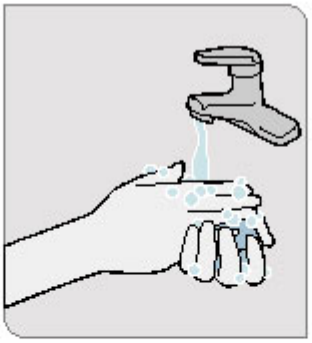
8. If you are outside when you see signs of a chemical attack, you must quickly decide the fastest way to get away from the chemical threat.



9. Consider if you can get out of the area or if it would be better to go inside a building and follow your plan to "shelter-in-place."



10. If your eyes are watering, your skin is stinging, you are having trouble breathing or you simply think you may have been exposed to a chemical, immediately strip and wash. Look for a hose, fountain, or any source of water.



11. Wash with soap and water, if possible, but do not scrub the chemical into your skin.

12. Seek emergency medical attention.

General principles of triage for chemical exposures (ATSDR, 2001)

- Check triage tag/card for any previous treatment or triage.
- Survey for evidence of associated traumatic/blast injuries.
- Observe for sweating, labored breathing, coughing/vomiting, and secretions.
- Severe casualty triaged as immediate if assisted breathing is required.
- Blast injuries or other trauma, where there is question whether there is chemical exposure, victims must be tagged as immediate in most cases. Blast victims evidence delayed effects.
- Mild/moderate casualty: self/buddy aid, triaged as delayed or minimal, and release is based on strict follow up and instructions.
- If there are chemical exposure situations that may cause delayed but serious signs and symptoms, then triage to the proper facilities that can observe and manage any delayed onset symptoms is considered appropriate.
- Expectant categories in multi-casualty events are those victims who have experienced a cardiac arrest, respiratory arrest, or continued seizures immediately. Resources should not be expended on these casualties if there are large numbers of casualties requiring care and transport with minimal or scant resources available.

Triage Categories

1. *Immediate*: casualties who require lifesaving care within a short time, when that care is available and of short duration. This care may be a procedure that can be done within minutes at an emergency treatment station (e.g., relief of an airway obstruction, administering antidotes) or may be acute lifesaving surgery.

2. *Delayed*: casualties with severe injuries who are in need of major or prolonged surgery or other care and who will require hospitalization, but delay of this care will not adversely affect the outcome of the injury (e.g., fixation of a stable fracture).

3. *Minimal*: casualties who have minor injuries, can be helped by nonphysician medical personnel, and will not require hospitalization.

4. *Expectant*: casualties with severe life-threatening injuries who would not survive with optimal medical care, or casualties whose injuries are so severe that their chance of survival does not justify expenditure of limited resources. As circumstances permit, casualties in this category may be reexamined and possibly be retriaged to a higher category.

General Decontamination Guidelines

Immediately after exposure to a chemical weapon, decontamination is necessary. Depending on the agent, the amount of agent released and the method of release, decontamination may be the only needed intervention.

Ideally, knowing which chemical agent was released allows for the best decontamination. However, most often this information will not necessarily be immediately available. General guidelines are provided to offer information on how to proceed even when the specific agent has not been identified.

Proper decontamination is often the most important first step in treating a patient exposed to chemical agents. Immediate removal of patient clothing can remove up to 90 percent of the contaminant. Removed clothing should be double bagged, sealed (NYSDOH, 2003) and retained as possible evidence.

After the clothing is removed, the patient's skin and eyes may need to be decontaminated. In most cases, decontamination of skin can be accomplished by gentle and thorough washing with soap and water followed by a thorough water rinse. For eyes, flush with plenty of water or normal saline. Decontamination water may need to be contained, as it is likely contaminated with the chemical (NYSDOH, 2003).

Bleach solutions, concentrated or dilute, should not be used on people. Diluted bleach (1 part household bleach to 9 parts water) can be used on equipment and other hard surfaces. Because bleach solutions irritate the eyes, skin and respiratory tract, they must be handled with caution and used with adequate ventilation (NYSDOH, 2003).

It is important not to abrade the skin during washing or rinsing. This is especially true after exposure to blistering/vesicant agents which bind to skin. These agents may leave the skin compromised and susceptible to further damage. For choking/pulmonary-damaging agents or incapacitating/behavior-altering agents, a rinse in water alone may be adequate (NYSDOH, 2003).

Some chemical agents are accompanied by a characteristic odor that may provide a warning. However, after a while, people may become used to the chemical and no longer detect the smell. The chemical may still be present even if there is no detectable odor.

Categories of Chemical Weapons Threats

Chemical weapons threats have been divided into multiple categories, based on the health effects of the chemical agents. The Centers for Disease Control and Prevention (CDC) divides chemical weapon agents in the following categories: nerve agents, vesicants or blister agents, pulmonary agents, blood agents and incapacitating agents.

In 2005, the CDC (2005a) held a web broadcast on the issue of foodborne chemical threats. Some of the same agents as listed in the above categories can also be used in foodborne contamination, however other agents can also cause significant illness using the foodborne route.

Nerve Agents

Nerve agents are the most toxic and rapidly acting of the known chemical warfare agents. They were originally developed as pesticides in Germany in 1936. Although nerve agents are chemically similar to organophosphate pesticides in method of action and harmful effects, they are significantly more potent.

Nerve agents cause their toxic effects by inhibiting acetylcholinesterase enzymes, which then cannot hydrolyze acetylcholine. Historically, this has been considered to be the explanation for the toxic effects of nerve agents, however, they also inhibit other enzymes, contributing to toxic effects. Unlike some organophosphate pesticides, nerve agents have not been associated with neurological problems lasting more than 1 to 2 weeks after the exposure. Nerve agents are readily absorbed through inhalation, ingestion and dermal contact. Rapidly fatal systemic effects can occur (Siddell, 1997).

While there are differences among the various nerve agents, the health effects, the decontamination and the treatment of the various nerve agents are similar, regardless of the specific agent. Descriptions of two specific agents appear first, and then the commonalities are reviewed.

<u>Sarin</u>

Sarin (isopropyl methylphosphonofluoridate), is also known as GB. It was originally was developed in 1938 in Germany as a pesticide. It was further developed as a chemical weapon by the Germans during World War II, but was never used. As previously mentioned, sarin was used as a chemical warfare weapon by Iraq during the Iran-Iraq War in the 1980s and it was also used in two terrorist attacks in Japan in 1994 and 1995 (Siddell, 1997).

Sarin is a clear, colorless, and tasteless liquid that has no odor in its pure form. It can be weaponized in both liquid and gas forms. Following release of sarin into the air, exposure occurs through skin or eye contact or through inhalation. The clothing of an exposed victim can release sarin for about 30 minutes after it has come in contact with sarin vapor, which can lead to further exposure of others (Siddell, 1997).

Sarin is the most volatile of the nerve agents, which means that it can easily and quickly evaporate from a liquid into a vapor and spread into the environment. People can be exposed to the vapor even if they do not come in contact with the liquid form of sarin. Sarin vapor is heavier than air; it will sink to low-lying areas and create a greater exposure hazard there. Because it evaporates so quickly, sarin presents an immediate but short-lived threat.

Sarin can be used to contaminate water or food; it mixes easily with water. Exposure occurs through dermal contact or ingestion.

VX

VX was developed in the early 1950s by British scientists; it is not found naturally in the environment. VX is a clear, amber colored, odorless, oily liquid. Following the release of VX into the air, exposure occurs through dermal contact, inhalation of VX mist and eye contact. VX is primarily a liquid exposure hazard, but if it is heated to very high temperatures, it can turn into small amounts of vapor.

While VX does not combine with water as easily as do the other nerve agents, it can still be used to contaminate water. It can also be used to contaminate food. Exposure after contamination of water or food with VX occurs through dermal contact or ingestion.

Clothing contaminated with VX can release the nerve agent for approximately 30 minutes after contact with the VX vapor. This can lead to secondary exposure to others. VX breaks down slowly in the body, allowing repeated exposures to VX and/or other nerve agents to have a cumulative effect.

VX is the most potent of all nerve agents. Compared with the nerve agent sarin, VX is considered to be much more toxic by entry through the skin and somewhat more toxic by inhalation. It is possible that any visible VX liquid contact on the skin, unless washed off immediately, would be lethal.

VX is the least volatile of the nerve agents, which means that it is the slowest to evaporate from a liquid into a vapor. It evaporates slowly, at about the same rate as motor oil. Therefore, VX is very persistent in the environment. Under average weather conditions, VX can last for days on surfaces with which it has come in contact. Under very cold conditions, VX can last for months. Because VX vapor is heavier than air, it will sink to low-lying areas and create a greater exposure hazard there. Its slow evaporation time makes VX a long-term, as well as short-term threat. The former is particularly true when surfaces are contaminated with VX.

Adverse Health Effects of Nerve Agents

Inhalation of nerve agent inhibits blood cholinesterase enzyme activity and produces signs and symptoms of exposure much more quickly than does dermal contact (Siddell, 1997). The effects of nerve agent vapor can be seen in seconds to minutes after exposure, depending on the concentration of vapor. Maximal severity is usually reached within minutes after exposure. There is no delay in onset, as there is with liquid nerve agent.

At low concentrations of nerve agent vapor, generally, the eyes and nose are affected first; the eyes can be affected equally or unequally. There may be some miosis, eye pain or conjunctival injection. Rhinorrhea is likely. As exposure increases the classic triad of eyes, nose and lung involvement occurs. Persons may complain of decreased vision, or tightness in the chest. The chest tightness may be a complaint even when there are no physical findings. At higher exposures, marked miosis, copious secretions from the eyes, nose and mouth as well as increasing respiratory distress occurs (Siddell, 1997).

According to the US Army Soldier Chemical and Command Center (March, 2002) the immediate signs and symptoms of nerve agent exposure are copious secretions. A mnemonic "**SLUDGE"** can be used to remember the specific secretions, which can help in the identification of which category of chemical agent a victim was exposed to: **S**alivation, **L**acrimation, **U**rination, **D**efecation, **G**astric disturbances, **E**mesis.

In severe exposures to nerve agent vapor, loss of consciousness may occur within the minute, convulsive jerking movements of the limbs, copious secretions from the mouth and nose, very labored breathing, generalized muscle fasciculations and miosis. Fasciculations are the visible contractions of a small number of fibers innervated by a single motor nerve filament. They appear as ripples under the skin. Flaccid paralysis, apnea and death due to respiratory failure can occur (Siddell, 1997).

The initial effects of liquid nerve agent on the skin depends on the amount of agent, the site on the body, as well as air temperature and humidity. Generally there is a delay, in which the exposed person is asymptomatic. Sometimes the delay can be as long as several hours between the time of exposure and the appearance of any signs or symptoms. At times symptoms occur even when the individual has been decontaminated. Initially, the exposed individual experiences sweating at the site and less commonly, localized fasciculations of the underlying muscle. Gastrointestinal symptoms generally occur next, including nausea, vomiting and diarrhea. The exposed person also may exhibit generalized sweating and complain of fatigue or feeling ill (Siddell, 1997). Even a small drop of nerve agent on the skin can cause sweating and fasciculations where chemical touched the skin.

After large exposure, the amount of time of any delay between exposure and the occurrence of symptoms is significantly reduced.

Additionally, behavior changes have been noted with exposure to nerve agents. The effects can begin as late as one day after exposure, but generally occur within a few hours and last from several days to several weeks. Behavioral experiences include: feelings of uneasiness, tension, fatigue, forgetfulness, irritability, cognitive slowing such as not answering questions as quickly or precisely as previously, impaired judgment, poor comprehension, decreased ability to communicate and mild confusion.

Symptoms of Nerve Agent Exposure
○ Copious secretions: runny nose, watery eyes, drooling, sweating ○ Miosis, eye pain, blurred vision ○ Muscle twitching ○ Cough ○ Chest tightness ○ Rapid or slowed heart rate ○ Hypotension or hypertension

- Rapid respirations
- Nausea, vomiting, diarrhea
- Abdominal pain
- Polyuria
- Confusion
- Drowsiness
- Weakness
- Headache
- Loss of consciousness
- Convulsions
- Paralysis
- Respiratory failure
- Death

Management of Exposure/Decontamination

A critical aspect of the management of exposure and decontamination is the protection of the healthcare provider. If the caregiver is exposed to the chemical he or she can quickly become a casualty. Caregivers can be protected by the use of personal protective equipment (PPE) or through the thorough decontamination of those exposed to the chemical. This is true of any toxic substance exposure management.

If nerve agent vapor is the source of exposure, utilizing a protective mask and then removing the exposed person from the area of nerve agent release may be the most critical intervention.

Decontamination is important in order to prevent the further absorption of the agent by the skin of the exposed person, to prevent the further spread of the agent on the exposed person, to prevent the spread of the agent to others, including healthcare providers, who must handle or come into contact with the exposed person (Siddell, 1997).

To successfully reduce the damage of the nerve agent, decontamination must begin as soon as possible, but certainly within minutes of exposure. This means that in order for decontamination to be effective, anyone who may potentially be exposed to chemical threats should use the process of self-decontamination. Patients who are able and cooperative may assist with their own decontamination. Remove and double bag contaminated clothing and all personal belongings.

For patients exposed to nerve agent vapor only, remove outer clothing and wash exposed areas including the head and hair with soap and water. For patients exposed to liquid agent, remove all clothing and wash entire body and hair with soap and water or 0.5% hypochlorite followed by a water rinse.

See the Basic Decontamination procedure covered earlier in this course. Irrigate exposed eyes with plain water or saline for about 5 to 10 minutes. Remove contact lenses if present and easily removable without additional trauma to the eye.

Leave the area where the nerve agent was released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing the possibility of death from

exposure to nerve agent vapor. If the nerve agent release was outdoors, move away from the area where the release occurred. Go to the highest ground possible, because nerve agents are heavier than air and will sink to low-lying areas. If the nerve agent release was indoors, get out of the building. Sometimes the room or building can be sealed off.

Evacuation of an exposed area may be necessary; however, emergency coordinators may direct the population to "shelter in place" inside a building to avoid being exposed to the chemical. Both evacuation and sheltering in place will be covered later in this course.

If exposure was only to the nerve agent vapor, skin decontamination is not necessary (Siddell, 1997). Liquid agent on the skin should be physically removed, detoxified by chemical degradation and neutralization.

Exposed persons should remove any clothing that was exposed to nerve agents in liquid form. Clothing should be cut off the body instead of pulled over the head. Seal the clothing in a plastic bag, double bagging whenever possible. Removing and sealing the clothing in this way will help protect others from further exposure to any chemicals that might be on the clothing. When helping others to remove their clothing, avoid touching any contaminated areas, and remove the clothing as quickly as possible.

Decontamination of the skin must occur after the clothing is removed. Neutralizing agents include: dilute hydroxide, household bleach, and water. When used in large amounts, water is effective in diluting chemical agents and physically removing them from the exposed person's skin. As quickly as possible, wash any liquid nerve agent from the skin with large amounts of water. Rinse the eyes with plain water for 10 to 15 minutes if they are burning or if vision is blurred. If nerve agent has been swallowed, **do not induce vomiting or give fluids to drink**. Treatment is needed immediately (See Treatment of Nerve Agent Exposure below).

According to the Army Soldier Biological and Chemical Command (2002), any surfaces exposed to sarin should be flushed with large amounts of a 5% bleach and water solution in order to decontaminate objects.

Treatment of Exposure to Nerve Agents

Treatment of exposure to nerve agents is similar to that of any toxic substance exposure: termination of exposure, maintaining ventilation, administering an antidote if one exists, and correct any cardiovascular abnormalities. Treatment must begin immediately for the best outcomes.

First of all, exposure to the nerve agent must be terminated as soon as possible.

Exposure to mild or moderate amounts of nerve agent can result in complete recovery, although those with severe exposure are not likely to survive. Unlike some organophosphate pesticides, nerve agents have not been associated with neurological problems lasting more than 1 to 2 weeks after the exposure. Recovery from nerve agent exposure is possible with treatment; the antidotes available must be used quickly to be effective.

For persons with mild exposures who have been through the decontamination process often can return to baseline within 20 minutes, despite having experienced severe dyspnea immediately after exposure (Siddell, 1997).

If severe respiratory distress occurs, the standard antidote, since the 1940s, has been atropine. Atropine interferes with receptor binding of acetylcholine at muscarinic receptor sites. Pralidoxime chloride (2-Pam) is an oxime that has been approved by the Food and Drug Administration (FDA) for use as an antidote to nerve agent exposure. According to the NYSDOH (2003), nerve agent antidotes can be obtained as auto-injector syringes which can be rapidly delivered, typically into the thigh or buttocks. Atropine in auto-injector form, is available as the AtroPen in amounts 0.5mg, 1mg or 2mg. 2-PAM chloride, in auto-injector form, is available as

the 600 mg ComboPen. A Mark 1 kit contains 2 auto-injector syringes; the smaller one with 2mg atropine and the larger one with 600 mg 2-PAM chloride (NYSDOH, 2003).

