CLINICAL TREATMENT GUIDELINES

FOR

WEAPONS OF MASS DESTRUCTION

(Based on 1996 Olympic Protocols)

Revised February 2, 2000

Guideline Development and Use

Guidelines are systematically developed statements to assist health care providers and patients with decisions about appropriate care/treatment for specific clinical conditions. This supplement was developed by a multidisciplinary panel of health care providers and other experts in consultation with the Department of Health and Human Services.

This supplement is organized to provide a fact sheet on the individual chemical or biological agent, followed by a treatment protocol. The pediatric protocol sections for the chemical agents are located immediately following the chemical agents and before the biological agents. EMS providers may implement these protocols (1) with medical consultation for chemical agent exposure patients and/or (2) in the jurisdictional declared mass caualty incident biological event where antidotes or antibiotics are available.

The guidelines reflect the state of knowledge, current at the time of publication, on effective and appropriate care. Health care providers and patients are encouraged to use the information provided in this clinical practice guideline. The recommendations may not be appropriate for use in all circumstances. Decisions to adopt any particular recommendation must be made by the care provider in light of the available resources and circumstances presented by individual patients.

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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.

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 with resultant hypoxia may be present. Any apparent infant or child distress should be immediately 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
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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 cyanide-containing 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.

Hydrogen Cyanide and Cyanogen Chloride (continued)

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.

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/m^3); 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 non-cardiac 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, clinical 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).

Mustard (continued)

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. 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.

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

3. Treatment:

a. Initial Management:

(1) EMT-B may administer MARK I kits (up to total of three kits) as buddy care to public safety personnel or when directed to do so by an ALS provider based on signs and symptoms in a mass casualty incident (MCI) or on-site chemical testing, confirming nerve or organophosphate agent presence in a mass casualty incident. The Diazepam 10 mg auto-injector (CANA) can only be administered when three MARK I kits are administered in a severe exposure by an ALS provider. Medical Consultation is not required in these situations.

(2) Mild to moderate: Dyspnea should be treated with one or two doses of atropine (MARK I) IM or IV (2-4 mg) and 1-2 doses of pralidoxime (MARK I) or IV drip 600–1200 mg 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).

(3) Severe: Administer 3 doses of atropine IM (three MARK I) or 6 mg IV with caution if hypoxic patient (and start 3 doses of pralidoxime (MARK I) or 2 grams 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 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).

(4) 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 not persistent.

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, produce phosgene 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 low-dose 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 copious quantities 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.

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.

ATROPINE dosage chart at 0.1 mg/ml drug concentration (0.02 mg/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 (0.02 mg/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 (0.02 mg/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 at 2 mg/ml drug concentration (0.02 mg/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

PRALIDOXIME (2-PAM, Protopam) dosage chart at 50 mg/mL (For IV use) – (50 mg/kg Pediatric, 2000 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 = 1000 mg
8 years	25 kg (55 lb)	25 mL = 1250 mg
10 years	30 kg (66 lb)	30 mL = 1500 mg
11 years	35 kg (77 lb)	35 mL = 1750 mg
12 years	40 kg (88 lb)	40 mL = 2000 mg
13 years	45 kg (99 lb)	40 mL
14 years or more	50 kg (110 lb) or more	40 mL
Adult	50 kg (110 lb) or more	40 mL

PRALIDOXIME (2-PAM, Protopam) dosage chart at 300 mg/mL (For IM use) – (40 mg/kg Pediatric, 1800 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 = 200 mg (One MARKI - if only available means)
12 months	10 kg (22 lb)	1.3 mL = 400 mg (One MARKI - if only available means)
3 years or more	15 kg (33 lb) or more	2 mL = 600 mg = One MARK I
Adult	50 kg (110 lb) or more	6 mL = 1800 mg = Three MARK I

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

FACT SHEET

Anthrax

Description of Agent: Inhalation anthrax is a highly lethal infection caused by inhalation of aerosols of the spore form of the bacteria Bacillus anthracis. In naturally occurring cases, anthrax may be spread by entry through skin wounds, causing a localized infection.

