Appendix C
Chemical Agents

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FACT SHEET

Facts About Personal Cleaning and Disposal of Contaminated Clothing

Some kinds of chemical accidents or attacks may cause you to come in contact with dangerous chemicals. Coming in contact with a dangerous chemical may make it necessary for you to remove and dispose of your clothing right away and then wash yourself. Removing your clothing and washing your body will reduce or remove the chemical so that it is no longer a hazard. This process is called decontamination.

People are decontaminated for two primary reasons:

1. to prevent the chemical from being further absorbed by their body or from spreading on their body, and
2. to prevent the chemical from spreading to other people, including medical personnel, who must handle or who might come in contact with the person who is contaminated with the chemical.

Most chemical agents can penetrate clothing and are absorbed rapidly through the skin. Therefore, the most important and most effective decontamination for any chemical exposure is decontamination done within the first minute or two after exposure.

How to know if you need to wash yourself and dispose of your clothing

In most cases, emergency coordinators will let you know if a dangerous chemical has been released and will tell you what to do.

In general, exposure to a chemical in its liquid or solid form will require you to remove your clothing and then thoroughly wash your exposed skin. Exposure to a chemical in its vapor (gas) form generally requires you only to remove your clothing and the source of the toxic vapor.

If you think you have been exposed to a chemical release, but you have not heard from emergency coordinators, you can follow the washing and clothing disposal advice in the next section.

What to do

Act quickly and follow the instructions of local emergency coordinators. Every situation can be different, so local emergency coordinators might have special instructions for you to follow. The three most important things to do if you think you may have been exposed to a dangerous chemical are to (1) quickly remove your clothing, (2) wash yourself, and (3) dispose of your clothing. Here's how:

- *Removing your clothing:*
  - Quickly take off clothing that has a chemical on it. Any clothing that has to be pulled over your head should be cut off instead of being pulled over your head.
  - If you are helping other people remove their clothing, try to avoid touching any contaminated areas, and remove the clothing as quickly as possible.
- *Washing yourself:*

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  - As quickly as possible, wash any chemicals from your skin with large amounts of soap and water. Washing with soap and water will help protect you from any chemicals on your body.
  - If your eyes are burning or your vision is blurred, rinse your eyes with plain water for 10 to 15 minutes. If you wear contacts, remove them and put them with the contaminated clothing. Do not put the contacts back in your eyes (even if they are not disposable contacts). If you wear eyeglasses, wash them with soap and water. You can put your eyeglasses back on after you wash them.
- *Disposing of your clothes:*
  - After you have washed yourself, place your clothing inside a plastic bag. Avoid touching contaminated areas of the clothing. If you can't avoid touching contaminated areas, or you aren't sure where the contaminated areas are, wear rubber gloves or put the clothing in the bag using tongs, tool handles, sticks, or similar objects. Anything that touches the contaminated clothing should also be placed in the bag. If you wear contacts, put them in the plastic bag, too.
  - Seal the bag, and then seal that bag inside another plastic bag. Disposing of your clothing in this way will help protect you and other people from any chemicals that might be on your clothes.
  - When the local or state health department or emergency personnel arrive, tell them what you did with your clothes. The health department or emergency personnel will arrange for further disposal. Do not handle the plastic bags yourself.

After you have removed your clothing, washed yourself, and disposed of your clothing, you should dress in clothing that is not contaminated. Clothing that has been stored in drawers or closets is unlikely to be contaminated, so it would be a good choice for you to wear.

You should avoid coming in contact with other people who may have been exposed but who have not yet changed their clothes or washed. Move away from the area where the chemical was released when emergency coordinators tell you to do so.

How you can get more information about personal cleaning and disposal of contaminated clothing

You can contact one of the following:

- State and local health departments
- Centers for Disease Control and Prevention (CDC)
  - Public Response Hotline (CDC)
    - English (888) 246-2675
    - Español (888) 246-2857
    - TTY (866) 874-2646
  - Emergency Preparedness and Response Web site (http://www.bt.cdc.gov/)
  - E-mail inquiries: cdcresponse@ashastd.org
  - Mail inquiries:
    Public Inquiry c/o BPRP
    Bioterrorism Preparedness and Response Planning
    Centers for Disease Control and Prevention
    Mailstop C-18

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This fact sheet is based on CDC's best current information. It may be updated as new information becomes available.

Last reviewed on 05/22/03.

The Centers for Disease Control and Prevention (CDC) protects people's health and safety by preventing and controlling diseases and injuries; enhances health decisions by providing credible information on critical health issues; and promotes healthy living through strong partnerships with local, national, and international organizations.

For more information, visit www.bt.cdc.gov or call the CDC public response hotline at (888) 246-2675 (English), (888) 246-2857 (Español), or (866) 874-2646 (TTY)

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FACT SHEET
Chlorine

Military Designation: None

Description: Chlorine is found as an amber liquid or greenish-yellow gas with a very characteristic irritating, pungent odor. Chlorine is severely irritating to the skin, eyes, and respiratory tract. Although generally stored as a liquid, when released, the resulting gas is approximately two times heavier than air.

Non-military Uses: Chlorine is used widely in industrial settings in the organic synthesis and manufacture of antifreeze agents, solvents, refrigerants, resins, bleaching agents, and other inorganic chemicals. There is an exceptionally wide use of chlorine in noncommercial and home settings as a cleaning agent, bleaching agent, bacteriostatic, and disinfecting agent. Storage of this substance in a variety of liquid and granular forms
is widespread.

Military Use: Chlorine was first used by the German military on April 22, 1915 in a cylinder-released gas attack that resulted in an estimated 15,000 Allied wounded and 5000 Allied deaths. Because of its tendency to dissipate rapidly, very large concentrations were required. Chlorine was weaponized in projectiles, mortars, and bombs. There is no current chlorine weaponry.

Health Effects: Chlorine exposure causes an immediate severe irritation to the eyes and mucous membranes. The upper airways are first involved with nose, throat, and sinus irritation. The lower airways are irritated with severe cough and chest pain. There may be nausea, vomiting, and fainting. Very high doses may cause excess fluid to develop in the lungs (pulmonary edema). Wheezing respiration is likely to occur in individuals with previous asthma. Bronchitis often occurs, sometimes progressing to pneumonia. Chronic exposures may increase the susceptibility to respiratory infections. High concentrations also irritate the skin, causing burning, itching, and occasional blister formation. There is no
animal or human epidemiologic data suggesting that chronic chlorine exposure may cause cancer or the occurrence of adverse developmental effects in the unborn fetus.