The nurse should be careful during administration, as the spring-loaded design of the auto-injectors provides a forceful delivery that can cause tissue damage, especially to children and smaller patients. Children weighing less than 15 lbs (approximately 7 kg), generally those who are younger than 6 months of age, should not ordinarily be treated with the nerve agent antidote auto-injectors. For those patients, atropine should be individualized at doses of 0.05mg/kg (NYSDOH, 2003).

Suctioning due to secretions and assisted ventilation may be needed. The use of atropine to dry the thinner secretions may contribute to thick mucoid plug formation, which must be removed prior to assisted ventilation. Diazepam is used to treat convulsions and other muscular adverse reactions to nerve agents.

TABLE 1: ANTIDOTE RECOMMENDATIONS FOLLOWING EXPOSURE TO NERVE AGENTS

NYSDOH RAPID RESPONSE CARDS

Patient Age	Antidotes		Other Treatment
	Mild/Moderate Effects[1]	Severe Effects[2]	
			Assisted ventilation after antidotes for severe exposure.
Child	Atropine: 0.05 mg/kg IM **or** IV (minimum 0.1 mg, maximum 5 mg); **and** 2-PAM chloride: 25 mg/kg IM or IV (maximum 2 g IM, or 1 g IV)	Atropine: 0.1 mg/kg IM **or** IV (minimum 0.1 mg, maximum 5 mg); **and** 2-PAM chloride: 50 mg/kg IM or IV (maximum 2 g IM, or 1 g IV)	**Repeat atropine** at 2-5 minute intervals until secretions have diminished and breathing is comfortable or airway resistance has returned to near normal. **Repeat 2-PAM chloride** once at 30-60 minutes, then at 1 hour intervals for 1-2 doses as necessary. **Diazepam** for seizures: Child: 0.05 to 0.3 mg/kg IV (maximum 10 mg); Adults: 5 mg IV **Other benzodiazepines (eg. lorazepam, midazolam)** may provide relief. **Phentolamine** for 2-PAM chloride-induced hypertension: Child: I mg IV Adult: 5 mg IV
Adult	Atropine: 2 to 4 mg IM **or** IV; **and** 2-PAM chloride[3]: 600 mg IM, **or** 25 mg/kg IV slowly	Atropine: 6 mg IM; **and** 2-PAM chloride[3]: 1,800 mg IM, **or** 50 mg/kg IV slowly	

1. **Mild/Moderate effects of nerve agents** include localized sweating, muscle fasciculations, nausea, vomiting, weakness, dyspnea.
2. **Severe effects of nerve agents** include unconsciousness, seizure, apnea, flaccid paralysis.
3. Dose selection of 2-PAM chloride for elderly patients should be cautious (usually starting at 600 mg IM or 25 mg IV slowly) to account for the generally decreased organ functions of this population.

NOTE: 2-PAM chloride is pralidoxime chloride or Protopam Chloride.

Routine laboratory studies for all admitted patients include complete blood count (CBC), glucose, and serum electrolyte determinations. Chest X-ray and pulse oximetry (or arterial blood gas [ABG] measurements) are recommended for severe exposures. Symptomatic and asymptomatic patients suspected of significant exposure should have determinations of red blood cell (RBC) cholinesterase activity, the most useful test for nerve agent poisoning. Severe symptoms of toxicity are usually present when more than 70% of RBC cholinesterase is inhibited. However, there is no correlation between cholinesterase activity and severity of topical signs and symptoms

(e.g., miosis, rhinorrhea, dyspnea). If this test is not available, plasma cholinesterase can be measured (ATSDR, 2001).

Patients exposed to nerve agent vapor who have experienced only miosis and/or mild rhinorrhea when they reach the medical facility do not need to be admitted. All other patients who have had exposure to nerve agent should be hospitalized and observed closely (ATSDR, 2001).

Patients who have severe exposure should be evaluated for persistent central nervous system (CNS) sequelae. Patients should be advised to avoid organophosphate insecticide exposure until sequential RBC cholinesterase activity (measured at weekly to monthly intervals) has stabilized in the normal range, a process that may take 3 to 4 months after severe poisoning (ATSDR, 2001).

A patient information sheet on nerve agents appears in Appendix 1.

Vesicants/Blister Agents

Blister agents are also called vesicants, because they cause vesicles or blistering of the skin and mucous membranes on contact. The most common clinical effects after exposure to vesicants include dermal (skin erythema and blistering), respiratory (cough, dyspnea, pneumonitis, and acute lung injury), ocular (conjunctivitis and burns), and gastrointestinal (vomiting) signs and symptoms. The effects of the majority of vesicants manifest rapidly (within minutes). However, clinical findings might be delayed for hours after exposure (e.g., sulfur mustard).

A number of specific agents will be discussed: mustard, of which there are two types, sulfur mustard and the nitrogen mustards; arsenicals, of which lewisite is an example that will be covered, and phosgene oxime.

Sulfur Mustard

Sulfur mustard is also known as “mustard gas” or “mustard agent,” or by the military designations H, HD, and HT. First developed in the early to mid-1800s, sulfur mustard was introduced as a chemical warfare/terror agent in 1917 during World War I. Mustard has been used extensively as a chemical warfare/terror agent. During World War I, it caused more casualties than all the other chemical agents combined. Iraq used sulfur mustard against Iran during the 1980s.

Sulfur mustard is not found naturally in the environment. In the past it was used as a treatment for psoriasis; currently it has no clinical use. Sulfur mustard is still considered to be a major chemical weapons threat, particularly because after 70 years of use, there is still no antidote.

Description

Sulfur mustard sometimes smells like garlic, onions, or mustard, but sometimes has no odor. It can be a vapor, an oily-textured liquid, or a solid. Sulfur mustard can be clear to yellow or brown when it is in liquid or solid form. Allegedly the name of the chemical agent was derived from either its smell or color (Siddell, et al., 1997).

Routes of Exposure

Sulfur mustard may not initially be detected because it often has no smell or has a smell that might not cause alarm.

Sulfur mustard can be released into the air or into water supplies; exposure occurs through inhalation, dermal contact and ingestion. Sulfur mustard vapor can be carried long distances by wind currents. It survives from 1-2 days in the environment under average conditions and from weeks to months under very cold conditions. Sulfur mustard vapor is heavier than air, so it will settle in low-lying areas.

Sulfur mustard does not vaporize at low temperatures, because of its very high freezing temperature of 57 degrees. This makes it unsuitable for dispersal during winter months or

through aerial spraying. It must be mixed with another chemical, often with Lewisite (see next section) in order to increase volatility in colder weather (Siddell et al., 1997). In warm temperatures, mustard is 7 to 87-fold more persistent than Lewisite, making it a useful agent in warm climates. Additionally, if sulfur mustard is released during a fairly cool nighttime, its effects would not be likely until the following day's heat released the mustard vapors. Reportedly this is how many combat troops were exposed and injured during Desert Storm.

Identification of Sulfur Mustard as a Chemical Weapon Agent

While sometimes the chemical agent may not be identified at the release or exposure site, sulfur mustard is relatively easy to identify clinically. The distinguishing characteristic is the onset of effect. If several hours have passed prior to the exhibition of the symptoms below, the agent can be reasonably identified as sulfur mustard. By comparison, Lewisite, which is described later in this course, is a vesicant that causes immediate pain after exposure (Siddell et al., 1997).

Adverse Health Effects of Sulfur Mustard

Sulfur mustard is a powerful irritant and blistering agent that damages the skin, eyes, and respiratory tract; cellular changes occur within minutes of exposure, but the onset of pain or other symptoms may be delayed for 1 to 24 hours. Because of its potent alkalizing properties, it may cause bone marrow suppression as well as neurological and gastrointestinal toxicity (ATSDR, 2001).

Sulfur mustards are vesicants and alkylating agents; however, the biochemical mechanisms of action are not clearly understood. They are highly reactive and combine rapidly with proteins, DNA, or other molecules. Therefore, within minutes following exposure intact mustard or its reactive metabolites are not found in tissue or biological fluids. Sulfur mustards also have cholinergic activity, stimulating both muscarinic and nicotinic receptors. The onset of clinical symptoms and their time of onset depend on the severity of exposure (ATSDR, 2001).

It breaks down slowly in the body so repeated exposure may have a cumulative effect. Adverse health effects caused by sulfur mustard depend on the amount of exposure, the route of exposure, and the length of time of exposure.

Dermal contact of approximately 1-1and ½ tsp of liquid sulfur mustard is lethal to 50% of the population (Siddell et al., 1997).

Exposure to sulfur mustard is usually not fatal. When sulfur mustard was used during World War I, it killed fewer than 5% of the people who were exposed and who received medical care. Death occurs from the 5^{th} to 10^{th} day after exposure and is typically from respiratory insufficiency complicated by infection due to immune system compromise.

Typically, signs and symptoms do not occur immediately. Depending on the severity of the exposure, symptoms may not occur for 2 to 24 hours. Some people are more sensitive to sulfur mustard than are other people, and may have symptoms sooner.

Table 2 provides the symptoms of mild, moderate and severe exposure to the eyes, the respiratory system and skin, as well as the onset of symptoms (Siddell et al., 1997).

Table 2. Initial Clinical Effects of Sulfur Mustard Exposure

ORGAN	SEVERITY	EFFECTS	ONSET OF 1ST EFFECTS
Eyes	Mild	Tearing	4 – 12 hours
		Itching	
		Burning	
		Gritty feelings	
	Moderate	Above effects, plus:	3-6 hours
		Reddening	
		Lid edema	
		Moderate pain	
	Severe	Marked lid edema	1-2 hours
		Corneal damage	
		Severe pain	
Airway	Mild	Rhinorrhea	6-24 hours
		Sneezing	
		Epistaxis	
		Hoarseness	
		Hacking cough	
	Severe	Above effects, plus:	2-6 hours
		Productive cough	
		Mild to severe dyspnea	
Skin	Mild	Erythema	2-24 hours
	Severe	Vesication	

According to the Agency for Toxic Substance and Disease Registry (ATSDR) (2001). Sulfur mustard can have the following effects on specific parts of the body:

- *Skin*: Direct exposure causes erythema and blistering. It begins with a pruritic rash generally within 4 to 8 hours followed by blistering, 2-18 hours later. Blisters begin around the periphery of the erythemous area, resembling a "string of pearls," which eventually merge. Contact with sulfur mustard vapor causes first and second degree burns while exposure to sulfur mustard liquid typically causes second and third degree chemical burns. Exposure covering over 25% of the body surface area can be fatal.

- *Eyes*: The eyes are the most sensitive part of the body to the effects of sulfur mustard. It can cause intense conjunctival and scleral pain, swelling, lacrimation, blepharospasm, and photophobia. Symptoms may be delayed depending on the amount of exposure. A severe exposure may cause symptoms within 1 to 2 hours and may include the above symptoms as well as miosis, corneal edema, perforation, blindness and scarring (ATSDR, 2001). Photophobia may appear and even with mild exposure may last for weeks (Siddell et al., 1997).

- *Respiratory tract*: Dose dependent inflammatory reactions in the upper and lower airway develops several hours after exposure and progresses over several days. Burning nasal pain, epistaxis, sinus pain, laryngitis, loss of taste and smell, cough, wheezing and dyspnea. Necrosis of respiratory epithelium can lead to local airway obstruction (ATSDR, 2001).

- *Digestive tract*: Ingestion may cause chemical burns in the GI tract. Nausea and vomiting can occur after ingestion or inhalation. When nausea, vomiting and diarrhea occurs early after exposure, it is usually transient and not severe; however if it occurs several days after exposure, it is indicative of significant GI damage and has a poor prognosis.

- *Central nervous system*: High doses of sulfur mustard can cause hyperexcitability, insomnia and convulsions.

- *Hematopoietic:* Systemic absorption of sulfur mustard leads to bone marrow suppression, increased risk for fatal infections, hemorrhage and anemia.

- *Delayed effects/sequelae:* Relapsing keratitis or keratopathy may develop years after initial healing (ATSDR, 2001). Persistent eye conditions, loss of smell or taste, asthmatic bronchitis, recurrent respiratory infections, and lung fibrosis can develop.

Exposure to sulfur mustard liquid is more likely to produce second and third degree burns and later scarring than is exposure to sulfur mustard vapor. Extensive skin burning can be fatal (Siddell et al., 1997; ATSDR, 2002). Long-term effects can be areas of hypopigmentation on the skin, as well as areas of hyperpigmentation.

Extensive breathing in of the vapors can cause chronic respiratory disease, repeated respiratory infections, or death. Extensive eye exposure can cause permanent blindness. Exposure to sulfur mustard may increase a person's risk for lung and respiratory cancer.

Those exposed will need treatment for hours or days after the health effects begin. Skin lesions may take the longest to heal, oftentimes requiring months of hospitalization.

Death occurs most often from inhalation of sulfur mustard which causes massive pulmonary damage and secondary infection such as bronchopneumonia or sepsis resulting from loss of immune function. Death that is a result of liquid exposure is not as well understood, however sulfur mustard damage appears similar to radiation damage on autopsy. Sulfur mustard is sometimes considered to be a radiomimetic agent because of this effect (Siddell et al., 1997).

Long-term chronic bronchitis can result when there is a history of cardiopulmonary disorders, severe or inadequately treated bronchitis or pneumonitis, prior history of smoking, and advanced age.

Additional long-term effects that have been associated with sulfur mustard exposure include: cancer and other chronic respiratory diseases, skin cancer, chronic eye conditions, psychological conditions and sexual dysfunction.

Management of Exposure and Decontamination

Because no antidote exists for sulfur mustard exposure, the best thing to do is avoid it. Immediately leave the area where the sulfur mustard was released. Try to find higher ground, because sulfur mustard is heavier than air and will settle in low-lying areas.

If the release was out of doors, and it is not safe to remain outdoors, shelter inside a safe location. Evacuation may be necessary.

If avoiding sulfur mustard exposure is not possible, rapidly remove the sulfur mustard from the body. Getting the sulfur mustard off as soon as possible after exposure is the only effective way to prevent or decrease tissue damage to the body. This decontamination process is not likely to be done in a medical facility but should be done within 1-2 minutes after exposure. Sometimes a person may not know that he or she is contaminated with sulfur mustard until hours later when symptoms develop, at that point absorption into the skin has already occurred and tissue damage is underway. However, decontamination at that point may help to further spread the exposure, to the affected individual and others, particularly to healthcare providers. Victims whose skin or clothing is contaminated with liquid sulfur mustard can contaminate rescuers by direct contact or through off-gassing vapor.

Quickly remove any clothing that has liquid sulfur mustard on it. Seal the clothing in a plastic bag, double bagging whenever possible.

Immediately flush any exposed part of the body (eyes, skin, etc.) thoroughly with plain, clean water. Take care not to rub the skin. Eyes need to be flushed with water for 5 to 10 minutes. Do NOT cover eyes with bandages, but do protect them with dark glasses or goggles.

If someone has ingested sulfur mustard, **do not induce vomiting**. Give the person milk to drink. Immediate medical attention is needed.

Treatment of Exposure

The most important factor is removing sulfur mustard from the body. Exposure to sulfur mustard is treated by giving the victim supportive medical care to minimize the effects of the exposure. Although no antidote exists for sulfur mustard, exposure is usually not fatal.

Sodium thiosulfate given IV within minutes after exposure may prevent lethality (ATSDR, 2001).

Pain control is important in sulfur mustard exposure; fluid and electrolytes must be carefully monitored. Parenteral fluids and vitamin supplements may be of benefit, especially if the exposed person is dehydrated or too ill to eat or drink fluids.

Small blisters should be kept clean to avoid infection. Daily irrigation and antibiotic ointment is suggested (Siddell et al., 1997). Large blisters should be “unroofed” and irrigated with normal saline, sterile water, clean soapy water and then covered liberally with an antibiotic ointment. Although the pathophysiology is different, the treatment of mustard skin lesions is the same as the treatment for second degree thermal burns. Systemic analgesics should be given liberally, particularly prior to manipulation of the chemically burned skin. Fluid loss is not as great with mustard burns as with thermal burns and healthcare providers experienced in the treatment of thermal burns must use caution not to over hydrate these patients. Skin grafting is rarely needed (Siddell et al., 1997).

Adequate oxygenation needs to be maintained in persons who have respiratory exposure to sulfur mustard. Treatment can include: bronchodilaters, oxygen supplementation, antitussives, steroids and steam inhalation treatments, and ventilatory support. Intubation should occur prior to laryngospasm. Superinfection can occur, but usually not before 3-4 days after exposure. Leukopenia can occur on day 4-5 after exposure and signals severe immune system dysfunction. See appendix 3 Blister Agents Sulfur Mustard (H, HD, and HT) Patient Information Sheet.