Signs and Symptoms: Incubation period for inhalation anthrax is 1-6 days. Fever, malaise, fatigue, cough, and mild chest discomfort are followed by severe respiratory distress with dyspnea, diaphoresis, stridor, and cyanosis. Shock and death occur within 24-36 hours of severe symptoms.

In cutaneous anthrax, a papule develops, then vesicles, followed by a black eschar surrounded by moderate to severe edema. The lesions are usually not painful. Without treatment, the disease may progress to septicemia and death, with a case-fatality rate of 20%. With treatment, fatalities are rare.

Diagnosis: Physical findings are nonspecific in inhalation cases with initial complaints of malaise, fever, headache, and possibly some substernal chest pain. A widened mediastinum is often seen on x-ray. Anthrax is detectable by Gram stains of the blood and by blood culture late in the course of illness.

Treatment: Although usually not effective for inhalation cases after symptoms are present, high-dose antibiotic treatment with penicillin, ciprofloxacin, or doxycycline should be undertaken. Without antibiotic sensitivities, treatment should be started with IV ciprofloxacin (400 mg q 8-12 hr) or IV doxycycline (200 mg initially, followed by 100 mg q 12 hr). Supportive therapy may be necessary.

Prophylaxis: There is a licensed vaccine for use in those considered to be at risk of exposure. The vaccine is administered at 0, 2, and 4 weeks for the initial series, followed by boosters at 6, 12, and 18 months and then an annual booster. Oral ciprofloxacin (500 mg po bid) or doxycycline (100 mg po bid) should be given for known or imminent exposure. After confirmed exposure, all unimmunized individuals should have two 0.5 ml doses of the vaccine 2 weeks apart, and those vaccinated with less than three doses prior to exposure should have a single 0.5 ml booster. Anyone vaccinated with the initial three-dose series in the previous 6 months does not need a booster. Everyone exposed should continue antibiotics for 4 weeks. If no vaccine is available, antibiotics should be used beyond 4 weeks and withdrawn under medical supervision.

Decontamination: Secretion and lesion precautions should be practiced. Anthrax has not been transmitted by the aerosol route person-to-person. After an invasive procedure or autopsy is performed, the instruments and area used should be thoroughly disinfected with a sporicidal agent (iodine or 0.5% sodium hypochlorite).

TREATMENT PROTOCOL

Anthrax

1. General:

Anthrax is a highly lethal infection spread by inhalation or entry through an opening in the skin. The inhalation route will result in a more rapid and deadly infection. The incubation period for both routes is 1-6 days. Fever, malaise, fatigue, cough, and mild chest discomfort are followed by severe respiratory distress with dyspnea, diaphoresis, stridor, and cyanosis. Shock and death occur within 24-36 hours of severe symptoms.

2. Treatment:

a. Evaluate the patient for fever, cyanosis, and respiratory distress.

b. The patient should be given oxygen during transport, as needed.

c. All patients should receive cardiac monitoring and evaluation of oxygenation saturation via pulse oximeter.

d. Obtain IV access with lactated Ringer's at KVO rate.

e. Although usually not effective after severe symptoms are present, high-dose antibiotic treatment with penicillin, ciprofloxacin, or doxycycline should be undertaken. Without antibiotic sensitivities, treatment should be started with IV ciprofloxacin (400 mg q 8-12 hr) or IV doxycycline (200 mg initially, followed by 100 mg q 12 hr). Supportive therapy may be necessary.

f. Before transporting the patient, check for additional victims.

g. Transport the patient to the most appropriate medical facility as directed by medical consultation.

h. Secretion and lesion precautions should be practiced. Anthrax has not been transmitted by the aerosol route person-to-person. After an invasive procedure or autopsy is performed, the instruments and area used should be thoroughly disinfected with a sporicidal agent (iodine or chlorine). Wiping the ambulance interior with a 70% alcohol or other disinfectant is probably unnecessary, but would not be unreasonable. That need not be completed before the next run.

i. Public health officials may recommend that others who may have been initially exposed take prophylactic antibiotics and immunizations before they show signs of illness. If a registry is established, all emergency personnel should identify themselves and indicate when, where, and to what extent they might have been exposed.