Environmental Fate: Chlorine is not persistent in surface water, ground water, or soil. Oxidation of environmental organic materials occurs rapidly, reducing its concentration rapidly. Dispersal of chlorine gas is rapid to the atmosphere.

TREATMENT PROTOCOL
Chlorine

1. General:

Chlorine is found as a greenish-yellow gas. There is a pungent, acrid, characteristic odor. Sensitivity to the odor is below toxic levels; however, since some sensory adaptation occurs, repeat exposures are more likely to produce toxic effects. Exposures irritate eyes and central (upper) airways within minutes. Low doses produce some cough and choking sensation. Moderate doses also produce a sense of suffocation, hoarseness, and substernal pain. High doses also produce a severe dyspnea, with pulmonary edema, nausea, vomiting, headache, and syncope. Very high doses may produce sudden death without obvious pulmonary lesions—possibly via laryngospasm. All recognized exposures should be referred for direct observation/care.

2. Patient Evaluation:

a. Victim should be immediately removed from the toxic environment by fully masked personnel. Chemically protective clothing is required for liquid/solution exposures.

b. Liquid contamination causes eye and skin burns on contact. Contaminated clothing should be removed and properly disposed.

3. Treatment:

a. Eyes: Liquid exposures should be flushed with copious quantities of water; medical attention should be sought. Gas exposures, if symptomatic, should be flushed with water. Medical attention should be sought if symptomatic.

b. Skin: Liquid exposures should be flushed with copious quantities of water. Contaminated clothing should be removed and disposed. Gas exposures require no specific therapy unless symptomatic. Intense gas exposure produces burns; wash with water.

c. Breathing: Evaluate respiration, cyanosis, and bronchospasm.

If apneic: CPR with intubation. Be aware that laryngospasm may be present with intense exposures; hence intubation may be very difficult and tracheostomy could be required. Medical attention should be sought.

If stridorous/hoarse: Consider intubation under direct vision since laryngospasm may be imminent (see above). Medical attention should be sought.

If dyspnea/cough/chest tightness: Consider intubation for impending pulmonary edema. Also consider possible bronchospasm sufficiently severe to have so little air exchange that wheezes are absent. Medical attention should be sought. Codeine containing demulcents may help. Be wary of sedation.

TREATMENT PROTOCOL
Chlorine Treatment (continued)

Note: *Wheezing is a less reliable indicator of bronchospasm in infants and children due to the anatomical configuration of their airways. Severe smaller airway constriction withresultant hypoxia may be present. Any apparent infant or child distress should beimmediately assessed with oximetry.*

If bronchospasm: Provide aggressive bronchodilation:

Adult:
Inhaled albuterol: unit dose q 2 hr.
Steroids: methylprednisolone, load 120 mg, then 60-mg q 6 hr.
Theophylline: load 150 mg, then 30 mg/hr.

Infants and children (0-12 yr.):
Inhaled albuterol: 0.15 mg/kg per nebulized dose
up to 5 mg/20 minutes for first 2 hr.
Steroids: methylprednisolone: 1 mg/kg q 6 hr.
Theophylline: 10-mg/kg/24 hr.

Elderly:
Inhaled albuterol: unit dose q 3 hr.
Steroids: methylprednisolone, load 125 mg, then 60-mg q 6 hr.
Theophylline (occasional use): load 100 mg, then 25 mg/hr.

If asymptomatic: Maintain direct observation for at least 1 hour.

If becomes symptomatic, treat as above.

If still asymptomatic, monitor for additional 12 hours since some bronchospasm may appear late.

If hypoxic from bronchospasm: Administer bronchodilators and supplemental oxygen.

If pulmonary edema: Treat as noncardiac pulmonary edema (Adult Respiratory Distress Syndrome or ARDS) (e.g., BiPAP, CPAP, or if intubated, PEEP 5-7 cm). Diuretic therapy risks severe hypotension if intubation is required.

If infection: Inhalational exposures may produce pulmonary infiltrates, fever, and white blood cell elevations leading to an erroneous diagnosis of (presumed bacterial) pneumonia. Prophylactic antibiotics are not indicated. Surveillance bacteriologic cultures are obtained anticipating an approximate 50% risk of nosocomial pneumonia at days 3-6.

If pain: Airway discomfort may benefit from codeine. Be wary of sedation.

FACT SHEET
Hydrocyanic Acid - Hydrogen Cyanide and Cyanogen Chloride

Military Designations: AC (hydrocyanic acid) and CK (cyanogen chloride)

Description: Both of these substances are liquids, but they vaporize (evaporate) at approximately 73°F and 58°F, so they will be in the gaseous form under most temperate conditions. AC has an odor of bitter almonds; CK is pungent. AC vapor is lighter than air, whereas CK gas is heavier than air. Cyanogen chloride is quickly metabolized to cyanide once absorbed into the body, and causes the same biological effects as hydrogen cyanide. In addition, CK is irritating to the eyes, nose, and throat (similar to riot control agents), whereas AC is nonirritating.

Non-military Uses: Large amounts of cyanide (most in the form of salts) are produced, transported, and used by U.S. industry annually. Cyanide is used in fumigation, photography, and extraction of metals, electroplating, metal cleaning, tempering of metals, and the synthesis of many compounds. It is released when synthetic fibers and plastics burn.

Military Uses: The French and the English used small amounts of cyanide during World War I, but the compound was not effective as a weapon because the amount needed is large (and small munitions were used) and because cyanide, being lighter than air, drifted away from the target. Japan allegedly used cyanide against China before World War II, and Iraq allegedly used cyanide against the Kurds in 1988. The U.S. once had cyanide munitions, but the known ones have been destroyed. However, some of these munitions may have been abandoned at sites around the U.S. Small amounts of cyanogen chloride
were incorporated in chemical agent identification sets, which were also abandoned.