Routine laboratory studies should be done for all patients requiring admission. These include CBC, glucose, and serum electrolytes. Chest x-ray and pulse oximetry (or ABG measurements) are recommended for inhalation exposures. A test for urine thiodiglycol, a metabolite of mustard, can be performed at specialized laboratories, but is not a routine laboratory measure (ATSDR, 2001).

Nitrogen Mustard

Another vesicant, or blister agent, similar to the sulfur mustards are the nitrogen mustards. The nitrogen mustards are also known by their military designations of HN-1, HN-2, and HN-3. Nitrogen mustards are not found naturally in the environment; they were produced in the 1920s and 1930s. HN-1 was originally designed to remove warts, but was discovered to be potential chemical warfare agent. HN-2 was designed to be a chemical warfare agent, but was then used in chemotherapy. HN-3 was also developed as a chemical warfare agent. None of the nitrogen mustards have ever been used in warfare. The Surgeon General's *Report on Medical Management of Chemical Agents* (1997) identifies the nitrogen mustards as a minimal threat because they have not been used in wartime. However, the ATSDR (2001) recognizes the nitrogen mustards as posing a significant threat.

Description

Nitrogen mustards can be clear, pale amber, or yellow colored when in liquid or solid form. Nitrogen mustards come in different forms that can smell fishy, musty, soapy, or fruity. They can be in the form of an oily-textured liquid, a vapor (the gaseous form of a liquid), or a solid. Nitrogen mustards are liquids at normal room temperature (70^{o}F).

Routes of Exposure

If nitrogen mustards are released into the air as a vapor, exposure occurs through respiratory, dermal and eye contact. Nitrogen mustards are heavier than air, so the vapor will settle in low-lying areas. If liquid nitrogen mustards are released into water, exposure is through ingestion of contaminated drinking water or through dermal contact.

Adverse Health Effects

Adverse health effects caused by nitrogen mustards depend on the amount of exposure, the route of exposure, and the length of time of exposure.

Nitrogen mustards are powerful alkalizing agents, however the exact action is not well understood. They are irritants that damage the skin, eyes, and respiratory tract. Nitrogen mustards can enter the cells of the body very quickly and cause damage to the immune system and bone marrow.

As with sulfur mustard, signs and symptoms of nitrogen mustard exposure typically do not occur immediately. Depending on the severity of the exposure, symptoms may not occur for several hours.

Nitrogen mustards can have similar health effects to sulfur mustard, however, in general the effects tend to be more systemic and less localized than those of the sulfur mustard. The nitrogen mustards have the following effects on specific parts of the body:

- *Central nervous system:* High dosages of nitrogen mustards have caused tremors, seizures, incoordination, ataxia, and coma in laboratory animals.

- *Skin*: Erythema and blistering occurs with direct exposure. A rash develops within several hours after exposure followed by blistering within 6 to 12 hours. Prolonged contact or repeated contact can lead to second and third degree chemical burns.

- *Eyes*: Exposure to nitrogen mustard vapor or liquid can cause intense conjunctival and scleral inflammation, pain, swelling, lacrimation, photophobia and corneal damage. High concentrations can cause burns and blindness.

- *Respiratory tract*: Damage to respiratory mucosa begins within hours and may progress over several days. Nasal and sinus pain or discomfort, cough, pharyngitis, laryngitis, and dyspnea may occur within hours; pulmonary edema is uncommon.

- *Digestive tract*: Ingestion can cause chemical burns to the GI tract and hemorrhagic diarrhea. Nausea and vomiting may occur with exposure to any of the routes: inhalation, ingestion or dermal contact.

- *Hematopoietic*: Systemic absorption of nitrogen mustard may induce bone marrow suppression, and an increased risk for fatal complicating infections, hemorrhage and anemia.

- *Delayed Effects/Sequelae*: Chemotherapeutic effects of HN-2 have been associated with menstrual irregularities, alopecia, hearing loss, tinnitus, jaundice, impaired spermatogenesis, generalized swelling and hyperpigmentation. Chronic respiratory and eye conditions may persist; narrowing of the esophagus and severe corrosive damage to the lining of the stomach can occur following ingestion.

Exposure to nitrogen mustard liquid is more likely to produce second and third degree burns and later scarring than is exposure to nitrogen mustard vapor.

Extensive breathing in of the vapors can cause chronic respiratory disease. Extensive eye exposure can cause long-lasting eye problems. Nitrogen mustards may cause bone marrow suppression beginning as early as three to five days after exposure. Bone marrow suppression can cause anemia, bleeding, and increased risk for infection. If severe, these effects could lead to death.

Prolonged or repeated exposures to nitrogen mustards have caused cancer in animals. Some evidence exists that prolonged or repeated exposures to nitrogen mustards cause leukemia in humans.

Management of Exposure

Because no antidote exists for nitrogen mustard exposure, the best thing to do is avoid it. If the nitrogen mustard release was indoors, get out of the building. If the release was outdoors, move away from the area of the release, stay upwind if possible, and seek higher ground. Quickly moving to an area where fresh air is available is highly effective in reducing the possibility of death from exposure to nitrogen mustard. Emergency coordinators may direct the population to evacuate or to shelter in place.

Decontamination

Quickly take off clothing that has liquid nitrogen mustard on it. Cut off any clothing that has to be pulled over the head, to avoid recontamination. Whenever possible, seal the clothing in a plastic bag and then double bag. Removing and sealing the clothing in this way will help protect others from any chemicals that might be on the clothing. When helping others remove contaminated clothing, avoid touching any contaminated areas, and remove the clothing as quickly as possible.

As quickly as possible, wash any liquid nitrogen mustard from the skin with large amounts of soap and water.

Rinse the eyes with plain water for 10 to 15 minutes if there is burning or if vision is blurred. If the exposed person wears contact lenses, remove them and place them in the bags with the contaminated clothing. Do not put the contacts back into the eyes. Even if they are not disposable lenses, they should be discarded into the contaminated bag. Those who wear eyeglasses can wash them with soap and water to decontaminate them; they can then be used again.

If nitrogen mustard has been ingested, **do not induce vomiting or give fluids to drink.**

Treatment of Exposure

No antidote exists for nitrogen mustard exposure. Treatment consists of removing the nitrogen mustard from the body as soon as possible and providing supportive medical care in a hospital setting.

Lewisite

Lewisite is an arsenical agent, considered to be a vesicant or blistering agent. However, it does not cause vesicles, but rather solid lesions that resemble urticaria. Lewisite is also known by its military designation, "L." Lewisite was produced in 1918 to be used in World War I, but its production was too late for it to be used in that war. According to the ATSDR (2001) large amounts were produced by the United States to be used in Europe; however, World War I ended while the shipment was at sea and the vessel was sunk. There have been allegations that it was used by Japan against Chinese forces in the late 1930s; however, there are no confirmed reports that it has been ever been used in warfare. Its only use is as a chemical warfare agent; it has no medical or other practical use. Lewisite is not found naturally in the environment.

Lewisite is used as a single agent or combined with distilled mustard. The mustard-lewisite mixture is discussed in the next section of this course.

Description

Lewisite is an oily, colorless liquid in its pure form and can appear amber to black in its impure form. Lewisite has an odor like geraniums. It contains arsenic, a poisonous element.

Management of Exposure

The risk for exposure depends on how close one is to where the lewisite was released. If lewisite gas is released into the air, exposure can occur through inhalation, dermal contact or eye contact.

If lewisite liquid is released into water, exposure occurs through ingestion of the drinking water that contains lewisite or by dermal contact with the water. Food can be contaminated with lewisite liquid; exposure occurs through ingestion. Ingestion of Lewisite is an uncommon route for exposure but can lead to local effects and systemic absorption (ATSDR, 2001).

Lewisite vapor is heavier than air, so it will settle in low-lying areas. Lewisite remains a liquid under a wide range of environmental conditions, from below freezing to very hot temperatures. This property allows it to be effective in cold climates; it can last for a long time in the environment.

Adverse Health Effects of Lewisite

Lewisite can be distinguished from sulfur mustard because Lewisite causes severe pain on contact. Another vesicant, phosgene oxime (covered later in the course) also causes pain, but it does not cause the liquid filled blisters that result from lewisite exposure. Additionally, urinary arsenic excretion may be helpful in identifying possible lewisite exposure (Office of the Surgeon General, 1997).

Adverse health effects caused by lewisite depend on the amount of exposure, the route of exposure, and the length of time of exposure. It is a powerful irritant and blistering agent that immediately damages the skin, eyes, and respiratory tract. Liquid lewisite causes severe eye damage within minutes of contact. The vapor also acts quickly, with pain on contact, followed by edema of the conjunctiva and eyelids, and iritis and corneal damage with high doses (ATSDR, 2001). Because it contains arsenic, lewisite has some effects that are similar to arsenic poisoning, including stomach ailments and low blood pressure (Siddell et al., 1997).

After exposure to low amounts of lewisite or to the mixture, temporary loss of eyesight may occur because of blepharospasm or eyelid edema. After exposure to high amounts, permanent loss of sight may occur because of corneal damage; however, this is unusual (ATSDR, 2001).

Lewisite liquid or vapor produces pain and skin irritation within seconds to minutes after contact. With liquid lewisite, erythema occurs within 15 to 30 minutes after exposure and blisters start within several hours, developing fully by 12-18 hours. For the vapor, response times are a little longer. The lewisite blister starts as a small blister in the center of the erythematous area and expands to include the entire inflamed area. Skin burns take up to 18 hours to fully develop. Chemical pneumonitis may begin within 24 hours or up to 3 days after inhalation exposure (ATSDR, 2001). Most information on the health effects of lewisite is based on animal studies. Signs and symptoms occur immediately following a lewisite exposure. Lewisite can have the following effects on specific parts of the body:

- *Skin:* pain and irritation within seconds to minutes, redness within 15 to 30 minutes followed by blister formation within several hours. The blister begins as a small blister in the middle of the red areas and then expands to cover the entire reddened area of skin. The lesions from lewisite heal much faster than lesions caused by the other blistering agents, sulfur mustard and nitrogen mustards, and the discoloring of the skin that occurs later is much less noticeable.

- *Eyes:* irritation, pain, swelling, and tearing may occur on contact. Lewisite vapor causes pain and blepharospasm on contact. Edema of the conjunctiva and eyelids follows, and the eyes may be swollen shut within an hour. With high doses, corneal damage and iritis may follow. Liquid lewisite causes severe eye damage on contact.

- *Respiratory tract:* lewisite is extremely irritating to the respiratory tract mucosa. Burning nasal pain, epistaxis, sinus pain, laryngitis, cough, runny nose, sneezing, hoarseness, bloody nose and dyspnea may occur. Necrosis can cause pseudomembrane formation and local airway obstruction. Pulmonary edema may occur following exposure to high concentrations.

- *Digestive tract:* Ingestion or inhalation of lewisite may cause nausea, vomiting and/or diarrhea.

- *Cardiovascular:* High-dose exposure to lewisite may cause "lewisite shock," a condition resulting from increased capillary permeability and subsequent intravascular fluid loss, hypovolemia, and organ congestion; hypertension.

- Hepatic: Hepatic necrosis may occur due to shock and hypoperfusion following exposure to high levels of lewisite.

- Renal: Exposure to high levels of lewisite may cause decreased renal function secondary to hypotension.

Long-term adverse health effects include:

- Extensive skin burning, as seen with sulfur mustard, is less likely.

- Extensive breathing in of the vapors may cause chronic respiratory disease.

- Extensive eye exposure may cause permanent blindness.
- Unlike sulfur mustard, lewisite is not known to suppress the immune system.

Management of Exposure to Lewisite

Immediately leave the area where the lewisite was released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing the possibility of death from exposure to lewisite.

If the lewisite release was outdoors, move away from the area where the lewisite was released. Go to the highest ground possible, because lewisite is heavier than air and will sink to low-lying areas. If the lewisite release was indoors, get out of the building.

Emergency coordinators will direct the population regarding evacuation or sheltering in place.

Decontamination of Lewisite

Quickly take off all clothing that has liquid lewisite on it. Any clothing that has to be pulled over the head should be cut off the body instead of pulled over the head. Seal the clothing in a plastic bag, double-bagging whenever possible.

When helping others remove clothing, avoid touching any contaminated areas so as not to expose yourself to the chemical threat; remove the clothing as quickly as possible.

As quickly as possible, wash any liquid lewisite from the skin with large amounts of soap and water. Washing with soap and water will help protect people from any chemicals on their bodies.

If the eyes are burning or vision is blurred, rinse the eyes with plain water for 10 to 15 minutes. If contact lenses are worn, remove them and place them in the bags with the contaminated clothing. Do not put the contacts back in the eyes. Eyeglasses can be washed with soap and water and then worn again after cleaning.

If lewisite was ingested, **do not induce vomiting or drink fluids**. Immediate medical care is required.

Treatment of Exposure

Treatment consists of removing lewisite from the body as soon as possible and providing supportive medical care in a hospital setting. An antidote for lewisite is available and is most useful if given as soon as possible after exposure.

As above, for exposure by ingestion, **do not induce emesis**. Treat nausea and vomiting with anti-emetics.

The antidote used is British Anti-Lewisite (BAL), also called Dimercaprol; it is a chelating agent shown to reduce systemic effects from Lewisite exposure. Due to toxic side effects, **BAL should be administered only to patients who have signs of shock or significant pulmonary injury**.

Chelation therapy should be performed only by trained personnel. Consultation with the regional poison control center is recommended. The standard dosage regimen is 3 to 5 mg/kg IM every 4 hours for four doses. This regimen can be adjusted depending on the severity of the exposure and the symptoms. Contraindications to BAL include pre-existing renal disease, pregnancy (except in life threatening circumstances) and concurrent use of medicinal iron (ATSDR, 2001).

Alkalization of the urine stabilizes the Dimercaprol-metal complex and has been proposed to protect the kidneys during chelation therapy. If acute renal insufficiency develops, hemodialysis should be considered to remove the Dimercaprol-arsenic complex. Side effects of BAL administered at 3 mg/kg are mostly pain at the injection site. At 5 mg/kg, the effects may include nausea; vomiting; headache; burning sensation of the lips, mouth, throat, and eyes; lacrimation;

rhinorrhea; salivation; muscle aches; burning and tingling in the extremities; tooth pain; diaphoresis; chest pain; anxiety; and agitation (ATSDR, 2001).

Routine laboratory studies should be done for all patients requiring admission. These include CBC, glucose, serum electrolytes, and liver and kidney function tests. Consider monitoring hourly fluid intake and output. Chest X-ray and pulse oximetry (or ABG measurements) are recommended for all patients with inhalation exposures. Since Lewisite contains arsenic, urinary arsenic excretion may be helpful if the diagnosis is in doubt. A test for urine thiodiglycol, a metabolite of mustard, can be performed at specialized laboratories, but is not a routine laboratory measure (ATSDR, 2001).

Patients who initially had mild symptoms should be observed for at least 18 to 24 hours after exposure. If no further symptoms develop and there is no significant progression, the patient may be discharged. Discharged patients should be advised to rest and to seek medical care promptly if symptoms develop (See Lewisite and Mustard-Lewisite Mixture Patient Information Sheet in Appendix 2) (ATSDR, 2001).

Follow-up laboratory evaluation of bone marrow, hepatic, and renal function should be arranged for severely exposed patients until they are completely recovered. Patients who have mild skin burns or corneal lesions should be reexamined within 24 hours (ATSDR, 2001).

Mustard-Lewisite Mixture

Description

Mustard-Lewisite Mixture is a liquid mixture of distilled Mustard (HD) and Lewisite. Due to its low freezing point, the mixture remains a liquid in cold weather and at high altitudes. The mixture with the lowest freezing point consists of 63% Lewisite and 37% Mustard. Mustard-Lewisite Mixture is a mixture of distilled Mustard and Lewisite developed to achieve a lower freezing point for ground dispersal and aerial spraying (ATSDR, 2001). It has a garlic-like odor.

Routes of Exposure

Exposure to Mustard-Lewisite Mixture vapor induces immediate respiratory tract irritation and severe inflammation after a few hours latency period. Both agents are readily absorbed from the lungs. Ingestion of Mustard-Lewisite Mixture is an uncommon route for exposure but can lead to local effects and systemic absorption (ATSDR, 2001).

Adverse Health Effects

After exposure to low amounts of Lewisite or to the mixture, temporary loss of eyesight may occur because of blepharospasm or eyelid edema. After exposure to high amounts, permanent loss of sight may occur because of corneal damage; however, this is unusual (ATSDR, 2001).