FACT SHEET

Botulinum Toxins

Description of Agent: Botulinum toxins are poisonous substances produced by a bacterium, Clostridium Botulinum. They are usually formed in canned foods and eaten but can be spread by aerosol and inhalation. The toxin blocks acetylcholine release at the neuromuscular junction and in the central and peripheral nervous systems.

Signs and Symptoms: Ptosis, generalized weakness, dizziness, dry mouth and throat, blurred vision and diplopia, dysarthria, dysphonia, and dysphagia followed by symmetrical descending flaccid paralysis and development of respiratory failure. Symptoms begin as early as 24-36 hours but may take several days after inhalation of toxin.

Diagnosis: Clinical diagnosis. No routine laboratory findings. Biowarfare or terrorist attack should be suspected if numerous collocated casualties have progressive descending bulbar, muscular, and respiratory weakness.

Treatment: Intubation and ventilatory assistance for respiratory failure. Tracheostomy may be required. Administration of Botulinum antitoxin as soon as possible--trivalent licensed product made by CDC or heptavalent IND product--may prevent or decrease progression to respiratory failure and hasten recovery. Skin testing must be performed before administration of the antitoxin.

Prophylaxis: Pentavalent toxoid (types A, B, C, D, and E) is available as an IND product for those at high risk of exposure. The dosage schedule is 0, 2, and 12 weeks, with yearly boosters.

Decontamination: Hypochlorite and/or soap and water. Toxin is not dermally active and secondary aerosols are not a hazard from patients.

TREATMENT PROTOCOL

Botulinum Toxins

1. General:

Botulinim toxin is a poisonous substance produced by a bacterium, Clostridium Botulinum. It is usually formed in canned foods and eaten but can be spread by aerosol and inhalation. Onset of symptoms is hours to days after taking the poison into the body, so there is virtually no chance that emergency responders would be endangered by the poison carried by a victim. Symptoms typically include drooping eyelids, blurred or double vision, trouble swallowing, dry mouth, and sore throat, followed by a flaccid (limp) paralysis that begins near the head and moves downward. Death most often results from respiratory failure, so respiratory support is the most important aspect of prehospital care. Symptoms begin as early as 24-36 hours but may take several days after inhalation of toxin.

2. Treatment:

a. Evaluate the patient for paralysis, cyanosis, respiratory distress, and signs of pneumonia superimposed on paralysis.

b. The patient may require artificial respiration during transport.

c. All patients should receive cardiac monitoring and evaluation of oxygenation saturation via pulse oximeter.

d. Patient should be given oxygen during transport, as needed, but mechanical ventilation may be more important than oxygen.

e. IV access is not critical, but will be helpful in the hospital setting, where a specific antitoxin will be administered and where the patient will probably remain for a few days to several weeks. If desired, obtain IV access with lactated Ringer's at KVO rate.

f. Intubation and ventilatory assistance may be necessary for respiratory failure. Tracheostomy may be required. Administration of Botulinum antitoxin — trivalent licensed product made by CDC or heptavalent IND product — may prevent or decrease progression to respiratory failure and hasten recovery. Skin testing must be performed before administration of the antitoxin.

g. Before transporting the patient, check for additional victims.

h. Transport the patient to the most appropriate medical facility as directed by medical consultation.

i. Decontaminate with hypochlorite and/or soap and water. Toxin is not dermally active and secondary aerosols are not a hazard from patients.