Health Effects: Cyanide blocks the use of oxygen in cells of the body and thus causes asphyxiation in each cell. The cells of the brain and the heart are most susceptible to an oxygen deficit. High concentrations of vapor may cause a brief increase in rate and depth of breathing (in 15 seconds), seizures (30 seconds), cessation of breathing (3-5 minutes) and of cardiac activity (4-10 minutes), and death. A smaller concentration will cause headache, flushing, light-headedness, and other nonspecific effects. (In addition, CK produces irritation of the eyes, the nose, and the airways.) Antidotes (nitrites and thiosulfate) are very effective if administered in time. A large exposure may result in prolonged neurologic damage, probably because of hypoxia. Chronic ingestion of cyanidecontaining
foods (e.g., cassava, which is a staple in many parts of Africa) has been
associated with thyroid and nerve disturbances. Evidence does not suggest that cyanides are carcinogenic.

Environmental Fate: Because of their volatility, these substances are not expected to persist in surface water or soil.

TREATMENT PROTOCOL
Hydrogen Cyanide and Cyanogen Chloride

1. General:

a. Patient should be removed from the toxic environment immediately.

b. These substances are very volatile so there is little need for decontamination if exposure was to vapor alone. If liquid was present, remove patient's clothing and wash liquid off skin.

c. The effects of vapor from either form of cyanide appear within seconds to a minute. If patient has no or only mild effects when seen 5 to 30 minutes after exposure, he/she will need no treatment.

d. Severe cyanide poisoning produces metabolic acidosis. If cyanide poisoning is suspected in a patient who does not have moderate or severe acidosis, treatment for cyanide poisoning should not be delayed, but the diagnosis should be reconsidered.

2. Patient Evaluation: level of consciousness, respiratory rate, and heart rate.

a. Exposure to a high concentration: transient hyperpnea, followed by convulsions (30 seconds after exposure), gradual decrease in respiratory rate and depth to apnea (3-5 minutes) and cessation of cardiac activity (5-8 minutes).

b. Exposure to low concentration: flushing, headache, anxiety, agitation, vertigo, feeling of weakness, nausea, muscular trembling (cyanogen chloride may cause irritation of eyes, nose, and airways). Prolonged exposure may lead to effects listed above.

c. Odor of bitter almonds may be detected (half of the population cannot smell this); normal pupils (may be dilated in terminal stage); "cherry-red" skin (may not be present); diaphoresis; venules in fundus are same color as arterioles; cyanosis occurs only after circulatory collapse and apnea.

3. Treatment:

a. For a mild exposure (conscious and breathing): observe; no antidotes; oxygen may be given to adult or pediatric patients in the presence of a patient experiencing the mild symptoms of heart disease.

b. Severe exposure (unconscious, not breathing): should immediately receive 100% oxygen. Cardiac monitoring and evaluation of oxygen saturation should be done when possible. (Saturation will be normal even in cases of severe cyanide exposure until the terminal stage; however, additional oxygen may assist in therapy.) Antidotes should be administered as soon as possible (see below). It is important to note that pulse oximeter results are completely unreliable in the setting of methemoglobinemia, which is induced by amyl nitrite or sodium nitrite therapy.

c. For a severe exposure: Ventilate using bag-valve-mask with one ampule of amyl nitrite (crushed) in bag; after several minutes add another (crushed) ampule; keep adding an ampule every several minutes. This is a temporary measure until IV medications can be given, but it may assist in recovery.

d. Administer 300 mg (10 ml) of sodium nitrite IV over 5 minutes. Flush line. [Children's dose: 0.2-0.3 ml/kg, or 6-9 mg/kg of the 3% solution. No separate recommendation for infants. For elderly, use adult dose unless small and frail.] Be aware: Nitrites produce orthostatic hypertension, but a patient who can stand does not need them.

e. Follow with 12.5 grams (50 ml) of sodium thiosulfate IV. [Children's dose: 0.4 mg/kg, or 1.65 ml/kg of the 25% solution. No separate recommendation for infants. Adult dose should be used for elderly unless they are small and frail. Use care in giving nitrite in a patient with hypertension or heart disease.] (Amyl nitrite, sodium nitrite, and sodium thiosulfate are in the Pasadena (formerly Lilly) Cyanide Antidote Kit, the latter two in ampules of 300 mg/10 ml and 12.5 grams/50 ml.). Use one-half dose in 20 minutes if no improvement. See instructions on top of Antidote Kit box.

f. If patient continues to remain apneic, intubate and continue oxygen through tube with assisted ventilation.

g. Transfer apneic or unconscious patients to medical facility.

h. Patients often recover rapidly unless CNS hypoxia has occurred.

4. Laboratory Issues:

a. Metabolic acidosis is common; should be treated with bicarbonate.

b. Monitor arterial pO2; should be normal until near-terminal stage.

FACT SHEET

Methyl Isocyanate, Methylene Bisphenyl Isocyanate, and Methylene Diisocyanate (MDI)

Military Designations or Military Unique Use: None

Description: Methylene Bisphenyl Isocyanate is found as a solid in white to yellow flakes. Various liquid solutions are used for industrial purposes. There is no odor to the solid or the liquid solutions. The vapor is approximately eight times heavier than air. This chemical is a strong irritant to the eyes, mucus membranes, skin and respiratory tract. This chemical is also a very potent respiratory sensitizer.

Non-military Uses: Very large quantities of MDI are produced, transported, and used annually in the U.S. Various industrial processes utilize MDI in production and usage of (poly)urethane foams, lacquers, and sealants. MDI is a commonly used precursor in the industrial production of insecticides and laminating materials. Noncommercial uses of polyurethanes such as in isocyanate paints or in cutting of uncured urethanes may also cause exposure. Thermal degradation of these substances may produce MDI as a combustion by-product.

Health Effects: MDI as either a solid or liquid solution is a strong irritant to the eyes and the skin, resulting in discomfort and burning sensation. Severe inflammation may occur. Irritation of the respiratory tract results in cough, shortness of breath, and chest pain. Very high concentrations may irritate the respiratory tract sufficiently to cause excess fluid accumulation within the lung, resulting in very severe respiratory distress and pulmonary edema. MDI vapor is a strong sensitizer of the respiratory tract. In some individuals, particularly those with prior history of asthma, repetitive exposures, even to very low doses, may trigger an asthmatic episode. Such sensitized individuals may also experience asthma with subsequent skin or eye exposures. This sensitization may persist indefinitely. Repeated or long-term exposure may result in permanent respiratory problems. Repeated or long-term exposure of the skin may cause a rash. There are no animal or human epidemiologic data that suggest that chronic MDI exposure may cause cancer or the occurrence of adverse developmental effects in the unborn fetus.