Systemic absorption may occur following skin or eye exposure to liquid or vapor Mustard-Lewisite Mixture. The mixture causes immediate stinging pain of the skin, with blistering delayed for hours. Graying of the skin will follow within a very short time if exposure is from liquid (because of Lewisite). Erythema and blisters will appear earlier than from mustard alone. Exposure of the eyes to Mustard-Lewisite Mixture produces lacrimation and inflammation of the conjunctiva and cornea (ATSDR, 2001).

Decontamination

The process of decontamination for mustard-Lewisite mixture is similar to that of Lewisite. Please see the previous section on Lewisite decontamination.

Phosgene Oxime

Phosgene oxime is a manufactured chemical warfare agent, first produced in 1929; it was never used on the battlefield. Information about phosgene oxime is limited. It is also known by its

military designation, “CX.” Phosgene oxime, although categorized as a vesicant, is not a true vesicant. It is an urticant or nettle agent because dermal contact produces erythema, urticaria, intense itching and a rash similar to hives. Phosgene oxime is also referred to as a corrosive agent because it causes extensive tissue damage.

Phosgene, as opposed to phosgene oxime, is classified as a pulmonary agent by the US Department of the Army’s *Medical Aspects of Chemical and Biological Warfare*, and will be covered under the section on pulmonary agents later in this course.

Description

Phosgene oxime, in its pure form, is a colorless, crystalline solid; munitions grade compound is a yellowish-brown. Phosgene oxime has a disagreeable, irritating odor. Phosgene oxime is not found naturally in the environment.

Exposure to Phosgene Oxime

The risk for exposure depends on the amount of exposure, the route of exposure and the length of time of the exposure, as well as how close one is to the location of the release of phosgene oxime.

If phosgene oxime gas is released into the air, exposure occurs through inhalation, dermal contact or eye contact. In its liquid form it can be released into water or food; exposure then occurs through ingestion of the water or food, or by direct dermal contact with the water or food.

Phosgene oxime vapor is heavier than air, so it will settle in low-lying areas. Phosgene oxime does not last in the environment for very long. It breaks down in soil within two hours when temperatures are normal, and it breaks down in water within a few days.

Adverse Health Effects of Phosgene Oxime

The extent of the adverse effects of phosgene oxime depends on the amount of exposure, the route of exposure and the length of time of exposure to phosgene oxime. Limited information is available on the effects of this agent on humans (Siddell et al., 1997).

Phosgene oxime is an attractive warfare agent because of its rapid and prolonged effect; it can penetrate clothing and rubber faster than other chemical warfare agents (Siddell et al., 1997). This rapidity of onset of symptoms and the rapid necrosis that follows helps to identify this agent in a chemical attack.

Phosgene oxime produces instant and almost unbearable pain on exposed skin and exposed eyes. When inhaled, it causes immediate irritation to the respiratory tract. Signs and symptoms occur immediately following a phosgene oxime exposure:

- *Skin:* pain occurring within a few seconds, and blanching of the skin surrounded by red rings occurring on the exposed areas within 30 seconds. Within about 15 minutes, the skin develops hives. After 24 hours, the whitened areas of skin become brown and die. Over the next 7 days an eschar forms and healing occurs from below through granulation. The lesion can extend to the underlying panniculus and muscle and is surrounded by an inflammatory response. Healing can take over 4-6 months and be accompanied by itching and pain throughout the healing process.

- *Eyes:* severe pain and irritation, tearing, and possibly temporary blindness.

- *Respiratory tract:* immediate irritation to the upper respiratory tract, causing runny nose, hoarseness, and sinus pain. Absorbing phosgene oxime through the skin or inhaling it may result in pulmonary edema with symptoms of shortness of breath and cough. The pulmonary edema can develop after a delay of several hours. Pulmonary thromboses can be prominent.

- *Digestive tract:* no information exists on digestive tract effects in humans.

No information is available on the long-term health effects of phosgene oxime in humans.

Management of Exposure

Leave the area where the phosgene oxime was released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing exposure to phosgene oxime.

If the phosgene oxime release was outdoors, move away from the area where the phosgene oxime was released. Go to the highest ground possible, because phosgene oxime is heavier than air and will sink to low-lying areas. If the phosgene oxime release was indoors, get out of the building.

Emergency coordinators will direct the population regarding evacuation or sheltering in place.

Decontamination

Decontamination must occur immediately after contact because phosgene oxime is absorbed from the skin within seconds.

Quickly remove all clothing containing liquid phosgene oxime. Any clothing that has to be pulled over the head should be cut off the body. Clothing should be sealed in a plastic bag, double bagging whenever possible.

When helping others remove clothing, avoid touching any contaminated areas; remove the clothing as quickly as possible.

Immediately wash any liquid phosgene oxime from the skin with large amounts of soap and water. If the eyes are burning or if vision is blurred, rinse the eyes with plain water for 10 to 15 minutes. If contact lenses are used, remove them before rinsing your eyes, and then discard them in the bags with the contaminated clothing. Do not put the contacts back into the eyes; they should be discarded even if they are not disposable contacts. If eyeglasses are worn, wash them with soap and water; they can be worn after decontamination.

If phosgene oxime was ingested, **do not induce vomiting or drink fluids**.

Treatment of Exposure

No antidote exists for phosgene oxime. Treatment consists of removing the phosgene oxime from the body as soon as possible and providing supportive medical care in a hospital setting. These include fluid restriction; necrotic areas must be kept clean and infection avoided.

Blood Agents

Blood agents are chemicals that primarily impact on the hematological system or are those that are readily absorbed by the blood. These include the various preparations of cyanide including: cyanogens chloride, hydrogen chloride, potassium cyanide, sodium cyanide, carbon monoxide and arsine. This course will focus on cyanide and arsine.

<u>Cyanide</u>

Cyanide is a rapidly acting, potentially deadly chemical that can exist in various forms. Cyanide is also known by the military designations AN (for hydrogen cyanide) and CK (for cyanogen chloride). Cyanide has been used in warfare for centuries. The ancient Romans used cyanide against their enemies. The emperor Nero used cherry laurel water with cyanide as the chief toxic agent, to poison those who displeased him. Cyanide agents were used by the French and the Austrians during World War I, with little success. Hydrogen cyanide, under the name Zyklon B, was used as a genocidal agent by the Germans in World War II against civilians and enemy soldiers in the death camps. Reports have indicated that during the Iran-Iraq War in the 1980s,

hydrogen cyanide gas may have been used along with other chemical agents against the inhabitants of the Kurdish city of Halabja in northern Iraq (Baskin and Brewer, 1997).

Description

Cyanide can be a colorless gas, such as hydrogen cyanide (HCN) or cyanogen chloride (CNCl), or a crystal form such as sodium cyanide (NaCN) or potassium cyanide (KCN). At temperatures below 78ºF, hydrogen cyanide is a colorless or pale-blue liquid (hydrocyanic acid); at higher temperatures, it is a colorless gas (ATSDR, 2001).

Cyanide sometimes is described as having a “bitter almond” smell, but it does not always give off an odor, and not everyone can detect this odor.

Cyanide is widely used in industry and it is naturally present in some foods. It is present in cassava, which is a staple in many parts of the world. In some areas of Africa cassava is responsible for the high rate of ataxic neuropathy (Baskin and Brewer, 1997). Cyanide is contained in cigarette smoke and the combustion products of synthetic materials such as plastics. It is responsible for the high rate of death, along with carbon monoxide, in residential fires (Baskin and Brewer, 1997).

In manufacturing, cyanide is used to make paper, textiles, and plastics. It is present in the chemicals used to develop photographs. Cyanide salts are used in metallurgy for electroplating, metal cleaning, and removing gold from its ore. Cyanide gas is used to exterminate pests and vermin in ships and buildings (Baskin and Brewer, 1997). Hydrogen cyanide gas is a by-product of coke-oven and blast-furnace operations (ATSDR, 2001).

Cyanide has a very short half-life, so victims of residential fires, who have blood drawn when they are sent to the Emergency Department, often do not present with high serum cyanide levels (Baskin and Brewer, 1997). Although cyanide is considered a lethal substance, compared with other chemical warfare agents, it is among the least toxic.

Exposure

Exposure to cyanide occurs through inhalation, ingestion of contaminated drinking water or food or through dermal contact with soil that contains cyanide.

Cyanide enters water, soil, or air as a result of both natural processes and industrial activities. In air, cyanide is present mainly as gaseous hydrogen cyanide.

Smoking cigarettes is probably one of the major sources of cyanide exposure for people who do not work in cyanide-related industries.

The extent of poisoning caused by cyanide depends on the amount of exposure, the route of exposure and the length of time of exposure. Breathing cyanide gas causes the most harm, but ingestion is also toxic.

Cyanide gas is most dangerous in enclosed places, where the gas becomes trapped. It is less dense than air, so it will rise. This is a unique feature among the chemical agents used in warfare or terrorism, as most of the others are heavier than air. Cyanide gas evaporates and disperses quickly in open spaces, making it less harmful outdoors.

Victims whose clothing or skin is contaminated with hydrogen cyanide liquid or solution can secondarily contaminate response personnel by direct contact or through off-gassing vapors. Avoid dermal contact with cyanide-contaminated victims or with gastric contents of victims who may have ingested cyanide-containing materials. Victims exposed only to hydrogen cyanide gas do not pose contamination risks to rescuers.

Adverse Health Effects

The effects from cyanide poisoning are those of progressive histotoxic tissue hypoxia. The symptoms, signs, and physical findings are directly related to the dose of cyanide, the route of exposure, and the type of cyanide compound (Baskin and Brewer, 1997).

In humans, hydrogen cyanide acts as a cellular asphyxiant. It combines with the ferric ion in mitochondrial cytochrome oxidase, preventing electron transport in the cytochrome system and bringing oxidative phosphorylation and ATP production to a halt. The inhibition of oxidative metabolism puts increased demands on anaerobic glycolysis, which results in lactic acid production and may produce severe acid-base imbalance. The CNS is particularly sensitive to the toxic effects of cyanide, and exposure to hydrogen cyanide generally produces symptoms within a short period of time (ATSDR, 2001).

Exposure to cyanide occurs through inhalation, skin absorption or through ingestion.

Exposure to cyanide gas can be fatal within minutes if exposure is to high concentrations. An initial hyperpnea (15 sec after exposure), due to the effect of cyanide on the chemoreceptor bodies, is closely followed by a loss of consciousness (30 sec after exposure). This progresses to apnea (3–5 min after exposure), cessation of cardiac activity (5–8 min after exposure), and death (Baskin and Brewer, 1997). The effects of exposure to lower concentrations of inhaled cyanide gas, or exposure to lethal amounts via the oral or percutaneous routes, are slower to develop.

For example, after ingestion of a lethal dose of a cyanide salt, the casualty might have 15 to 30 minutes of survival time during which an antidote could be administered (Baskin and Brewer, 1997).

Prominent early signs and symptoms of cyanide poisoning include a transient hyperpnea, headache, dyspnea, and findings of general central nervous system (CNS) excitement, including anxiety, personality changes, and agitation progressing to seizures. Diaphoresis, flushing, weakness, and vertigo may also be present. Late-appearing indications of CNS depression, such as coma and dilated, unresponsive pupils, are prominent signs of cyanide intoxication. These signs are not specific for cyanide poisoning, which makes the distinction from other types of poisoning very difficult without a history of exposure. The telltale odor of bitter almonds cannot be used as a guide because 40% to 60% of the population is unable to detect the odor (Baskin and Brewer, 1997).

Because the toxic effect of cyanide is to block tissue uptake and utilization of oxygen, the casualty is transiently flushed and may have other, related signs of poor tissue oxygen extraction. For example, funduscopic examination shows an equally bright red color for retinal arteries and veins because of poor oxygen extraction. Increased oxygenation of venous blood is also responsible for a “cherry-red” skin color, but this sign may not always be present (Baskin and Brewer, 1997).

Symptoms of cyanide poisoning include:

- Tachypnea
- Restlessness
- Dizziness
- Weakness
- Headache
- Nausea and vomiting
- Rapid heart rate

Exposure to a large amount of cyanide by any route may cause these other health effects as well:

- Convulsions
- Hypotension
- Slow heart rate
- Loss of consciousness
- Lung injury
- Respiratory failure leading to death

If accidentally ingested (swallowed), chemicals found in acetonitrile-based products that are used to remove artificial nails can produce cyanide.

Survivors of serious cyanide poisoning may develop heart and brain damage.

Management of Exposure

First, get fresh air by leaving the area where the cyanide was released. Moving to an area with fresh air is a good way to reduce the possibility of death from exposure to cyanide gas.

If the cyanide release was outside, move away from the area where the cyanide was released. If the cyanide release was indoors, get out of the building.

If leaving the area that was exposed to cyanide is not an option, stay as low to the ground as possible because cyanide rises.

Decontamination

Victims exposed only to hydrogen cyanide gas do not pose secondary contamination risks to rescuers, but do not attempt resuscitation without a barrier. Victims whose clothing or skin is contaminated with hydrogen cyanide liquid or solution can secondarily contaminate response and medical personnel by direct contact or through off-gassing vapor. Avoid dermal contact with cyanide-contaminated victims or with gastric contents of victims who may have ingested cyanide-containing materials (ATSDR, 2001).

If the patient's clothing is wet with hydrogen cyanide solution, quickly remove contaminated clothing while flushing exposed skin and hair with plain water for 2 to 3 minutes (preferably under a shower), then wash twice with mild soap. Use caution to avoid hypothermia when decontaminating children or the elderly. Use blankets or warmers when appropriate (ASTDR, 2001).

Rinse thoroughly with water. Double-bag contaminated clothing and personal belongings.

Irrigate exposed eyes for at least 5 minutes. Remove contact lenses if easily removable without additional trauma to the eye.

In cases of ingestion, **do not induce emesis**. If activated charcoal has not been administered previously, and the victim is alert, asymptomatic, and has a gag reflex, administer a slurry of activated charcoal (administer at 1 gm/kg, usual adult dose 60-90 g, child dose 25-50 g). A soda can and a straw may be of assistance when offering charcoal to a child. Consider gastric lavage if the patient is conscious and it can be performed shortly after ingestion. Because cyanide absorption from the gut is rapid, the effectiveness of activated charcoal will depend on how quickly after ingestion it can be administered. Isolate gastric washings and vomitus; they may off-gas hydrogen cyanide (ATSDR, 2001).

Treatment

It is essential that the victim of cyanide exposure seek medical treatment as soon as possible. **Speed is critical**. For symptomatic victims, provide treatment with 100% oxygen and specific antidotes as needed. Treatment should be given simultaneously with decontamination procedures (ATSDR, 2001).

The most important elements of therapy are general supportive actions, which can, by themselves, affect the recovery of most casualties without further risk from specific antidotal therapy. They are probably the only indicated therapies for casualties of cyanide poisoning who arrive conscious at the emergency medical treatment station.

Patients who have signs or symptoms of significant systemic toxicity should be evaluated for antidotal treatment. In the United States, antidotes for cyanide include amyl nitrite perles and intravenous infusions of sodium nitrite and sodium thiosulfate (ATSDR, 2001). See Appendix 4 Hydrogen Cyanide Patient Information Sheet.

If a dose of the antidotes were administered by response or prehospital personnel and inadequate clinical response has occurred, a second dose of one-half the initial amounts may be given 30 minutes after the initial dose. Further doses should be guided by the patient's clinical condition and not by the percentage of methemoglobin induced. The usual methods of monitoring methemoglobin levels are unreliable in cases of cyanide poisoning and may seriously underestimate the levels of inactive hemoglobin.

Amyl nitrite perles should be broken onto a gauze pad and held under the nose, over the Ambu-valve intake, or placed under the lip of the face mask. Inhale for 30 seconds every minute and use a new perle every 3 minutes if sodium nitrite infusions will be delayed (ATSDR, 2001).

If the patient has not responded to oxygen and amyl nitrite treatment, infuse sodium nitrite intravenously as soon as possible. The usual adult dose is 10 mL of a 3% solution (300 mg) infused over **absolutely no less than 5 minutes**; the average pediatric dose is 0.12 to 0.33 mL/kg body weight up to 10 mL infused as above. Monitor blood pressure during sodium nitrite administration, and slow the rate of infusion if hypotension develops (ATSDR, 2001).

Then sodium thiosulfate is infused intravenously. The usual adult dose is 50 mL of a 25% solution (12.5 g) infused over 10 to 20 minutes; the average pediatric dose is 1.65 mL/kg of a 25% solution. Repeat one-half of the initial dose 30 minutes later if there is an inadequate clinical response (ATSDR, 2001).

Amyl nitrite and sodium nitrite oxidize the ferrous iron of hemoglobin to methemoglobin. Methemoglobin levels should not exceed 20%. Repeat treatment with nitrite and thiosulfate as required (ATSDR, 2001).

The efficacy of hyperbaric oxygen in cyanide poisoning is unproven. It has been reported to be useful in severe cases of smoke inhalation combined with exposure to hydrogen cyanide and carbon monoxide (ATSDR, 2001). Supplemental oxygen with or without assisted ventilation may be beneficial.