FACT SHEET

Cholera

Description of Agent: Cholera is a bacterial infection causing severe diarrhea and fluid loss. The causal organism, Vibrio cholerae, is spread through water or food. IV fluids may be exhausted in a hospital or an isolated community during an epidemic.

Signs and Symptoms: The incubation period is 1-5 days. Asymptomatic to severe with sudden onset. Vomiting, abdominal distention, and pain with little or no fever followed rapidly by a profuse, watery diarrhea with a 'rice-water' appearance. Fluid losses may exceed 5 to 10 liters per day. Without treatment, death may result from severe dehydration, hypovolemia, and shock.

Diagnosis: Clinical diagnosis. Watery diarrhea and dehydration. Microscopic exam of stool samples reveals few or no red or white cells. The causal organism can be identified in stool by darkfield or phase contrast microscopy and can be grown on a variety of culture media.

Treatment: Fluid and electrolyte replacement. This often can be accomplished by the use of oral rehydration salts or diluted Gatorade™. IV fluids are needed if there is severe dehydration. Antibiotics will shorten the duration of diarrhea and thereby decrease fluid loss - tetracycline (500 mg q 6 hr x 3 days) or doxycycline (300 mg once or 100 mg q 12 hr x 3 days). There is widespread tetracycline resistance; therefore, ciprofloxacin (500 mg q 12 hr x 3 days), or erythromycin (500 mg q 6 hr x 3 days) should also be considered.

Prophylaxis: A licensed, killed vaccine is available but provides only about 50 percent protection that lasts for no more than 6 months. Vaccination schedule is at 0 and 4 weeks, with a booster every 6 months.

Decontamination: Personal contact rarely causes infection; however, enteric precautions and careful hand washing should be employed. Gloves should be used for patient contact and specimen handling. Bactericidal solutions (hypochlorite) would provide adequate decontamination.

TREATMENT PROTOCOL

Cholera

1. General:

Cholera is a bacterial infection causing severe diarrhea and fluid loss. The causal organism, *Vibrio cholerae*, is spread through water or food. When growing in the intestines, the organism releases a toxin. The toxin, not the infection itself, is the cause of diarrhea. Fluid loss through watery diarrhea is profound and may exceed 5-10 liters/day. IV fluids may be exhausted in a hospital or an isolated community during an epidemic. Without treatment, death may result from severe dehydration, hypovolemia, and shock.

2. Treatment:

a. Evaluate the patient for dehydration and shock.

b. Obtain IV access with a large-bore needle and run lactated Ringer's at a rate sufficient to correct volume loss and replace fluids.

c. Telemetered EKG may provide information on electrolyte balance.

d. Protect yourself and others from contact with diarrheal fluids; they are highly infectious.

(1) Gloves, aprons, and other protective garments should be worn.

(2) Try to contain stools, to minimize contamination of the ambulance. Blanket rolls may be used to create a dike, and plastic or other sheeting may be used to contain fluid within the dike.

(3) Change contaminated clothing and wash hands thoroughly.

e. Before transporting, check for additional victims.

f. Transport the patient to the most appropriate medical facility as directed by medical consultation.

g. Fluid and electrolyte replacement should be undertaken and often can be accomplished by the use of oral rehydration salts or dilute Gatorade™. IV fluids are needed with severe dehydration. Antibiotics will shorten the duration of diarrhea and thereby decrease fluid loss — tetracycline (500 mg q 6 hr x 3 days) or doxycycline (300 mg once or 100 mg q 12 hr x 3 days). There is widespread tetracycline resistance; therefore, ciprofloxacin (500 mg q 12 hr x 3 days) or erythromycin (500 mg q 6 hr x 3 days) should also be considered.

h. Personal contact rarely causes infection; however, enteric precautions and careful hand washing should be employed. Bactericidal solutions (hypochlorite) would provide adequate decontamination. Wash the ambulance interior if necessary and wipe with a 70% alcohol, dilute chlorine bleach, or other disinfectant. If practical, complete the decontamination before the next run.