Environmental Fate: Since the reported vapor pressure of Methyl isocyanate (MIC) is 348 mm Hg at 20°C, MIC is expected to remain almost entirely in vapor phase when released into the atmosphere. MIC is susceptible to hydrolysis and photooxidation in the atmosphere with a half-life of 11 days at an atmospheric concentration of 5.0E+5 hydroxyl radicals/M3. In the aquatic media, MIC is rapidly hydrolyzed with half-lives of 20 and 9 minutes at 14° and 25°C, respectively. The products of hydrolysis-N-carboxymethylamine, methylamine, carbon dioxide, and N, N'-dimethylurea are nontoxic. Due to its rapid hydrolysis in aqueous media, MIC is not expected to bioconcentrate or bioaccumulate in the environment. MIC released to soil is hydrolyzed and the degradative process is rapid in the presence of moisture. Hydrolysis minimizes adsorption and volatilization of MIC from the soil, although these conditions are favorable for its mobility. Depending upon the concentration of MIC in soil and prevailing moisture conditions, volatilization from the surface soil may be a significant environmental transport and fate process.

TREATMENT PROTOCOL
Methyl Isocyanate, Methylene Bisphenyl Isocyanate, and Methylene Diisocyanate (MDI)

1. General:

MDI is found as a solid, which has a melting point of 37°C. Vapor exposures occur with liquids containing dissolved solid. Gas exposures may occur with high-temperature volatilization. Thermal decomposition produces carbon monoxide and oxides of nitrogen. Sensitivity to this substance (eye, nose irritation) occurs at concentrations five times higher than OSHA limits (0.2 mg/m3); hence toxic exposures may go unrecognized.

Exposures lead to: Irritant effects: Eyes, mucous membranes, and skin may be irritated, particularly with prolonged, repetitive, or intense exposures. High concentrations may also produce cough, dyspnea, and lethal pulmonary edema. Sensitizing effects: Respiratory sensitization may occur, particularly in individuals with known asthma, allergies, or recognized isocyanate sensitivity (e.g., TDI).

2. Patient Evaluation:

The victim should be immediately removed from the toxic environment by personnel in chemically protective clothing. Vapor or gas hazards should be anticipated with full (positive pressure) masks. Liquid/solid contamination should be corrected by clothing removal and soap and water decontamination.

3. Treatment:

a. Eyes: There is no specific therapy appropriate. Liquid/solid exposures should be irrigated with copious quantities of water. Subsequently symptomatic individuals should seek medical attention.

b. Skin: There is no specific therapy appropriate. Liquids/solids should be removed with soap and water. Single exposures are unlikely to create rashes unless the individual was previously sensitized. Intense exposure may produce dermatitis and require referral.

c. Ingested: Liquids/solids should be removed by induced vomiting in the conscious victim or by lavage otherwise.

d. Respiratory: Symptoms due to sensitivity may be delayed up to 8 hr after exposure. Respiratory symptoms may appear with skin, ocular, or GI exposure in previously sensitized individual.
If apneic: Initiate CPR. Intubation may be required for pulmonary edema.
Consider severe bronchospasm in previously sensitized victim.

Methyl Isocyanate, Methylene Bisphenyl Isocyanate, and Methylene Diisocyanate (MDI) (continued)

If stridorous/hoarse: Consider intubation under direct vision.

If dyspnea/cough/chest tightness: Consider intubation for impending pulmonary edema. Also consider possible bronchospasm sufficiently severe to have so little air exchange that wheezes are absent. Medical attention should be sought.

Codeine containing demulcents may help. Be wary of sedation.

Note: *Wheezing is a less reliable indicator of bronchospasm in infants and children due to the anatomical configuration of their airways. Severe smaller airway constriction with resultant hypoxia may be present. Any apparent infant or child distress should be immediately assessed with oximetry.*

If bronchospasm: Treat as asthma with inhaled albuterol. Bronchospasm may be particularly severe, especially in previously sensitized individuals.

Treat aggressively:

Adults:
Inhaled albuterol: unit dose q 2 hr. or continuous neb 15 g/hr.
Steroids: methylprednisolone load 250 mg, then 80-mg q 6 hr.
Theophylline: load 150 mg, then 30-mg/hr.

Infants and children (0-12 yr.):
Inhaled albuterol: 0.15 mg/kg per nebulized dose
up to 5 mg/20 minutes for first 2 hr.
Steroids: methylprednisolone; 1 mg/kg q 6 hr.
Theophylline: 10-mg/kg/24 hr.

Elderly:
Inhaled albuterol: unit dose q 3 hr.
Steroids: methylprednisolone load 125 mg, then 60-mg q 6 hr.
Theophylline (occasional use): load 100-mg then 25 mg/hr.

Upper airway obstruction: This is very rarely seen and only with intense exposure. Hoarseness and stridor suggest impending laryngospasm: Consider intubation under direct vision. If pulmonary edema (may rarely occur with intense exposures): Treat as noncardiac pulmonary edema (Adult Respiratory Distress Syndrome or ARDS see PHOSGENE).

If hypoxia (commonly from bronchospasm, rarely from pulmonary edema): Treat with above bronchodilation and oxygen.

If cough: Codeine-containing demulcents (tissue-soothing agents) may help. Be wary of sedation.

*[**Note:** cough typically indicates inadequately treated bronchospasm.]*

If pain: Airway discomfort from irritant effect may benefit from codeine. Be wary of sedation.

FACT SHEET

Mustard (Sulfur Mustard)

Military Designations: H; HD; HS

Description: Mustard is a "blister agent" that causes cell damage and destruction. It is a colorless to light yellow to dark brown oily liquid with the odor of garlic, onion, or mustard. It does not evaporate readily, and may pose a vapor hazard in warm weather. It is a vapor and liquid hazard to skin and eyes, and a vapor hazard to airways. Its vapor is five times heavier than air.