Lactic acidosis resulting from anaerobic metabolism should be treated by intravenous administration of sodium bicarbonate, and seizures should be controlled by the administration of anticonvulsants such as diazepam. Because correction of deficiencies in tissue perfusion and oxygenation is the ultimate goal of supportive therapy and is also important for the success of specific antidotal therapy, it is critically important to maintain an effective cardiac rhythm; this can be accomplished with cardiopulmonary resuscitation, if necessary, in the early stages of treatment.

Specific antidotes are used in the treatment of cyanide exposure, such as sodium nitrite and sodium thiosulfate, as well as supportive medical care described above. Table 3 describes NYSDOH treatment for cyanide exposure.

TABLE 3:ANTIDOTE RECOMMENDATIONS FOLLOWING EXPOSURE TO CYANIDE (NYS, 2001)

Note –If the patient is a victim of recent smoke inhalation (may have high carboxyhemoglobin levels), administer only sodium thiosulfate.

Patient	Mild (conscious)	Severe (unconscious)	Other Treatment
Child	If patient is conscious and has no other signs or symptoms, antidotes may not be necessary.	Sodium nitrite[1]: 0.12 - 0.33 ml/kg, not to exceed 10 ml of 3% solution[2] slowly IV over no less than 5 minutes, or slower if hypotension develops **and** Sodium thiosulfate: 1.65 ml/kg of 25% solution IV over 10 - 20 minutes	For sodium nitrite-induced orthostatic hypotension, normal saline infusion and supine position are recommended. If still apneic after antidote administration, consider sodium bicarbonate for severe acidosis.
Adult	If patient is conscious and has no other signs or symptoms, antidotes may not be necessary.	Sodium nitrite[1]: 10 - 20 ml of 3% solution[2] slowly IV over no less than 5 minutes, or slower if hypotension develops **and** Sodium thiosulfate: 50 ml of 25% solution IV over 10 - 20 minutes	

1. If sodium nitrite is unavailable, administer amyl nitrite by inhalation from crushable ampules. If neither is available, then use sodium thiosulfate alone.
2. Available from Taylor Pharmaceuticals in cyanide antidote kit, formerly known as the Pasadena Cyanide Antidote Kit or the Lilly Cyanide Kit.

The diagnosis of acute cyanide toxicity is primarily a clinical one (based on rapid onset of CNS toxicity and cardiorespiratory collapse). Laboratory testing is useful for monitoring the patient and evaluating complications. Cyanide exposure can be diagnosed through laboratory testing, whenever cyanide concentration is higher than the normal reference range (0.02--0.05 µg/mL) in whole blood.

Routine laboratory studies for all exposed patients include CBC, blood glucose, and electrolyte determinations.

- OR-

Environmental: Detection of cyanide in environmental samples, as determined by NIOSH or FDA.

Additional studies for patients exposed to hydrogen cyanide include ECG monitoring, determinations of serum lactate, chest radiography, and pulse oximetry (or ABG measurements).

In severe poisonings, venous blood is oxygenated and has a bright red color. Elevated venous PO_2 and venous percent O_2 saturation occurs, narrowing the gap between arterial and central venous PO_2 or percent O_2 saturation.

After treatment with nitrites, serum methemoglobin levels may be monitored. However, the usual methods of monitoring methemoglobin levels are unreliable in cases of cyanide poisoning and may seriously underestimate the levels of inactive hemoglobin. Alternative methods exist, but may not be available. Whole blood cyanide tests generally require several hours and cannot be used to guide emergency treatment. However, blood cyanide levels may be useful in documenting exposure.

Patients with histories of significant exposure and who are symptomatic should be hospitalized. Admission to intensive care units is indicated whenever infusions of the cyanide antidotes are needed (ATSDR, 2001).

Patients who remain asymptomatic 4 to 6 hours after exposure may be discharged with instructions to seek medical care promptly if symptoms develop (ATSDR, 2001).

Table 4: Chemical Agents

AGENT	SIGNS AND SYMPTOMS	DECONTAMINATION	PERSISTENCE
Nerve Agents			
Tabun (GA)	Salivation Lacrimation Urination Defecation Gastric disturbances Emesis	Remove contaminated clothing Flush with a soap and water solution for patients Flush with large amounts of a 5% bleach and water solution for objects	1–2 days if heavy concentration
Sarin (GB)			1–2 days will evaporate with water
Soman (GD)			Moderate, 1–2 days
V Agents (VX)			High, 1 week if heavy concentration As volatile as motor oil
Vesicants (Blister Agents)			
Sulfur Mustard (H)	Acts first as a cell irritant, then as a cell poison. Conjunctivitis, reddened skin, blisters, nasal irritation, inflammation of throat and lungs.	Remove contaminated clothing Flush with soap and water solution for patients. Flush with large amounts of a 5% bleach and water solution for objects	Very high, days to weeks
Distilled Mustard (HD)			
Nitrogen Mustard (HN 1,3)			
Mustargen (HN2)			Moderate
Lewisite (L)	Immediate pain with blisters later.		Days, rapid hydrolysis with humidity
Phosgene Oxime (CX)	Immediate pain with blisters later—necrosis equivalent to second and third degree burns		Low, 2 hours in soil
Chemical Asphyxiants (Blood agents)			
Hydrogen Cyanide (AC)	Cherry red skin or ~ 30% cyanosis.	Remove contaminated	Extremely volatile, 1-2 days

Cyanogen Chloride (CK)	Patients may appear to be gasping for air. Seizures prior to death. Effect is similar to asphyxiation, but is more sudden.	clothing. Flush with a soap and water solution for patients. Flush with large amounts of 5% bleach and water solution for objects.	Rapidly evaporates and disperses
Arsine (SA)			Low

Arsine

Description

Arsine is a colorless, nonirritating toxic gas with a mild garlic odor. The odor can be detected only at levels greater than those necessary to cause poisoning. Arsine is formed when arsenic comes in contact with an acid.

Arsine is similar to a gas called stibine, which is formed when the metal antimony comes in contact with an acid. Stibine has health effects similar to those of arsine, but it is not as widely available, and it has a much more noticeable odor (like rotten eggs).

Exposure

Although arsine was investigated as a warfare agent during WWII, it was never used on the battlefield. Arsine is most commonly used in the semiconductor and metals refining industries.

Reports of exposure to arsine have most commonly been after accidental formation of arsine in the workplace.

The most frequent route of exposure is through inhalation after arsine is released into the air. Absorption into the body through the eyes and the skin has not been known to occur.

Arsine vapor is heavier than air, so it would be more likely to settle in low-lying areas.

Because no antidote exists for arsine exposure, the best thing to do is avoid it. First, get fresh air by leaving the area where the arsine was released. Moving to an area with fresh air is a good way to reduce the possibility of death from exposure to arsine.

If the arsine release was outside, move away from the area where the arsine was released. If the arsine release was indoors, get out of the building. Emergency Management Personnel will direct those with potential exposure to arsine. You may be directed to shelter in place or evacuate, depending on the location of the release of arsine.

Clothing should be removed if there is suspicion of arsine exposure; cut off clothing that must be pulled over the head. Utilize the decontamination process previously described.If you think you may have been exposed to arsine, you should remove your clothing, rapidly wash your entire body with soap and water, and get medical care as quickly as possible. Seek medical attention as soon as possible.

Adverse Health Effects

The extent of poisoning caused by arsine depends on the amount of arsine to which a person has been exposed and on the length of time of the exposure.

Depending on the intensity of exposure to arsine, symptoms may occur 2 to 24 hours after exposure. However, exposure to high doses of arsine can be immediately fatal.

After arsine enters the bloodstream, it damages the red blood cells and leads to symptoms as a direct result of this damage.

At lower doses, people may not know they have been exposed to arsine, because it has no odor. At higher doses, a mild garlic odor has been reported. Stibine, on the other hand, has a strong odor, so people will probably be aware that they may have been exposed to something. People exposed to a low or moderate dose of arsine by inhalation may experience some or all of the following symptoms within 2 to 24 hours of exposure:

- Weakness
- Fatigue
- Headache
- Drowsiness
- Confusion
- Dyspnea
- Nausea, vomiting, and/or abdominal pain
- Red or dark urine
- Jaundice
- Muscle cramps

Exposure to a large dose of arsine by any route may result in these additional health effects:

- Oliguria
- Renal failure
- Loss of consciousness
- Convulsions
- Paralysis
- Respiratory failure, possibly leading to death
- Showing these signs and symptoms does not necessarily mean that a person has been exposed to arsine.

Severely exposed people are not likely to survive. If people survive the initial exposure, long-term effects may include kidney damage, numbness and pain in the extremities, and neuropsychological symptoms such as memory loss, confusion, and irritability.

Treatment of Exposure

No antidotes are available for arsine. Treatment consists of providing supportive medical care in a hospital setting. Blood transfusions and intravenous fluids may be needed. Some people may require hemodialysis for kidney failure.

Pulmonary Agents

Inhalation of selected chemicals can result in acute lung injury and varying degrees of pulmonary edema, usually after a symptom-free period that varies in duration with the amount inhaled.

Chlorine

Chlorine is an element used in industry and found in many household products; it is one of the most commonly manufactured chemicals in the United States. It's most important use is as a bleach in the manufacture of paper and cloth, but it is also used to make pesticides, rubber, and solvents.

Chlorine is used in drinking water and swimming pool water to kill pathogens; it is also as used as part of the sanitation process for industrial waste and sewage. Household chlorine bleach can release chlorine gas if it is mixed with other cleaning agents, most specifically ammonia.

Chlorine can be used as a liquid or a gas. Chlorine gas can be pressurized and cooled to change it into a liquid so that it can be shipped and stored. When liquid chlorine is released, it quickly turns into a gas that stays close to the ground and spreads rapidly.

Chlorine was used during World War I as a pulmonary or choking agent.

Description

Chlorine is a dense, acrid, pungent, greenish-yellow gas that is easily recognized by both color and odor, which is like the odor of bleach. Because of its density and tendency to settle in low-lying areas, this gas is hazardous in closed spaces. Because the gas has a characteristic odor, chlorine is said to have good warning properties. However, chronic exposures are thought to lead to a progressive degradation of the odor threshold. As a result, workers with frequent or long-term occupational exposures to chlorine are at greater risk of inhalational damage in later years (Urbanetti, 1997).

Chlorine itself is not flammable, but it can react explosively or form explosive compounds with other chemicals such as turpentine and ammonia.

Exposure

The risk for exposure depends on proximity to the place where the chlorine was released.

If chlorine gas is released into the air, exposure can occur through inhalation, dermal contact or eye contact. If chlorine liquid is released into water or food, exposure can occur through ingestion or dermal contact.

Chlorine gas is heavier than air; it settles in low-lying areas.

Adverse Health Effects of Chlorine Exposure

The extent of poisoning caused by chlorine is dependent on the amount of exposure, route of exposure, and length of time of the exposure. Chlorine gas is irritating and corrosive to the eyes, skin, and respiratory tract. Exposure to chlorine may cause burning of the eyes, nose, and throat; cough as well as constriction and edema of the airway and lungs can occur (ASTDR, 2004).

The characteristic initial complaint of chlorine exposure is that of suffocation: the inability to get enough air. Typically, low exposures produce a rapid-onset ocular irritation with nasal irritation, followed shortly by spasmodic coughing and a rapidly increasing choking sensation. Substernal tightness is noted early. Complaints are particularly evident in individuals who have a history of asthma or other hyperreactive airway conditions. Minimal to mild cyanosis may be evident during exertion, and complaints of exertional dyspnea are prominent. Deep inspiration produces a typical persistent, hacking cough.

Moderate chlorine exposures result in an immediate cough and a choking sensation. Severe substernal discomfort and a sense of suffocation develop early. Hoarseness or aphonia is often seen, and stridor may follow. Symptoms and signs of pulmonary edema may appear within 2 to 4 hours; radiological changes typically lag behind the clinical symptoms. There may be retching and vomiting, and the gastric contents often have a distinctive odor of chlorine. Pulmonary edema without significant cardiomegaly, severe resting dyspnea, diffuse crackles on auscultation, and very low blood oxygenation levels (PO_2 of 32 mm Hg breathing room air) are not uncommon (Urbanetti, 1997).

Intense toxic inhalant exposures may cause pulmonary edema within 30 to 60 minutes. Secretions from both the nasopharynx and the tracheobronchial tree are copious, with quantities of up to 1 L/ h reported. Severe dyspnea is so prominent that the patient may refuse to move. On physical examination, the chest may be hyperinflated. Mediastinal emphysema secondary to peripheral air trapping may dissect to the skin and present as subcutaneous emphysema. The sudden death that occurs with massive toxic inhalant exposure is thought to be secondary to laryngeal spasm (Urbanetti, 1997).

Management of Exposure

Leave the area where the chlorine was released and get to fresh air. Quickly moving to an area where fresh air is available is highly effective in reducing exposure to chlorine.

If the chlorine release was outdoors, move away from the area where the chlorine was released. Go to the highest ground possible, because chlorine is heavier than air and will sink to low-lying areas. If the chlorine release was indoors, get out of the building.

Quickly remove all clothing containing liquid chlorine. Any clothing that has to be pulled over the head should be cut off the body. Clothing should be sealed in a plastic bag, double bagging whenever possible.

When helping others remove clothing, avoid touching any contaminated areas; remove the clothing as quickly as possible.

Immediately wash the skin with large amounts of soap and water. If the eyes are burning or if vision is blurred, rinse the eyes with plain water for 10 to 15 minutes. If contact lenses are used, remove them before rinsing your eyes, and then discard them in the bags with the contaminated clothing. Do not put the contacts back into the eyes; they should be discarded even if they are not disposable contacts. If eyeglasses are worn, wash them with soap and water; they can be worn after decontamination.

If chlorine is ingested, **do not induce vomiting** or drink fluids.

Treatment

There is no chemically specific prophylactic or postexposure therapy for chlorine inhalation; therefore, postexposure therapy is directed toward treating the observed physiological signs and symptoms.

Most deaths occur within the first 24 hours and are caused by respiratory failure. Individuals who survive a single, acute exposure generally demonstrate little or no long-term pathological or physiological sequelae. Individuals with underlying cardiopulmonary disease or those who suffer complications (such as pneumonia) during therapy are at risk for developing chronic bronchitis or (rarely) a gradual and progressive bronchiolitis obliterans. Chronic bronchitis was thought to be common after World War I chlorine inhalant exposures. Current assessment of these gassed individuals suggests that their chronic or progressive illness is more likely to have resulted from a combination of inadequately treated complicating infections and cigarette smoking than from the destructive effects of a single, acute exposure (Urbanetti, 1997).

Secretions are typically copious but generally thin; mucolytics are not required. Careful attention to the appearance of secretions will assist in the early identification of bacterial superinfection, which may be associated with secretions that are other than clear or white. Bacterial superinfection is commonly noted 3 to 5 days postexposure. Antibiotic therapy should be initiated early and aggressively and should be directed as specifically as possible against identified organisms. Persistent fever, infiltrates, or elevated white blood cell count in the presence of thickened, colored secretions should prompt the institution of a broad-spectrum antibiotic (such as ampicillin or a cephalosporin). The choice of antibiotic should be based on local experience with either community-acquired or nosocomial organisms. Antibiotics are not used prophylactically in this setting; such therapy would only serve to select a resistant bacterial population in the injured individual (Urbanetti, 1997).

Bronchospasm is an early and prominent complication of chlorine exposure. Aggressive bronchodilator therapy (a combination of adrenergic agent and theophylline) is appropriate. Steroids are used if the patient has a history of hyperreactive airways. Bronchodilators are used at least until the antibiotics are discontinued and there is no further evidence of clinical response (e.g., as indicated by laboratory testing). Steroid doses should be tapered as rapidly as clinical circumstances warrant after the first three to four days of (uncomplicated) recovery. Superinfection may complicate prolonged steroid therapy (Urbanetti, 1997).

As bronchospasm improves, so does the hypoxia; generally long-term oxygen supplementation is not needed. If it is needed, other causes of hypoxia should be investigated. Early institution of positive airway pressure (such as using a PEEP mask) may be useful. Positive pressure ventilation may be necessary if PEEP is insufficient to maintain PO_2 greater than 60 mm Hg. Occasional reports of subcutaneous emphysema after chlorine exposure should not be considered a contraindication to using PEEP or positive pressure ventilation (Urbanetti, 1997).

Recovery from acute injury generally occurs within 36 to 72 hours, depending on the degree of exposure. Delay in recovery may be the result of superinfection. Pleural effusions of up to 600 mL have been identified, generally in association with pulmonary edema. Areas of pneumonic consolidation may be evident on the chest radiograph. Long-term or multiple, low-dose toxic inhalant exposures appear to produce no significant physiological defects when the results are corrected for smoking (Urbanetti, 1997).