FACT SHEET

Plague

Description of Agent: Plague is an infectious disease caused by the bacteria Yersinia pestis. In nature, plague is most often spread by fleas that feed on infected rodents, then incidentally bite humans. When spread by that route, it classically causes a local abscess with formation of very large, abscessed, regional lymph nodes called buboes. Plague can also spread by aerosol and inhalation of sputum droplets from a coughing patient. In that manner, a primary pneumonic form develops and progresses rapidly to death without treatment. The plague can also be spread from person to person.

Signs and Symptoms: Pneumonic plague: incubation period is 2-3 days. High fever, chills, headache, hemoptysis, and toxemia progress rapidly to dyspnea, stridor, and cyanosis. Death results from respiratory failure, circulatory collapse, and a bleeding diathesis. Bubonic plague: incubation period is 2-10 days. Symptoms are malaise, high fever, and tender lymph nodes (buboes); they may progress spontaneously to the septicemic form, with spread to the CNS, lungs, and elsewhere.

Diagnosis: Clinical diagnosis. A presumptive diagnosis can be made by Gram or Wayson stain of lymph node aspirates, sputum, or CSF. Plague can also be cultured.

Treatment: Early administration of antibiotics is very effective, but must be started within 24 hours of the onset of symptoms in pneumonic plague. The treatment of choice is streptomycin 30 mg/kg/day IM in 2 divided doses x 10 days. Intravenous doxycycline 200 mg, then 100 mg q 12 hr x 10-14 days is also effective. Chloramphenicol is necessary to treat plague meningitis. Supportive therapy for pneumonic and septicemic forms is required.

Prophylaxis: A licensed, killed vaccine is available. An initial dose is needed, followed by a second smaller dose 1-3 months later, and a third 3-6 months later. A booster dose is given at 6, 12, and 18 months and then every 1-2 years. This vaccine does not protect against aerosol exposure. After face-to-face contact with a pneumonic plague patient or after a confirmed or suspected attack with aerosolized plague, doxycycline 100-mg po bid x 7 days or for the duration of exposure, whichever is longer, should be used.

Decontamination and Isolation: Secretion and lesion precautions should be observed for patients with bubonic plague. Strict isolation of patients with pneumonic plague is needed. Respiratory isolation with the use of a filtered respirator for those with direct contact with patients, and secretion precautions are necessary until the patient has been on antibiotics for at least 48 hours and there has been a favorable response to treatment. Heat, disinfectants, and exposure to sunlight render the bacteria harmless.

TREATMENT PROTOCOL

Plague

1. General:

Plague is an infectious disease caused by a bacterium called *Yersinia pestis* (formerly *Pasteurella pestis*). In nature, plague is most often spread by fleas that feed on infected rodents, then incidentally bite humans. When spread by that route, it classically causes a local abscess with formation of very large, abscessed, regional lymph nodes called buboes (hence the term "bubonic plague"). The incubation period is 2-10 days. Symptoms of malaise, high fever, and tender lymph nodes may progress spontaneously to the septicemic form and spread to the CNS, lungs, and elsewhere. Plague can also spread by aerosol and inhalation of sputum droplets from a coughing patient. In that manner, a primary pneumonic form develops and progresses rapidly to death. Person-to-person spread from a pneumonic plague victim can occur; protective measures are needed to protect against plague as well as other, more common, diseases.

Pneumonic plague: Incubation period is 2-3 days. Symptoms of high fever, chills, headache, hemoptysis, and toxemia may progress rapidly to dyspnea, stridor, and cyanosis. Death results from respiratory failure, circulatory collapse, and a bleeding diathesis.