Non-military Uses: Sulfur mustard has been used as a research tool to study DNA damage and repair. A related compound, nitrogen mustard, was the first cancer chemotherapeutic agent, and is still used for some purposes.
Military Use: Mustard was used extensively in World War I and was the largest chemical casualty producer in that war. Mustard was used by Iraq against Iran in the 1980s. The U.S. has a variety of munitions filled with sulfur mustard, including projectiles, mortars, and bombs. It is also in chemical agent identification sets (which may be on abandoned sites)and in ton containers.

Health Effects: Mustard damages DNA in cells, which leads to cellular damage and death. Mustard penetrates skin and mucous membranes very quickly, and cellular damage begins within minutes. Despite this cellular damage, dinical effects do not begin until hours later; the range is 2 to 24 hours, but usually 4 to 8 hours. The initial effects are in the eyes (itching or burning), the skin (erythema with itching and burning), and airways (epistaxis, hoarseness, sinus pain, cough). After high doses, these may progress to more severe effects in the eyes (corneal damage), skin (blisters), and damage to the lower airways (dyspnea and productive cough). After absorption of a large amount, there may be damage to the gastrointestinal tract (vomiting, diarrhea) and bone marrow (damage to stem cells with cessation of production of white cells, red cells, and platelets). There is no antidote. Epidemiological studies indicate that frequent exposure to mustard over years may cause an increased incidence of cancer of the upper airways. An acute exposure may cause persistent damage to airways (e.g., stenosis) and eyes (keratitis). Animal studies suggest that mustard may have developmental effects.

Environmental Fate: Persistence of mustard in soil will depend on the soil type, the amount of mustard, the depth of contamination, and weather conditions. Mustard contamination of surface soil may persist for weeks, and deeper soil may remain contaminated for years. Mustard is relatively insoluble in water; once dissolved, however, it breaks down into less toxic products. Because of its relatively rapid hydrolysis once in solution, mustard is not thought to be transported through the soil by ground water.

TREATMENT PROTOCOL

Mustard (Sulfur Mustard)

1. General:

a. Mustard causes no immediate effects. The initial clinical effects of mustard (which usually involve the eyes, the skin, and the airways) appear 2 to 24 hours (usually 4 to 8 hours) after exposure to liquid mustard or to mustard vapor. However, liquid or vapor mustard penetrates the skin and mucous membranes and damages cells within minutes of exposure, so decontamination must be done immediately after exposure.

b. The patient should be immediately removed from the toxic environment.

c. If the patient has been exposed to liquid mustard, the clothing should be removed and skin decontaminated with soap and cool water, or thoroughly flushed with water alone. The patient's eyes should be flushed with large amounts of saline. If the patient has been exposed to vapor alone, remove the clothing.

d. If there is a history of definite exposure, the patient should be taken to a medical facility for observation.

2. Patient Evaluation: Initial effects (usually 2 to 24 hours after exposure):

a. Eyes: irritation, feeling of grit in eye, redness.

b. Skin: erythema (will progress to blisters 1 to 4 hours later if exposure was large).

c. **Respiratory:** irritation of nose, voice change, sinus pain, and hacking cough. (Very rarely a patient might inhale an extremely large amount and start to have these effects plus dyspnea within 2 hours. This patient should be intubated, and assisted ventilation with oxygen should be started. This patient should be taken to the nearest pulmonary intensive care unit as quickly as possible).

3. Treatment:

a. There is nothing to do for patients exposed to mustard until effects appear except to decontaminate. Tissue is damaged within minutes, so decontamination must be done immediately.

b. Eyes: Any commercial eye solution may relieve the irritation from a mild exposure.

Mustard (continued)

More severe effects: A mydriatic b.i.d. or q.i.d. (depending on the length of action of the drug); a topical antibiotic b.i.d.; Vaseline on lid edges b.i.d.; sunglasses if photophobia is present. Topical steroids within the first 24 hours may only reduce inflammation. Control pain with systemic, not topical, analgesics. Visual loss is usually due to lid edema and blepharospasm, not eye damage.

c. Skin: A soothing lotion (e.g., calamine) for erythema. Leave small blisters intact. Unroof large blisters and irrigate denuded area at least t.i.d. followed by liberal application of topical antibiotic. Watch for infection. Fluid requirements are much less than those for thermal burns; do not overhydrate.

d. Respiratory: Steam inhalation and cough suppressants will generally relieve mild symptoms. A chemical pneumonitis (increased temperature; white blood count; chest x-ray findings) may develop after large exposure: intubation; assisted ventilation with oxygen (and possibly with PEEP or CPAP); bronchodilators; watch sputum at least daily for organisms (no antibiotics until organism is identified).

e. Systemic absorption of a large amount of mustard may cause bone marrow and gastrointestinal tract damage. Watch WBC, Hct daily; mustard damages bone marrow.

FACT SHEET

Nerve Agents (GA, GB, GD, GF, VX)

Military Designations: GA, GB, GD, GF, and VX
Common Names: Tabun (GA); Sarin (GB); Soman (GD). None for GF and VX.

Description: Nerve agents are very toxic organophosphorus compounds that have biological activity similar to that of many insecticides. Their volatility ranges from that of water to that of motor oil; they present a hazard from vapor and liquid. Under temperate conditions, the liquids are clear, colorless, and mostly odorless. They cause biological effects by inhibiting acetylcholinesterase, thereby allowing acetylcholine to accumulate and cause hyperactivity in muscles, glands, and nerves.

Non-military Use: There is no non-military use. Nerve agents can be found in some research laboratories and storage facilities, and could pose a risk to human populations if used by terrorists.

Military Use: Nerve agents were first synthesized pre-World War II, but were not used in that war. They were used by Iraq in its war with Iran. The U.S. has a large stockpile of GA and VX in weapons; these are being destroyed.