The toxic effects of chlorine in the absence of superinfection are relatively short lived. Bronchospasm may require prolonged therapy, occasionally with steroids. There is little evidence for significant long-term pathophysiological abnormalities with either acute, severe chlorine exposure or repetitive low-dose, long-term exposures. A patient's failure to demonstrate substantial recovery within 3 to 4 days should prompt an investigation for the possible presence of bacterial superinfection or other complicating features (Urbanetti, 1997).

Phosgene

Phosgene, as opposed to phosgene oxime, is another pulmonary agent. It is designated by the US military as "CG". It has been used in warfare and appears as a white cloud whose density is due, in part, to hydrolysis. The gas is heavier than air and at low concentrations has a characteristic odor of newly mown hay. However, the characteristic odor cannot be used a reliable guide to phosgene exposure as, accommodation may occur and at higher concentrations, a more acrid, pungent odor may be noted (USAMRICD, 1999). Synonyms for phosgene include carbonyl chloride, D-Stoff, and green cross (Urbanetti, 1997).

Adverse Effects of Phosgene Exposure

Pulmonary agents are absorbed almost exclusively by inhalation. Because they are gases, they readily penetrate to the level of the respiratory bronchioles and the alveoli (USAMRICD, 1999).

Phosgene is an irritant to the skin, eyes, and respiratory tract; there may be minimal irritation immediately after exposure, but delayed damage may be severe (ATSDR, 2004).

Common initial symptoms include mild irritation of the eyes and throat, with some coughing, choking, feeling of tightness in the chest, nausea and occasional vomiting, headache, and lacrimation (ATSDR, 2004).

Phosgene poisoning may cause respiratory and cardiovascular failure, which results from low plasma volume, increased hemoglobin concentration, low blood pressure, and an accumulation of fluid in the lungs. Secondary systemic damage is the result of anoxia (ATSDR, 2004).

The most prominent symptom following the clinical latent period is dyspnea, perceived as shortness of breath, with or without chest tightness. These sensations reflect hypoxemia, increased ventilatory drive, and decreased lung compliance, all of which result from the accumulation of fluid in the pulmonary interstitium and peripheral airways. Fine crackles appear at the lung bases, but these may not be clearly audible unless auscultation is conducted after a forced expiration. Later, auscultation reveals coarse crackles and râles in all lung fields, and increasing quantities of thin, watery secretions are noted. The buildup of fluid in the lungs has two clinically pertinent effects. First, developing pulmonary edema interferes with oxygen delivery to alveolar capillaries and may lead to hypoxemia, and if a sufficient percentage of hemoglobin is unoxygenated, cyanosis will become apparent. Secondly, the sequestration of plasma-derived

fluid (up to one liter per hour) in the lungs may lead to hypovolemia and hypotension, interfering with oxygen delivery to the brain, kidneys, and other crucial organs (USAMRICD, 1999).

In the first 12 hours after toxic inhalant exposure, depending on the intensity of exposure, a substernal tightness with moderate resting dyspnea and prominent exertional dyspnea become evident. Pulmonary edema may develop next. Initially small, then greater, amounts of thin airway secretions may appear. The delayed and insidious onset of severe pulmonary edema often has resulted in a casualty's being medically evaluated and discharged from the medical facility, only to return some hours later with severe and occasionally lethal pulmonary edema. An individual may remain relatively asymptomatic for up to 72 hours after inhalant exposure. During that time, dyspnea or pulmonary edema may be triggered by exertion (Urbanetti, 1997).

Death results from respiratory failure, hypoxemia, hypovolemia, or a combination of these factors. Hypoxia and hypotension may progress particularly rapidly and suggest a poor prognosis. The development of symptoms and signs of pulmonary edema within four hours of exposure is an especially accurate indicator of a poor prognosis; in the absence of immediately available intensive medical support, such patients are at high risk of death. Complications include infection of damaged lungs and delayed deaths following such respiratory infections (USAMRICD, 1999).

Treatment

Terminating exposure is a vital first measure. This may be accomplished by physically removing the exposed person from the contaminated environment or by isolating him or her from surrounding contamination by supplying a properly fitting mask. Decontamination of the liquid agent on clothing or skin terminates exposure from that source (USAMRICD, 1999).

There is no antidote for phosgene. Treatment consists of support of respiratory and cardiovascular functions (ATSDR, 2004).

Execute the ABCs of resuscitation as required. Establishing an airway is especially crucial in a patient exhibiting hoarseness or stridor; such individuals may face impending laryngeal spasm and require intubation. Establishing a clear airway also aids in interpretation of auscultatory findings. Steps to minimize the work of breathing must be taken. Because of the always present danger of hypotension induced by pulmonary edema or positive airway pressure, accurate determination of the casualty's circulatory status is vital not just initially, but also at regularly repeated intervals and whenever indicated by the clinical situation (USAMRICD, 1999).

Enforce rest. Even minimal physical exertion may shorten the clinical latent period and increase the severity of respiratory symptoms and signs in an organohalide casualty, and physical activity in a symptomatic patient may precipitate acute clinical deterioration and even death. Strict limitation of activity (i.e., forced bed rest) and litter evacuation are mandatory for patients suspected of having inhaled any of the edematogenic agents. This is true whether or not the patient has respiratory symptoms and whether or not objective evidence of pulmonary edema is present (USAMRICD, 1999).

Pulmonary edema is the most serious clinical aspect of phosgene exposure and begins with few, if any, clinical signs. Consequently, early diagnosis of pulmonary edema requires that careful attention be paid to the patient's symptoms of dyspnea or chest tightness. The presence of these symptoms in a setting of possible inhalant exposure requires expeditious auscultation, chest radiograph, and arterial blood gas (ABG) measurements (Urbanetti, 1997).

If abnormal, these measurements mandate close observation and support at the intensive care level. If the measurements are normal, they all must be repeated four to six hours after the suspected exposure; only then can an individual be released to a lower medical priority status. Abnormality of any one of those measures, in the absence of other explanation, should signal the institution of therapy for noncardiac pulmonary edema. At the early stages of treatment, therapy should include positive airway pressure with early application of the PEEP mask. Later application of positive pressure ventilation through intubation may be required if the PEEP mask fails to maintain adequate arterial PO_2 (Urbanetti, 1997).

Diuretics may be of minor value, although this can lead to intravascular volume reduction which can lead to serious hypotension if positive pressure ventilation is required. Steroids have not been found to be clinically useful in treating phosgene-induced pneumonitis. Some reports of the use of hexamethamine tetramine have been made, however the therapeutic value is not known (Urbanetti, 1997).

The toxic effects of phosgene in the absence of superinfection or other complications are relatively short-lived. There is little evidence of significant long-term pathophysiological abnormalities with either acute, severe phosgene exposures or repetitive, low-dose, long-term exposures. A patient's failure to demonstrate substantial recovery within three to four days should prompt an investigation for the possible presence of bacterial superinfection or other complicating features (Urbanetti, 1997).

The following "rapid response card" was developed by the NYSDOH and can be downloaded from http://www.health.state.ny.us/nysdoh/bt/chemical_terrorism/chemical.htm.

Table 5: RECOGNIZING AND DIAGNOSING HEALTH EFFECTS OF CHEMICAL TERRORISM

Agent Type	Agent Names	Any Unique Characteristics	Initial Effects
Nerve	- Cyclohexyl sarin (GF) - Sarin (GB) - Soman (GD) - Tabun (GA) - VX	- Miosis (pinpoint pupils) - Copious secretions - Muscle twitching/ fasciculations	- Miosis (pinpoint pupils) - Blurred/dim vision - Headache - Nausea, vomiting, diarrhea - Copious secretions/ sweating - Muscle twitching/ fasciculations - Breathing difficulty - Seizures
Asphyxiant/Blood	- Arsine - Cyanogen chloride - Hydrogen cyanide	- Possible cherry red skin - Possible cyanosis - Possible frostbite*	- Confusion - Nausea - Patients may gasp for air, similar to asphyxiation but more abrupt onset - Seizures prior to death
Choking/ Pulmonary-damaging	- Chlorine - Hydrogen chloride - Nitrogen oxides - Phosgene	- Chlorine is a greenish-yellow gas with pungent odor - Phosgene gas smells like newly-mown hay or grass - Possible frostbite*	- Eye and skin irritation - Airway irritation - Dyspnea, cough - Sore throat - Chest tightness
Blistering/ Vesicant	- Mustard/Sulfur mustard (HD, H) - Mustard gas (H) - Nitrogen mustard (HN-1, HN-2,HN-3) - Lewisite (L) - Phosgene oxime (CX)	- Mustard (HD) has an odor like burning garlic or horseradish - Lewisite (L) has an odor like penetrating geranium - Phosgene oxime (CX) has a pepperish or pungent odor	- Severe irritation - Redness and blisters of the skin - Tearing, conjunctivitis, corneal damage - Mild respiratory distress to marked airway damage - May cause death
Incapacitating/ Behavior-altering	- Agent 15/BZ	- May appear as mass drug intoxication with erratic behaviors, shared realistic and distinct hallucinations, disrobing and confusion - Hyperthermia - Mydriasis (dilated pupils)	- Dry mouth and skin - Initial tachycardia - Altered consciousness, delusions, denial of illness, belligerence - Hyperthermia - Ataxia (lack of coordination) - Hallucinations - Mydriasis (dilated

			pupils)
*Frostbite may occur from skin contact with liquid arsine, cyanogen chloride or phosgene.			

The following “rapid response card” was developed by the NYSDOH and can be downloaded from http://www.health.state.ny.us/nysdoh/bt/chemical_terrorism/chemical.htm.

Table 6: DECONTAMINATION AND TREATMENT

Agent Type	Decontamination	First Aid Assess ABCs	Other Patient Considerations
Nerve	- Remove clothing immediately - Gently wash skin with soap and water - Do not abrade skin - For eyes, flush with plenty of water or normal saline	- Atropine before other measures - Pralidoxime (2-PAM) chloride	- Onset of symptoms from dermal contact with liquid forms may be delayed - Repeated antidote administration may be necessary
Asphyxiant/ Blood	- Remove clothing immediately if no frostbite* - Gently wash skin with soap and water - Do not abrade skin - For eyes, flush with plenty of water or normal saline	- Rapid treatment with oxygen - For cyanide, use antidotes (sodium nitrite and then sodium thiosulfate)	- Arsine and cyanogen chloride may cause delayed pulmonary edema
Choking/ Pulmonary-damaging	- Remove clothing immediately if no frostbite* - Gently wash skin with soap and water - Do not abrade skin - For eyes, flush with plenty of water or normal saline	- Fresh air, forced rest - Semi-upright position - If signs of respiratory distress are present, oxygen with or without positive airway pressure may be needed - Other supportive therapy, as needed	- May cause delayed pulmonary edema, even following a symptom-free period that varies in duration with the amount inhaled
Blistering/ Vesicant	- Immediate decontamination is essential to minimize damage - Remove clothing immediately - Gently wash skin with soap and water - Do not abrade skin - For eyes, flush with plenty of water	- Immediately decontaminate skin - Flush eyes with water or normal saline for 10-15 minutes - If breathing difficulty, give oxygen - Supportive care	- Possible pulmonary edema - Mustard has an asymptomatic latent period - There is no antidote or treatment for mustard - Lewisite has immediate burning pain, blisters later

	or normal saline		- Specific antidote British Anti-Lewisite (BAL) may decrease systemic effects of Lewisite - Phosgene oxime causes immediate pain
Incapacitating/ Behavior-altering	- Remove clothing immediately - Gently wash skin with water or soap and water - Do not abrade skin	- Remove heavy clothing - Evaluate mental status - Use restraints as needed - Monitor core temperature carefully - Supportive care	- Hyperthermia and self-injury are largest risks - Hard to detect because it is an odorless and non-irritating substance - Possible serious arrhythmias - Specific antidote (physostigmine) may be available
*For frostbite areas, do NOT remove any adhering clothing. Wash area with plenty of warm water to release clothing.			

References for Preparedness and Response Card:

1. Agency for Toxic Substances and Disease Registry (ATSDR). 2001. Managing Hazardous Materials Incidents Vol. I, II, III. Division of Toxicology, U. S. Department of Health and Human Services. Public Health Service: Atlanta, GA. **http://www.atsdr.cdc.gov/mhmi.html**

2. Chemical Casualty Care Division USAMRICD. 2000. Medical Management of Chemical Casualties Handbook, Third edition. U.S. Army Medical Research Institute of Chemical Defense (USAMRICD). Aberdeen Proving Ground: Aberdeen, MD. **http://ccc.apgea.army.mil/products/handbooks/RedHandbook/001TitlePage.htm**

3. U.S. Army Edgewood Research, Development and Engineering Center. 1999. Technician EMS Course. Domestic Preparedness Training Program, Version 8.0. U.S. Army SBCCOM. Aberdeen Proving Ground: Aberdeen, MD.

Incapacitating Agents

Incapacitating agents are those that are not necessarily “weapons of mass destruction,” but cause incapacitation, allowing for more standard military interventions to be utilized without retaliation from those exposed to the incapacitating agent

Reports of incapacitating agents extend back to 1000 BC, when burning cauldrons of hemp, which contain tetrahydrocannabinol or smoke from opium, which contains morphine alkaloids were used to incapacitate warring factions (Ketchum and Sidell, 1997).

Irritant agents, including lacrimators (such as CN, the original tear gas, and CS, its more potent successor), are generally fairly effective and safe when properly used. Their drawbacks are twofold: (1) their duration of action is relatively brief (adaptation to the chemical insult usually occurs after less than 30 min of continuous exposure) and (2) highly motivated individuals can fight through (ie, ignore) their effects. Thus, agents of this type would be relatively ineffective against dispersed, well-trained troops, but have application to chemical terrorist threats.

Nausea-producing agents (e.g., DM) may have substantial effectiveness but they can be toxic. Highly potent relatives of apomorphine (a well-known emetic) are known but they have rather low safety margins.

Psychochemical agents are those drugs that have psychological or behavioral impact. They are often sub-categorized as stimulants, depressants, psychedelics and deliriants. They exert their effects on the central nervous system to induce incapacitation. They impact the higher functions of the brain, such as attention, orientation, perception, memory, motivation, conceptual thinking, planning, and judgment. Intelligent behavior can be disabled at much lower doses than that that have lethal effect. They can impact behavior, but are not generally seen as military weapons.

Anticholinergic agents such as atropine and scopolamine have been used to incapacitate warring factions. Historically, they were obtained from of the botanical family Solanaceae, which includes Jimson (or loco) weed, mandrake root, henbane, belladonna, and nightshade.

Of the known psychochemical options, anticholinergics appear to be the most feasible for military use. 3-Quinuclidinyl benzilate (BZ) or a related potent glycolate seem to be the most likely candidates among the many that have been studied. Following an absorbed dose of less than 1 mg, BZ produces an acute brain syndrome, best described as delirium, which lasts 2 to 3 days.

Delirium, in its mildest form, is exhibited as a drowsy state, with occasional lapses of attention and slight difficulty following complex instructions. Moderate delirium generally is manifested by somnolence or mild stupor, indistinct speech, poor coordination, and a generalized slowing in thought process, with some confusion and perplexity.

When delirium is present in its full-blown state, the individual seems to be in a “waking dream,” often staring and muttering, sometimes shouting, as simple items in the environment are variably perceived as elaborate structures, animals, or people. These hallucinations may arise from some trivial aspect of the surroundings, such as a strip of molding, a pillow, or an irregular spot on the floor. A total lack of insight generally surrounds these misperceptions.

Another striking characteristic of delirium is its fluctuation from moment to moment, with occasional lucid intervals and appropriate responses. An individual might answer “Shakespeare” when asked who wrote *Hamlet*, but when asked the same question 5 minutes later, might get down on the floor and attempt to remove an imaginary manhole cover, or become absorbed in a miniature World Series game being played out before his eyes. “Phantom” behaviors, such as plucking or picking at the air or at garments, is characteristic (whence the old term “woolgathering”) (Ketcham & Sidell, 1997).

Reversal of the effects of BZ by physostigmine and other anticholinesterase agents has been demonstrated. Incapacitation produced by less likely candidates such as LSD and other indole derivatives, psychedelic phenethylamines, and potent opioids is theoretically possible, but it is unlikely that any of these compounds would be employed militarily. Covert use however is logistically easier to accomplish and has fewer constraints (Ketcham and Sidell, 1997).

Additional Preparedness Interventions

Sheltering in Place

Some kinds of chemical attacks may make going outdoors dangerous. Leaving the area not be feasible; it may take too long or put people in harm’s way. In such a case it may be safer to stay indoors than to go outside.

“Sheltering in place” is the term for making the interior space, or building as safe as possible in order to protect oneself until the danger subsides or until help arrives.