Health Effects: Nerve agents are the most toxic chemical agents. Initial effects from small amounts of a nerve agent differ, depending on the route of exposure. After a small vapor exposure, there is the immediate onset of effects in the eyes (small or pinpoint pupils [miosis], redness, eye pain, and dim vision), the nose (rhinorrhea), and airways (some degree of shortness of breath because of bronchoconstriction and secretions). After a small liquid exposure, there may be an asymptomatic interval of up to 18 hours before the onset of sweating and fasciculations at the site of the droplet, which may be followed by nausea, vomiting, and diarrhea. After exposure to a large amount of nerve agent by either
route, there is sudden loss of consciousness, convulsions, copious secretions, apnea, and death. There is usually an asymptomatic interval of minutes after liquid exposure before these occur; effects from vapor occur almost immediately. Antidotes (atropine and pralidoxime) are effective if administered before circulation fails. There is no evidence that nerve agents cause cancer or developmental effects.

Environmental Fate: GB will react with water to produce toxic vapors. Open-pit burning or burying is prohibited. GB mixes with water and would be mobile in surface and ground water should a release occur; however, because of its rapid hydrolysis, it is not a long-term water contaminant of concern. Most GB spilled will be lost by evaporation; because of this there is no long-term impact on health and environment. VX is moderately persistent in soil, and because it has low water solubility, it could be mobile in surface and ground water systems.

TREATMENT PROTOCOL

Nerve Agents (GA, GB, GD, GF, VX)

1. General:

Nerve agents are extremely toxic chemicals that cause effects by inhibiting the enzyme acetylcholinesterase, allowing excess acetylcholine to accumulate. This excess neurotransmitter then produces overstimulation and causes hyperactivity in muscles, glands, and nerves. The nerve agents are GA (tabun), GB (sarin), GD (soman), GF, and VX. Their effects are identical.

Remove the patient from contaminated atmosphere. If exposure was to vapor, remove clothing; if exposure was to liquid, remove clothing and wash skin with soap and water, or thoroughly flush with water alone.

2. Patient Evaluation:

If the patient is conscious, note ventilatory status and ask about nausea. If the patient is unconscious, note ventilatory status and heart rate (heart rate may be high, low, or normal in a nerve agent casualty).

Initial effects differ depending on whether exposure was to vapor or to liquid.

a. Vapor: Effects start within seconds to a minute or two.

(1) Mild to moderate: Miosis (possible redness in eye, eye pain, complaints of dim or blurred vision, nausea), rhinorrhea, excess secretions, dyspnea (mild to severe).

(2) Severe: Loss of consciousness, seizures, apnea, and flaccid paralysis.

b. Liquid: Effects start in minutes (large exposure) to 18 hours (small exposure) after an asymptomatic interval.

(1) Mild to moderate: Sweating and fasciculations at site of exposure; nausea, vomiting, diarrhea; weakness.

(2) Severe: Same as for vapor, but after a 1- to 30-minute asymptomatic interval.

TREATMENT PROTOCOL

Nerve Agents (GA, GB, GD, GF, VX) (continued)

3. Treatment:

a. Initial Management:

(1) Mild to moderate: Dyspnea should be treated with one or two doses of atropine IM or IV and 1 dose of pralidoxime (IV drip) initially, depending on severity of the dyspnea. (See paragraph b below for size of dose.) This should be supplemented with oxygen, particularly in infants, young children, and the elderly; healthy older children and adults will usually do well without it unless they have pulmonary or cardiac disease.
Atropine dose should be repeated at 7- to 10-minute intervals until improvement is noted. Failure to respond (i.e., no dry mouth, no decrease in secretions) confirms the need to administer additional doses of atropine. Gastrointestinal effects after liquid exposure is treated in the same manner. Do not treat for miosis (unless eye pain is severe) or rhinorrhea (unless severe).

(2) Severe: Administer 3 doses of atropine IM (not IV in hypoxic patient) and start 1 dose of pralidoxime by slow (20 minutes) IV drip. [More rapid administration will cause hypertension.] (See paragraph b below for size of dose.) Intubate and ventilate with oxygen (initial ventilation will be difficult because of airway resistance; atropine will relieve this). Administer diazepam if the patient is convulsing. Suction for secretions. Repeat 1 dose of atropine (IM until hypoxia is improved, then IV) every 5 minutes until (a) secretions diminish or (b) airway resistance is less or is normal. Failure to respond (i.e. no dry mouth, no decrease in secretions) confirms the need to administer additional doses of atropine.
Monitor via pulse oximeter; cardiac monitoring should also be done (cardiac arrhythmias are uncommon after atropine is given). Acidosis may develop after seizures or after period of hypoxia and will require therapy. This patient should be transported to a hospital after stabilization (adequate drug therapy and initiation of ventilation).

(3) Eyes: Do not treat miosis unless eye/head pain is severe. Use topical, not systemic, anticholinergic to relieve pain.

Nerve Agents (GA, GB, GD, GF, VX) (continued)

b. Recommended Doses:

Atropine:
Older child and adult: 2 mg q 5 minutes until secretions dry
Infant and young child: 0.02 mg/kg
Elderly: Use adult dose unless cardiac or pulmonary disease is present or patient is small or frail; in latter instances, use 1 mg as standard, but be prepared to administer additional amounts more frequently.

Pralidoxime:
Older child and adult: 1 gram (If IM 600 mg to 1.2 grams)
Infant and young child: 25-50 mg/kg
Elderly: Adult dose unless cardiac or renal disease is present, patient has hypertension, or patient is small and frail; decrease dose by half in these patients, but administer the other half 1 hour later if patient has not improved. Pralidoxime can cause hypertension when given rapidly by IV. Slow administration over 20 minutes will minimize the hypertensive effect. After rapid
administration, hypertension can be rapidly but transiently reversed by phentolamine (adult: 5 mg IV, child: 1 mg IV).

c. Further Care:

(1) Mild to moderate: After vapor exposure, a patient who is breathing normally does not need to be hospitalized. However, miosis should be followed until the patient's eyes are normal (4 to 6 weeks). After liquid exposure, a patient should be observed in a hospital for 18 hours until all the nerve agent is absorbed from the skin.

(2) Severe: Continue to ventilate the patient and to administer atropine following guidelines above. Treat acidosis if present. If patient has not had prolonged hypoxia, recovery of an unconscious patient will be gradual over 1 to 3 hours.

FACT SHEET

Phosgene — Carbonyl Chloride

Military Designation: CG

Description: Phosgene is a highly reactive halogenated compound. It is found as a colorless liquid or colorless or white (if hydrolysis occurs in air) gas. It has an odor of newly mown or moldy hay. It is primarily a vapor hazard at high concentrations to the upper respiratory tract, with severe irritation; and at lower concentrations, to the lower respiratory tract, with pulmonary edema. Phosgene vapors are heavier than air but are notpersistent.

Non-military Uses: Phosgene is an industrially widely used, extremely important substance for purposes of chemical synthesis. Large quantities are stored and transported within the continental U.S. Materials such as foamed plastics, insecticides, and aniline dyes are products of its use. These substances and many other halogenated hydrocarbons (e.g., carbon tetrachloride, methylene chloride, degreasing agents), if combusted, producephosgene as a degradation byproduct.

Military Use: Phosgene was first used by the Germans as a toxic war gas on December 19, 1915. By some estimates phosgene accounted for 85% of World War I chemical deaths. Phosgene was generally dispersed in combination with other agents (e.g., chlorine) due to its relatively low rate of evaporation from the liquid state.

Health Effects: Phosgene gas at high concentrations may cause immediate irritation of the eyes and upper respiratory tract (nose, larynx, and trachea). This effect is thought to be due to breakdown of the gas to hydrochloric acid with water vapor contact. After resolution of this irritation, a symptom-free period may occur. During this period, progressive damage to the walls of the capillaries allows fluids to leak from those vessels and gradually compromise lung function. The individual complains of progressive cough, chest tightness, and shortness of breath. Frothy secretions typical of pulmonary edema occur. This can be so rapid as to cause death if the early symptoms are not recognized and treated. If recovery is not complicated by infection, permanent lung damage is not likely to occur. There are no recognized long-term health risks from repetitive/chronic lowdose exposure. There are no data suggesting adverse effects on the unborn fetus.

Environmental Fate: Phosgene is not persistent in surface water, ground water, or soil containing moisture because of its rapid breakdown into carbon dioxide and hydrochloric acid. Phosgene is not persistent in dry soil because of its tendency to evaporate readily.

TREATMENT PROTOCOL

Phosgene — Carbonyl Chloride

1. **General:**

   Phosgene may be found as a colorless liquid or a colorless-to-white gas. There is an odor of newly mown or moldy hay. Sensitivity to the odor may degrade, making individuals unaware of toxic inhalation. High-intensity exposure irritates eyes and upper airways within minutes. Lower-dose exposures may produce a lethal pulmonary edema with a characteristic symptom-free or "latent" period up to 48 hours later. Some pulmonary symptoms may appear as late as 72 hours after exposure. All recognized exposures should be referred for direct, in-hospital observation and care.

2. **Patient Evaluation:**

   a. Victim should be immediately removed from the toxic environment by personnel with the appropriate PPE (positive pressure apparatus).

   b. Liquid contamination does not require additional protection for rescue personnel insofar as there are minimal topical effects to the skin and no substantial dermal absorption. Contaminated clothing should be removed.

3. **Treatment:** Maintain at rest at least 6 hours.

   a. Eyes: If exposed to liquid phosgene, eyes should be flushed with copious quantities of water. Medical attention should be sought. Eyes exposed to gas phosgene, if symptomatic, should be flushed with water. Medical attention should be sought if symptomatic.

   b. Skin: Patients exposed to liquid phosgene should be flushed with copiousquantities of water; contaminated clothing should be removed and disposed. Patients exposed to gas phosgene require no specific therapy unless symptomatic.

   c. Ingested: Do not induce vomiting. Medical attention should be sought.

   d. Respiratory: Evaluate respiration, cyanosis. Oxygen should always be used. If apneic: Initiate CPR with intubation. Be aware that laryngospasm may be present with intense exposures; hence, intubation may be very difficult and tracheostomy required. Medical attention should be sought. If stridorous/hoarse: Consider intubation under direct vision since laryngospasm may be imminent (see above). Medical attention should be sought. If dyspnea/cough/chest tightness: Consider intubation for impending pulmonary edema. Also consider possible bronchospasm sufficiently severe to have so little air exchange that wheezes are absent. Medical attention should be sought. Codeine containing demulcents may help. Be wary of sedation. Note: cough may presage pulmonary edema.

TREATMENT PROTOCOL
Phosgene — Carbonyl Chloride (continued)

Note: *Wheezing is a less reliable indicator of bronchospasm in infants and children due to the anatomical configuration of the airways. Severe smaller airway constriction with resultant hypoxia may be present. Any apparent infant or child distress should be immediately assessed with oximetry.*

If bronchospasm: Individuals with underlying asthma may suffer bronchospasm. Treat as any asthmatic: Inhaled albuterol, parenteral steroids, and theophylline. Watch for hypoxia.

Adult:
Inhaled albuterol: unit dose q 2 hr.
Steroids: methylprednisolone, load 120 mg, then 60 mg q 6 hr.
Theophylline: loading dose 5.6 mg/kg, then 30 mg/hr.

Infants and Children (0-12 yr.):
Inhaled albuterol: 0.15 mg/kg per nebulized dose
up to 5 mg/20 minutes for first 2 hr.
Steroids: methylprednisolone: 1 mg/kg q 6 hr.
Theophylline: 10 mg/kg/24 hr.

Elderly:
Inhaled albuterol: unit dose q 3 hr.
Steroids: methylprednisolone, load 125 mg, then 60 mg q 6 hr.
Theophylline (occasional use): load 100 mg, then 25 mg/hr.

If asymptomatic: Maintain direct observation for at least 6 hours;

If patient becomes symptomatic treat as above.

If patient is still asymptomatic after 6 hours, lesser observation is needed for an additional 36 hours.

If hypotensive (will occur rapidly with pulmonary edema): Immediate volume replacement should be undertaken. Colloid or crystalloid may be used to maintain adequate tissue perfusion.

If infection: Inhalational exposures may produce pulmonary infiltrates, fever, and white blood cell elevations, leading to an erroneous diagnosis of (presumed bacterial) pneumonia. Prophylactic antibiotics are not indicated. Surveillance bacteriologic cultures are obtained anticipating an approximate 50% risk of nosocomial pneumonia at days 3-6.