A room in the house or apartment should be identified as the shelter. The best room to use for the shelter is a room with as few windows and doors as possible. A large room, preferably with a

water supply, is desirable—something like a master bedroom that is connected to a bathroom. For chemical events, this room should be as high in the structure as possible to avoid vapors (gases) that sink. This guideline is different from the sheltering-in-place technique used in tornadoes and other severe weather, when the shelter should be low in the home.

Although the timing of a chemical attack may not coincide when much of the population is at home, the federal government recommends creating a shelter in the home. The following items should be on hand, and should be stored within the shelter:

- First aid kit – According to the US Department of Homeland Security, the following items should be included in a first aid kit (http://ready.gov/first_aid_kit.html):

  Things you should have:

  - Two pairs of Latex, or other **sterile gloves** (if you are allergic to Latex).
  - **Sterile dressings** to stop bleeding.
  - **Cleansing agent**/soap and antibiotic towelettes to disinfect.
  - **Antibiotic ointment** to prevent infection.
  - **Burn ointment** to prevent infection.
  - **Adhesive bandages** in a variety of sizes.
  - **Eye wash solution** to flush the eyes or as general decontaminant.
  - **Thermometer**
  - **Prescription medications** you take every day such as insulin, heart medicine and asthma inhalers. You should periodically rotate medicines to account for expiration dates.
  - **Prescribed medical supplies** such as glucose and blood pressure monitoring equipment and supplies.

  Things it may be good to have:

  - Cell Phone
  - Scissors
  - Tweezers
  - Tube of petroleum jelly or other lubricant

Non-prescription drugs:

- Potassium Iodide
- Aspirin or nonaspirin pain reliever
- Anti-diarrhea medication
- Antacid (for upset stomach)
- Syrup of Ipecac (use to induce vomiting if advised by the Poison Control Center)
- Laxative
- Activated charcoal (use if advised by the Poison Control Center)
- Food and bottled water. One gallon of water per person in plastic bottles as well as ready-to-eat foods that will keep without refrigeration should be stored at the shelter-in-place location. If bottled water no longer is available, water in a toilet tank (*not* the toilet bowl) is suitable for drinking.
- Flashlight, battery-powered radio, and extra batteries for both.
- Duct tape and scissors.
- Towels and plastic sheeting.

- A working telephone.
- Important family and personal documents.

A more detailed discussion of items that are useful to have in any emergency situation, including the possibility of a chemical weapons threat, is available from The Federal Emergency Management Agency's (FEMA) emergency preparedness information at http://www.fema.gov/areyouready/.

How to Know if Sheltering in Place is Needed

If the US Office of Homeland Security issues a "Code Red" or "Severe" terror alert, listening to radio and television broadcasts will provide information about what the population should do, including whether a shelter-in-place alert is announced for their area.

If people are away from their shelter-in-place location when a chemical event occurs, they should follow the instructions of emergency coordinators to find the nearest shelter. If children are at school, they will be sheltered there. Unless instructed to do so, parents should not try to get to the school to bring their children home.

What to Do

Act quickly and follow the instructions of their local emergency coordinators. Every situation can be different, so local emergency coordinators might have special instructions to follow. In general, do the following:

- Go inside as quickly as possible.
- *If there is time*, shut and lock all outside doors and windows. Locking them may provide a tighter seal against the chemical. Turn off the air conditioner or heater. Turn off all fans, too. Close the fireplace damper and any other place that air can come in from the outside.
- Go in the shelter-in-place room and shut the door.
- Tape plastic over any windows in the room. Use duct tape around the windows and doors and make an unbroken seal. Use the tape over any vents into the room and seal any electrical outlets or other openings. Sink and toilet drain traps should have water in them (you can use the sink and toilet as you normally would). Push a wet towel up against the crack between the door and the floor to seal it. If it is necessary to drink water, drink the stored water, not water from the tap.
- Turn on the radio. Keep a telephone close at hand, but don't use it unless there is a serious emergency.

Sheltering in this way should keep people safer than if they are outdoors. They will most likely not be in the shelter for more than a few hours. People should listen to the radio for an announcement indicating that it is safe to leave the shelter.

Evacuation

Some kinds of chemical accidents or attacks may make staying put dangerous. In such cases, it may be safer for people to evacuate, or leave the immediate area. People may need to go to an emergency shelter after they leave the immediate area.

How to Know if Evacuation is Necessary

Local police, emergency coordinators, or government sources on the radio and/or television will notify the public if evacuation is needed.

If there is a "code red" or "severe" terror alert, people should pay attention to radio and/or television broadcasts so they will know right away if an evacuation order is made for their area.

What to Do

Actions must be taken quickly and instructions of emergency coordinators must be followed. Every situation can be different, so local coordinators could have special instructions that need to be followed.

Local emergency coordinators may direct people to evacuate homes or offices and go to an emergency shelter. If so, emergency coordinators will tell people how to get to the shelter. If children are in school, they may be sheltered at the school. Parents should not try to get to the school.

The shelter will have most needed supplies. The emergency coordinators will inform the public of which supplies to bring with them. People should be sure to bring any medications they are taking.

Whenever possible, if there is sufficient time, those evacuated should call a friend or relative in another state to inform them where they are going and that they are safe. Local telephone lines may be jammed in an emergency, so people should plan ahead to have an out-of-state contact with whom to leave messages. People who do not have private transportation should make plans in advance of an emergency to identify people who can provide a ride.

Evacuating and sheltering in this way should keep people safer than if they stayed at home or at their workplace. People will most likely not be in the shelter for more than a few hours. Emergency coordinators will let people know when it is safe to leave the shelter.

For more information about evacuation during a chemical emergency, see “Facts about Evacuation” at http://www.bt.cdc.gov/planning/evacuationfacts.asp. For more information about sheltering in place during a chemical emergency, see “Facts about Sheltering in Place” at http://www.bt.cdc.gov/planning/Shelteringfacts.asp

Resources

The National Center for Environmental Health created Emergency Room Preparation Procedures in Chemical Hazard Emergencies: A Job Aid. (November 2002). This resource is located below:

Preparations

1. Try to determine agent identity.

2. Break out personal protection equipment, decontamination supplies, antidotes, etc.

3. Is chemical hazard certain or very likely?

   YES:
   - •Don personal protective equipment.
   - •Set up hot line.

4. Clear and secure all areas which could become contaminated.

5. Prepare to or secure hospital entrances and grounds.

6. Notify local emergency management authorities if needed.

7. If chemical is a military agent and Army has not been informed, call them.

8. If an organophosphate is involved, notify hospital pharmacy that large amounts of atropine and 2-PAM may be needed.

When victim arrives
(Note: A contaminated patient may present at an emergency room without prior warning.)

9. Does chemical hazard exist?

- Known release/exposure (including late notification)
- Liquid on victim's skin or clothing
- Symptoms in victim, EMTs, others
- Odor (H, L, phosgene, chlorine)
- M-8 paper, if appropriate

YES: Go to 10.

NO: Handle victim routinely.

10. Hold victim outside until preparations are completed (don personal protective equipment to assist EMT's as necessary).

11. If patient is grossly contaminated (liquid on skin, positive M-8 paper) OR if there is any suspicion of contamination, decontaminate patient before entry into building.

Initial Treatment and Identification of the Chemical Agent

1. Establish airway if necessary.
2. Give artificial respiration if not breathing.
3. Control bleeding if hemorrhaging.
4. Symptoms of cholinesterase poisoning?

- Pinpoint pupils
- Difficulty breathing (wheezing, gasping, etc)
- Local or generalized sweating
- Fasciculations
- Copious secretions
- Nausea, vomiting, diarrhea
- Convulsions
- Coma

YES: Go to NERVE AGENT PROTOCOL

5. History of chlorine poisoning?

YES: Go to CHLORINE PROTOCOL.

6. Burns that began within minutes of poisoning?

YES: Go to 7.

NO: Go to 8.

7. Thermal burn?

YES: Go to 9.

NO: Go to LEWISITE PROTOCOL

8. Burns or eye irritation beginning 2-12 hours after exposure?

YES: Go to MUSTARD PROTOCOL.

NO: Go to 9.

9. Is phosgene exposure possible?

- Known exposure to phosgene
- Known exposure to hot chlorinated hydrocarbons
- Respiratory discomfort beginning a few hours after exposure

YES: Go to PHOSGENE PROTOCOL.

10. Check other possible chemical exposures:

- Known exposure
- Decreased level of consciousness without head trauma.
- Odor on clothes or breath
- Specific signs or symptoms

PHOSGENE PROTOCOL

1. Restrict fluids, chest x-ray, blood gases

Results consistent with phosgene poisoning?
YES: Go to # 4

2. Dyspnea?

YES: OXYGEN, positive end-expiratory pressure

3. Observe closely for at least 6 hours.

- IF SEVERE DYSPNEA develops, go to 4.
- IF MILD DYSPNEA

MUSTARD PROTOCOL

1. Airway obstruction?

YES: Tracheostomy

2. If there are large burns:

- Establish IV line - do not push fluids as for thermal burns.
- Drain vesicles - unroof large blisters and irrigate area with tropical antibiotics.

3. Treat other symptoms appropriately:

develops after several hours, go to 1.

4. Severe dyspnea develops or x-ray or blood gases consistent with phosgene poisoning-

- Admit
- Oxygen under positive end-expiratory pressure
- Restrict fluids
- Chest x-ray
- Blood gases
- Seriously ill list

- Antibiotic eye ointment
- Sterile precautions prn
- Morphine prn (generally not needed in emergency treatment; might be appropriate for in-patient treatment.)

LEWISITE PROTOCOL

1. Survey extent of injury.

2. Treat affected skin with British Anti-Lewisite (BAL) ointment (if available).

3. Treat affected eyes with BAL ophthalmic ointment (if available).

4. Treat pulmonary/severe effects

- BAL in oil, 0.5 ml/25 lbs body wt. deep IM to max of 4.0 ml. Repeat q 4 h x 3 (at 4, 8, and 12 hours).
- Morphine prn

5. Severe poisoning?

YES: Shorten interval for BAL injections to q 2 h.

CHLORINE PROTOCOL

1. Dyspnea?

- Try bronchodilators
- Admit
- Oxygen by mask
- Chest X-ray

2. Treat other problems and reevaluate (consider phosgene).

3. Respiratory system OK?

YES: Go to 5.

4. Is phosgene poisoning possible?

YES: Go to PHOSGENE PROTOCOL.

5. Give supportive therapy; treat other problems or discharge.

NERVE AGENT PROTOCOL

1. Severe respiratory distress?
YES:

- Intubate and ventilate
- ATROPINE
  Adults: 6 mg IM or IV
  Inf/ped: 0.05 mg/kg IV
- 2-PAM C1
  Adults: 600-1000 mg IM or slow IV
  Inf/ped: 15 mg/kg slow IV

2. Major secondary symptoms?
NO: Go to 6.

YES:

- ATROPINE
  Adults: 4 mg IM or IV
  Inf/ped: 0.02 - 0.05 mg/kg IV
- 2-PAM C1
  Adults: 600-1000 mg IM or slow IV
  Inf/ped: 15 mg/kg
- OPEN IV LINE

3. Repeat atropine as needed until secretions decrease and breathing easier
Adults: 2 mg IV or IM
Inf/ped: 0.02 - 0.05 mg/kg IV

4. Repeat 2-PAM C1 as needed
Adults: 1.0 gm IV over 20-30 min
Repeat q lh x 3 prn
Inf/ped: 15 mg/kg slow IV

5. Convulsions?

NO: Go to 6.
YES: DIAZEPAM 10 mg slow IV
Inf/ped: 0.2 mg/kg IV

6. Reevaluate q 3-5 min.
IF SIGNS WORSEN, repeat from 3.

Note: Warn the hospital pharmacy that unusual amounts of atropine and 2-PAM may be needed

National:

The Center for Disease Control and Prevention www.bt.cdc.gov/

Federal Emergency Management Agency www.fema.gov

Joint Commission on Accreditation of Healthcare Organizations www.jointcommission.org

Occupational Safety and Health Administration www.osha.gov

Agency for Toxic Substances and Disease Registry www.atsdr.cdc.gov/

- Public health and emergency personnel may contact the ATSDR Emergency Response Center 24 hours a day at 1-404-498-0120.
- Further information about other hazardous substances may be obtained by visiting the ATSDR ToxFAQs™ (www.atsdr.cdc.gove/toxfaq.html) which provide answers to the most frequently asked questions about hazardous substances.
- You may also call the ATSDR Information Center at 1-888-422-8737.

American Red Cross www.redcross.org

American Public Health Association www.apha.org

Association of Emergency Managers www.nemaweb.org

World Health Organization www.who.org

New York State:

New York State Department of Health (NYSDOH)	
Bureau of Toxic Substance Assessment	**518-402-7800**
Wadsworth Center Laboratories	**518-474-7161**
After hours: NYSDOH Duty Officer	**1-866-881-2809**
After hours: SEMO State Warning Point (SEMO - State Emergency Management Office)	**518-457-2200**
New York City Department of Health	
Poison Control Center	**212-764-7667**
Your County Health Department	
For your local health department go to the website at http://www.nysacho.org/Directory/directory.html	
Poison Control Centers	**1-800-222-1222**

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Appendix 1

Patient Information

The following patient information sheets have been developed by The Agency for Toxic Substances and Disease Registry (ATSDR) (2001), accessed at http://www.atsdr.cdc.gov/mmg.html.

Nerve Agents Patient Information Sheet

This handout provides information and follow-up instructions for persons who have been exposed to nerve agents.

What are nerve agents?
Nerve agents are chemical warfare agents, similar to but much more potent than organophosphate insecticides. They are colorless to amber-colored, tasteless liquids that may evaporate to create a gas. GB and VX are odorless, while GA has a slight fruity odor, and GD has a slight camphor odor.

What immediate health effects can result from exposure to nerve agents?
Nerve agents are extremely toxic chemicals that attack the nervous system. As little as one drop to a few milliliters of nerve agent contacting the skin can cause death within 15 minutes. Nerve agent exposure can cause runny nose, sweating, blurred vision, headache, difficulty breathing, drooling, nausea, vomiting, muscle cramps and twitching, confusion, convulsions, paralysis, and coma. Symptoms occur immediately if you inhale nerve agent vapor but may be delayed for several hours if you get nerve agent liquid on your skin.

Can nerve agent poisoning be treated?
There are antidotes for nerve agent poisoning but they must be administered quickly after exposure. Immediate decontamination is critical and hospitalization may be needed.

Are any future health effects likely to occur?
Complete recovery may take several months. After a severe exposure with prolonged seizures, permanent damage to the central nervous system is possible.

What tests can be done if a person has been exposed to nerve agents?
Activity of a blood enzyme called acetylcholinesterase can be measured to assess exposure and recovery.

Where can more information about nerve agents be found?
More information about nerve agents can be obtained from your regional poison control center; the Agency for Toxic Substances and Disease Registry (ATSDR); your doctor; or a clinic in your area that specializes in toxicology or occupational and environmental health. Ask the person who gave you this form for help locating these telephone numbers.

Follow-up Instructions
Keep this page and take it with you to your next appointment. Follow *only* the instructions checked below.

[ ] Call your doctor or the Emergency Department if you develop any unusual signs or symptoms within the next 24 hours, especially:

- dizziness, loss of coordination, loss of memory
- coughing, wheezing, or shortness of breath
- nausea, vomiting, cramps, or diarrhea
- muscle weakness or twitching
- blurred vision

[ ] No follow-up appointment is necessary unless you develop any of the symptoms listed above.

[ ] Call for an appointment with Dr._____ in the practice of _____. When you call for your appointment, please say that you were treated in the Emergency Department at _____ Hospital by_____and were advised to be seen again in ____days.

[ ] Return to the Emergency Department/Clinic on _____(date) at _____AM/PM for a follow-up examination.

[ ] Do not perform vigorous physical activities for 1 to 2 days.

[ ] You may resume everyday activities including driving and operating machinery.

[ ] Do not return to work for _____days.

[ ] You may return to work on a limited basis. See instructions below.

[ ] Avoid exposure to cigarette smoke for 72 hours; smoke may worsen the condition of your lungs.

[ ] Avoid drinking alcoholic beverages for at least 24 hours; alcohol may worsen injury to your stomach or have other effects.

[ ] Avoid taking the following medications:
_____

[ ] You may continue taking the following medication(s) that your doctor(s) prescribed for you:
_____

[ ] Other instructions:
_____

- Provide the Emergency Department with the name and the number of your primary care physician so that the ED can send him or her a record of your emergency department visit.
- You or your physician can get more information on the chemical by contacting: _____or_____, or by checking out the following Internet Web sites: _____;_____.