If hypoxia: Commonly from pulmonary edema, treat as above; occasionally from bronchospasm, treat as above.

If pain: Airway discomfort may benefit from codeine. Be wary of sedation.

MEDICATION DOSAGE CHARTS

ATROPINE dosage chart at 0.1 mg/ml drug concentration (0.02mg/kg Pediatric, 2 mg adult)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	1 mL
12 months	10 kg (22 lb)	2 mL
3 years	15 kg (33 lb)	3 mL
6 years	20 kg (44 lb)	4 mL
8 years	25 kg (55 lb)	5 mL
10 years	30 kg (66 lb)	6 mL
11 years	35 kg (77 lb)	7 mL
12 years	40 kg (88 lb)	8 mL
13 years	45 kg (99 lb)	9 mL
14 years or more	50 kg (110 lb) or more	20 mL
Adult	50 kg (110 lb) or more	20 mL

ATROPINE dosage chart at 0.4 mg/ml drug concentration (0.02mg/kg Pediatric, 2 mg adult)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	0.25 mL
12 months	10 kg (22 lb)	0.5 mL
3 years	15 kg (33 lb)	0.75 mL
6 years	20 kg (44 lb)	1 mL
8 years	25 kg (55 lb)	1.25 mL
10 years	30 kg (66 lb)	1.5 mL
11 years	35 kg (77 lb)	1.75 mL
12 years	40 kg (88 lb)	2 mL
13 years	45 kg (99 lb)	2.25 mL
14 years or more	50 kg (110 lb) or more	5 mL
Adult	50 kg (110 lb) or more	5 mL

ATROPINE dosage chart at 1 mg/ml drug concentration (0.02mg/kg Pediatric, 2 mg adult)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	0.1 mL
12 months	10 kg (22 lb)	0.2 mL
3 years	15 kg (33 lb)	0.3 mL
6 years	20 kg (44 lb)	0.4 mL
8 years	25 kg (55 lb)	0.5 mL
10 years	30 kg (66 lb)	0.6 mL
11 years	35 kg (77 lb)	0.7 mL
12 years	40 kg (88 lb)	0.8 mL
13 years	45 kg (99 lb)	0.9 mL
14 years or more	50 kg (110 lb) or more	2 mL
Adult	50 kg (110 lb) or more	2 mL

ATROPINE dosage chart at 2 mg/ml drug concentration (0.02mg/kg Pediatric, 2 mg adult)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	0.05 mL
12 months	10 kg (22 lb)	0.1 mL
3 years	15 kg (33 lb)	0.15 mL
6 years	20 kg (44 lb)	0.2 mL
8 years	25 kg (55 lb)	0.25 mL
10 years	30 kg (66 lb)	0.3 mL
11 years	35 kg (77 lb)	0.35 mL
12 years	40 kg (88 lb)	0.4 mL
13 years	45 kg (99 lb)	0.45 mL
14 years or more	50 kg (110 lb) or more	1 mL
Adult	50 kg (110 lb) or more	1 mL

MEDICATION DOSAGE CHARTS

PRALIDOXIME (2-PAM, Protopam) dosage chart at 50 mg/mL (For IV use) – (50 mg/kg Pediatric, 1000 mg Adult)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	5 mL = 250 mg
12 months	10 kg (22 lb)	10 mL = 500 mg
3 years	15 kg (33 lb)	15 mL = 750 mg
6 years	20 kg (44 lb)	20 mL = 1000mg
8 years	25 kg (55 lb)	20 mL
10 years	30 kg (66 lb)	20 mL
11 years	35 kg (77 lb)	20 mL
12 years	40 kg (88 lb)	20 mL
13 years	45 kg (99 lb)	20 mL
14 years or more	50 kg (110 lb) or more	20 mL
Adult	50 kg (110 lb) or more	20 mL

PRALIDOXIME (2-PAM, Protopam) dosage chart at 300 mg/mL (For IM use) – (40 mg/kg Pediatric, 1000 mg Adult)

(reconstitute by adding 3 ml sterile water to a 1 g vial of pralidoxime)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	0.7 mL
12 months	10 kg (22 lb)	1.3 mL
3 years or more	15 kg (33 lb) or more	2 mL
Adult	50 kg (110 lb) or more	20 mL

MEDICATION DOSAGE CHARTS

AMYL NITRITE dosage chart

For all ages, crush ampule and allow it to be inhaled for up to 3 minutes. If patient is endotracheally intubated, place ampule or some of its contents in the large end of the ET tube where it connects to the bag or ventilator. If amyl nitrite use is to continue beyond 3 minutes, use a new vial approximately every 3 minutes until the patient recovers or until sodium nitrite can be administered. Once venous access is established and sodium nitrite is available, administer sodium nitrite and discontinue use of amyl nitrite as soon as possible.

SODIUM NITRITE dosage chart at 3% (300mg/10 ml) (Pediatric 0.3 ml/kg for Hgb 11 g/dL, Adult 10 ml)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	1.5 mL
12 months	10 kg (22 lb)	3 mL
3 years	15 kg (33 lb)	4.5 mL
6 years	20 kg (44 lb)	6 mL
8 years	25 kg (55 lb)	7.5 mL
10 years	30 kg (66 lb)	9 mL
11 years	35 kg (77 lb)	10 mL
12 years	40 kg (88 lb)	10 mL
13 years	45 kg (99 lb)	10 mL
14 years or more	50 kg (110 lb) or more	10 mL
Adult	50 kg (110 lb) or more	10 mL

SODIUM THIOSULFATE dosage chart at 25% concentration (Pediatric 1.65 ml/kg, Adult 50 ml)

Estimated age	Estimated weight	Dose in ML
3 months	5 kg (11 lb)	8 mL
12 months	10 kg (22 lb)	17 mL
3 years	15 kg (33 lb)	25 mL
6 years	20 kg (44 lb)	33 mL
8 years	25 kg (55 lb)	41 mL
10 years	30 kg (66 lb)	50 mL
11 years	35 kg (77 lb)	50 mL
12 years	40 kg (88 lb)	50 mL
13 years	45 kg (99 lb)	50 mL
14 years or more	50 kg (110 lb) or more	50 mL
Adult	50 kg (110 lb) or more	50 mL