Signature of patient _____Date _____

Signature of physician _____ Date _____

Appendix 2 Blister Agents Sulfur Mustard (H, HD, and HT) Patient Information Sheet

Blister Agents Lewisite and Mustard-Lewisite Mixture Information Sheet

This handout provides information and follow-up instructions for persons who have been exposed to nerve agents.

What are Lewisite and Mustard-Lewisite Mixture?
Lewisite is a chemical warfare agent that was first produced in 1918. It has not been used in warfare, although it may be stockpiled by some countries. Mustard-Lewisite Mixture is a mixture of Lewisite and Mustard. It was developed to achieve a lower freezing point for ground dispersal and aerial spraying.

What immediate health effects can be caused by exposure to Lewisite and Mustard-Lewisite Mixture?
Lewisite and Mustard-Lewisite Mixture produce pain and skin irritation immediately after exposure. Both compounds cause skin blisters and damage to the airways and eyes. They are also extremely irritating to the eyes, skin, nose, and throat. Exposure to very high levels may result in kidney and liver damage. Mustard-Lewisite Mixture can also damage the immune system.

Can Lewisite and Mustard-Lewisite poisoning be treated?
Immediate decontamination reduces symptoms. Intramuscular injection of British Anti-Lewisite (BAL) may be used to treat severe conditions but will not prevent lesions on the skin, eye, or airways. Persons who have been exposed to large amounts of Lewisite and Mustard-Lewisite Mixture will need to be hospitalized.

Are any future health effects likely to occur?
Adverse health effects, such as chronic respiratory diseases, may occur from exposure to high levels of these agents. Severe damage to the eye may be present for a long time after the exposure.

What tests can be done if a person has been exposed to Lewisite or Mustard-Lewisite?
There is no specific test to confirm exposure to Lewisite or Mustard-Lewisite Mixture; however, measurement of arsenic in the urine may help to identify exposure.

Where can more information about Lewisite or Mustard-Lewisite be found?
More information about Lewisite and Mustard-Lewisite Mixture can be obtained from your regional poison control center; the Agency for Toxic Substances and Disease Registry (ATSDR); your doctor; or a clinic in your area that specializes in toxicology or occupational and environmental health. Ask the person who gave you this form for help locating these telephone numbers.

Follow-up Instructions
Keep this page and take it with you to your next appointment. Follow *only* the instructions checked below.

[ ] Call your doctor or the Emergency Department if you develop any unusual signs or symptoms within the next 24 hours, especially:

- coughing, wheezing, shortness of breath, or discolored sputum
- increased pain or discharge from injured eyes
- increased redness, pain, or a pus-like discharge from injured skin; fever; or chills

[ ] No follow-up appointment is necessary unless you develop any of the symptoms listed above.

[ ] Call for an appointment with Dr._____ in the practice of .
When you call for your appointment, please say that you were treated in the Emergency Department at _____ Hospital by_____and were advised to be seen again in ____days.

[ ] Return to the Emergency Department/Clinic on _____(date) at _____AM/PM for a follow-up examination.

[ ] Do not perform vigorous physical activities for 1 to 2 days.

[ ] You may resume everyday activities including driving and operating machinery.

[ ] Do not return to work for _____days.

[ ] You may return to work on a limited basis. See instructions below.

[ ] Avoid exposure to cigarette smoke for 72 hours; smoke may worsen the condition of your lungs.

[ ] Avoid drinking alcoholic beverages for at least 24 hours; alcohol may worsen injury to your stomach or have other effects.

[ ] Avoid taking the following medications:
_____

[ ] You may continue taking the following medication(s) that your doctor(s) prescribed for you:
_____
_____

[ ] Other instructions: _____
_____
_____

- Provide the Emergency Department with the name and the number of your primary care physician so that the ED can send him or her record of your emergency department visit.
- You or your physician can get more information on the chemical by contacting: _____or_____, or by checking out the following Internet Web sites: _____;_____.

Signature of patient _____Date _____

Signature of physician _____ Date _____

Appendix 3 Blister Agents Sulfur Mustard (H, HD, and HT) Patient Information Sheet

This handout provides information and follow-up instructions for persons who have been exposed to sulfur mustard.

What are sulfur mustards?
Sulfur mustards are yellowish to brown liquids that have been used as chemical warfare agents since 1917.

What immediate health effects can result from exposure to sulfur mustards?
Sulfur mustards produce blistering and cell damage, but symptoms are delayed for hours. They cause damage to the skin, eyes, and respiratory tract. The eyes are the most sensitive. Nausea and vomiting may occur within the first few hours after exposure. Skin rashes, blisters, and lung damage may develop within a few hours of exposure but may take 12 to 24 hours to develop. Sulfur mustard can also suppress the immune system.

Can sulfur mustard poisoning be treated?
There is no antidote for sulfur mustard, but its effects can be treated and most exposed people recover. Immediate decontamination reduces symptoms. People who have been exposed to large amounts of sulfur mustard will need to be treated in a hospital.

Are any future health effects likely to occur?
Adverse health effects, such as chronic respiratory diseases, may occur from exposure to high levels of these agents. Severe damage to the eyes and skin may be present for a long time following the exposure.

What tests can be done if a person has been exposed to sulfur mustards?
There are no routine tests to determine if someone has been exposed to sulfur mustard. Thiodiglycol (a break-down product of mustard) may be detected in the urine up to 2 weeks following exposure; however, this test is available only in several specialized laboratories.

Where can more information about sulfur mustards be found?
More information about sulfur mustard can be obtained from your regional poison control center; the Agency for Toxic Substances and Disease Registry (ATSDR); your doctor; or a clinic in your area that specializes in toxicology or occupational and environmental health. Ask the person who gave you this form for help locating these telephone numbers.

Follow-up Instructions
Keep this page and take it with you to your next appointment. Follow *only* the instructions checked below.

[ ] Call your doctor or the Emergency Department if you develop any unusual signs or symptoms within the next 24 hours, especially:

- coughing, wheezing, shortness of breath, or discolored sputum
- increased pain or discharge from injured eyes
- increased redness, pain, or a pus-like discharge from injured skin
- fever or chills

[ ] No follow-up appointment is necessary unless you develop any of the symptoms listed above.

[ ] Call for an appointment with Dr._____ in the practice of _____.
When you call for your appointment, please say that you were treated in the Emergency Department at _____ Hospital by_____and were advised to be seen again in ____days.

[ ] Return to the Emergency Department/Clinic on _____(date) at _____AM/PM for a follow-up examination.

[ ] Do not perform vigorous physical activities for 1 to 2 days.

[ ] You may resume everyday activities including driving and operating machinery.

[ ] Do not return to work for _____days.

[ ] You may return to work on a limited basis. See instructions below.

[ ] Avoid exposure to cigarette smoke for 72 hours; smoke may worsen the condition of your lungs.

[ ] Avoid drinking alcoholic beverages for at least 24 hours; alcohol may worsen injury to your stomach or have other effects.

[ ] Avoid taking the following medications:
_____

[ ] You may continue taking the following medication(s) that your doctor(s) prescribed for you:
_____
_____

[ ] Other instructions: _____
_____
_____

- Provide the Emergency Department with the name and the number of your primary care physician so that the ED can send him or her a record of your emergency department visit.

- You or your physician can get more information on the chemical by contacting: _____or_____, or by checking out the following Internet Web sites: _____;_____.

Signature of patient _____ Date _____

Signature of physician _____ Date _____

Appendix 4 Hydrogen Cyanide Patient Information Sheet

This handout provides information and follow-up instructions for persons who have been exposed to hydrogen cyanide.

What is hydrogen cyanide?
At room temperature, hydrogen cyanide is a volatile, colorless-to-blue liquid (also called hydrocyanic acid). It rapidly becomes a gas that can produce death in minutes if breathed. Hydrogen cyanide is used in making fibers, plastics, dyes, pesticides, and other chemicals, and as a fumigant to kill rats. It is also used in electroplating metals and in developing photographic film.

What immediate health effects can be caused by exposure to hydrogen cyanide?
Breathing small amounts of hydrogen cyanide may cause headache, dizziness, weakness, nausea, and vomiting. Larger amounts may cause gasping, irregular heartbeats, seizures, fainting, and even rapid death. Generally, the more serious the exposure, the more severe the symptoms. Similar symptoms may be produced when solutions of hydrogen cyanide are ingested or come in contact with the skin.

Can hydrogen cyanide poisoning be treated?
The treatment for cyanide poisoning includes breathing pure oxygen, and in the case of serious symptoms, treatment with specific cyanide antidotes. Persons with serious symptoms will need to be hospitalized.

Are any future health effects likely to occur?
A single small exposure from which a person recovers quickly is not likely to cause delayed or long-term effects. After a serious exposure, a patient may have brain or heart damage.

What tests can be done if a person has been exposed to hydrogen cyanide?
Specific tests for the presence of cyanide in blood and urine generally are not useful to the doctor. If a severe exposure has occurred, blood and urine analyses and other tests may show whether the brain or heart has been injured. Testing is not needed in every case.

Where can more information about hydrogen cyanide be found?
More information about hydrogen cyanide can be obtained from your regional poison control center; your state, county, or local health department; the Agency for Toxic Substances and Disease Registry (ATSDR); your doctor; or a clinic in your area that specializes in occupational and environmental health. If the exposure happened at work, you may wish to discuss it with your employer, the Occupational Safety and Health Administration (OSHA), or the National Institute for Occupational Safety and Health (NIOSH). Ask the person who gave you this form for help in locating these telephone numbers.

Follow-up Instructions
Keep this page and take it with you to your next appointment. Follow *only* the instructions checked below.

[ ] Call your doctor or the Emergency Department if you develop any unusual signs or symptoms within the next 24 hours, especially:

- difficulty breathing, shortness of breath, or chest pain
- confusion or fainting
- increased pain or a discharge from your eyes
- increased redness, pain, or a pus-like discharge in the area of a skin burn

[ ] No follow-up appointment is necessary unless you develop any of the symptoms listed above.

[ ] Call for an appointment with Dr._____in the practice of _____.

When you call for your appointment, please say that you were treated in the Emergency Department at _____ Hospital by_____and were advised to be seen again in ____days.

[ ] Return to the Emergency Department/Clinic on _____(date) at _____AM/PM for a follow-up examination.

[ ] Do not perform vigorous physical activities for 1 to 2 days.

[ ] You may resume everyday activities including driving and operating machinery.

[ ] Do not return to work for _____days.

[ ] You may return to work on a limited basis. See instructions below.

[ ] Avoid exposure to cigarette smoke for 72 hours; smoke may worsen the condition of your lungs.

[ ] Avoid drinking alcoholic beverages for at least 24 hours; alcohol may worsen injury to your stomach or have other effects.

[ ] Avoid taking the following medications:
_____

[ ] You may continue taking the following medication(s) that your doctor(s) prescribed for you:
_____
_____

[ ] Other instructions: _____
_____
_____

- Provide the Emergency Department with the name and the number of your primary care physician so that the ED can send him or her a record of your emergency department visit.
- You or your physician can get more information on the chemical by contacting: _____or_____, or by checking out the following Internet Web sites: _____;_____.

Signature of patient _____Date _____

Signature of physician _____ Date _____

Chemical Terror Threats-Are You Prepared?
Course Exam

1. Healthcare providers prepare for potential chemical weapons attacks, at least in part, by increasing their knowledge and competence in the recognition and treatment of chemical exposures, while safeguarding one's own health.

    A. True.

    B. False

2. Chemical weapons threats are a product of the industrial and technological age.

    A. True.

    B. False

3. Chemical weapons threats are categorized by their adverse effects and include all the following EXCEPT:

    A. Vesicants/Blistering agents

    B. Nerve agents

    C. Gastrointestinal

    D. Pulmonary agents

4. Since January 1, 2003, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requires:

    A. That healthcare organizations have a comprehensive, functional emergency plan that is coordinated with community resources and is drilled at least twice annually.

    B. That healthcare organizations have the ability to test air quality in order to identify when a chemical release has occurred.

    C. That healthcare providers become active in agencies such as the American Red Cross, in order to prepare for potential emergencies.

    D. That healthcare providers obtain prophylactic treatment for potential chemical weapons threats.

5. The New York State Department of Health has identified clues that may help healthcare providers identify that there has been a chemical weapons release. These include:

    A. A sharp increase in the public's willingness to receive prophylactic treatment for chemical weapons threat should be seen as indicative of possible chemical release.

    B. Any unusual clustering of symptoms regarding time, location and age of victims, with simultaneous impact on human, animal and plant life.

    C. The delayed onset of symptoms in the population for at least 5-7 days to 2-3 weeks.

    D. Presentation severe respiratory symptoms and high fever in persons who have recently traveled to mainland China.

6. Personal protective equipment needed in the event of a chemical release, when the exact chemical is not known include all the following EXCEPT:

    A. Level A personal protective equipment

    B. Powered air purifying respirators (PAPR)

    C. Neoprene, Viton, nitrile, or butyl gloves

    D. A surgical mask

7. Proper decontamination is frequently the most important intervention for exposed persons. General decontamination guidelines include:

    A. Immediate removal of clothing (up to 90 % of the chemical can be removed by doing so) and double bagging all personal effects.

    B. Immediately wash the skin with mild soap and water, being careful not to abrade the skin; the water should be contained as it is likely contaminated.

    C. Eyes should be flushed with large amounts of water or normal saline; any contact lenses should be removed, if doing so will not further damage the eyes.

    D. All of the above.

8. Which of the chemical weapons threats are considered to be the most toxic and rapidly acting?

    A. Incapacitating agents

    B. Pulmonary agents

    C. Nerve agents

    D. Vesicants/Blistering agents

9. Antidotes for exposure to nerve agents include:

    A. Atropine and pralidoxime

    B. BAL

    C. Sodium nitrate

    D. Sodium thiosulfate

10. Characteristic symptoms of nerve agent exposure include all of the following EXCEPT:

    A. Miosis and copious secretions

    B. Pulmonary edema

    C. Respiratory Distress

    D. Muscle fasciculations and flaccid paralysis

11. Typically there is a delay in the onset of symptoms after exposure to vesicants or blistering agents, for as long as 24 hours.

    A. True.

    B. False

12. Symptoms of vesicants/blistering agents include all the following EXCEPT:

    A. Erythema and blistering in a classic “string of pearls” formation around the erythematous areas of the skin, progressing to 1^{st} to 3^{rd} degree burns.

    B. Intense conjunctival and scleral pain, swelling, lacrimation, blepharospasm, and photophobia.

    C. Characteristic cherry red skin color.

    D. Burning nasal pain, epistaxis, sinus pain, laryngitis, loss of taste and smell, cough, wheezing and dyspnea.

13. Most of the chemicals that can potentially be used as chemical weapons are heavier than air, so they settle in low-lying areas, making movement to higher ground important if exposure occurs. The exception is:

    A. Sarin

    B. Chlorine

    C. Cyanide

    D. Phosgene oxime

14. All the following is true of the British Anti-Lewisite (BAL) antidote, for exposure to lewisite EXCEPT:

    A. Is a chelating agent that should be used only by trained personnel.

    B. Should be used only on exposed persons who are in shock or who have significant pulmonary injury because of the antidote's toxic side effects.

    C. Consultation with the regional Poison Control Center is recommended.

    D. There are no contraindications for the use of BAL antidote.

15. Phosgene oxime is classified as a vesicant/blistering agent, but is often considered to be a corrosive agent because it causes significant tissue damage. A hallmark symptom of phosgene oxime exposure is

    A. Copious secretions.

    B. Intense pain.

    C. Neurological symptoms.

    D. High fever.

16. Secondary exposure of healthcare providers to victims of cyanide exposure can occur through the following methods EXCEPT:

    A. Off-gassing from contaminated clothing.

    B. The vomitus of exposed persons

    C. Moving to an area with fresh air, away from the cyanide release.

    D. Direct dermal contact.

17. The initial complaint of person exposed to chlorine is a feeling of suffocation, and hacking cough with inspiration. Pulmonary edema can occur within the hour if the exposure was severe.

    A. True.

    B. False

18. Immediate treatment for exposure to phosgene is:

    A. Rapid administration of atropine and frequent suctioning.

    B. Thorough decontamination utilizing a highly diluted bleach solution to cleanse the skin.

    C. Enforced bed rest whether or not symptoms of pulmonary edema are present.

    D. There is no known effective treatment for phosgene exposure.

19. Incapacitating agents are those that deliver an unwanted effect on the victim, not necessarily to kill, but to incapacitate her or him in order to utilize more traditional military interventions.

    A. True.

    B. False

20. Sheltering in place requires that the individual identify a room in the home, rather than at the workplace, to serve as a safe location during a potential chemical release. The room should have the following:

    A. It should be located on the highest level possible within the home.

    B. Have little or no direct access to outside air; plastic sheeting and duct tape can be used to seal the room's doors and windows, if applicable.

    C. Contain first aid kit, water and food supplies, prescription medications and important family documents.

    D. All of the above.