2. Treatment:

a. Wear a properly fit-tested mask with a high-efficiency particulate (HEPA) filter, following the guidelines for control of tuberculosis.

b. If breathing allows, the patient should be masked to stop as many of the cough droplets as possible before they evaporate to form small-diameter droplet nuclei, which are harder to filter out.

c. Evaluate the patient for fever, cyanosis, and respiratory distress.

d. The patient should be given oxygen during transport, as needed.

e. All patients should receive cardiac monitoring and evaluation of oxygenation saturation via pulse oximeter.

f. Obtain IV access with lactated Ringer's at KVO rate.

g. The early administration of antibiotics is very effective, but must be started within 24 hours of the onset of symptoms in pneumonic plague. The treatment of choice is streptomycin 30 mg/kg/day IM in 2 divided doses x 10 days. Intravenous doxycycline 200 mg, then 100 mg q 12 hr x 10-14 days is also effective. Chloramphenicol is necessary for plague meningitis. Supportive therapy for pneumonic and septicemic forms is required.

h. Before transporting the patient, check for additional victims.

Plague (continued)

i. Transport the patient to the most appropriate medical facility as directed by medical consultation.

j. Secretion and lesion precautions should be observed for patients with bubonic plague. Strict isolation of patients with pneumonic plague is needed. Respiratory isolation and secretion precautions are necessary until the patient has been on antibiotics for at least 48 hours and there has been a favorable response to treatment. Heat, disinfectants, and exposure to sunlight render bacteria harmless.

k. Wiping the ambulance interior with a 70% alcohol or other disinfectant must be done if there is gross contamination with secretions or pus; this is a reasonable precaution in all cases. The organisms do not survive well outside a host; therefore, in an emergency with heavy demand on transport resources, decontamination need not be done before the next run unless there is gross contamination.

l. Public health officials usually recommend that others who may have been exposed take prophylactic antibiotics before they show signs of illness. If a registry is established, all emergency personnel should identify themselves and indicate when, where, and to what extent they might have been exposed. Quarantine may be imposed on those who cannot take or who refuse to take prophylactic treatment.

FACT SHEET

Q Fever

Description of Agent: Q fever is an infectious disease caused by a rickettsial organism, Coxiella burnetti. It is usually spread by aerosolized organisms from infected animal products, such as the placenta, but could be made into an aerosol and disseminated as a terrorist weapon. Person-to-person transmission rarely, if ever, occurs. Case fatality rates are usually below 1%.

Signs and Symptoms: Fever, chills, sweats, coughs, headache, weakness, and pleuritic chest pain may occur as early as 10 days after exposure. Onset may be sudden or insidious and present as a "fever of unknown origin." Pneumonia is present in some cases, but pulmonary syndromes are usually not prominent. Patients are not generally critically ill, and the illness lasts from 2 days to 2 weeks.

Diagnosis: Q fever is not a clinically distinct illness and may resemble a viral illness or other types of atypical pneumonia. The diagnosis is confirmed serologically.

Treatment: Q fever is generally a self-limited illness even without treatment. Tetracycline (500 mg q 6 hr) or doxycycline (100 mg q 12 hr) are the treatments of choice and are given orally for 5 to 7 days. Q fever endocarditis (rare) is much more difficult to treat.

Prophylaxis: Treatment with tetracycline or doxycycline, starting between the 8th to12th day postexposure and continued for 5 days, should prevent the onset of symptoms. An inactivated whole cell vaccine (investigation) is effective in eliciting protection against exposure, but severe local reactions to this vaccine may be seen in those who already possess immunity.

Decontamination: Patients who are exposed to Q fever by aerosol do not present a risk for secondary contamination or re-aerosolization of the organism. Decontamination is accomplished with soap and water or by the use of weak (0.5 percent) hypochlorite solutions.

TREATMENT PROTOCOL

Q Fever

1. General:

Q fever is an infectious disease caused by a rickettsial organism. Rickettsia is smaller than bacteria but larger than viruses. They usually live within cells, but have more complete metabolic systems than viruses. The organism that causes Q fever is called Coxiella burnetti.. The organism is robust and infection occurs via inhalation of organisms. After an incubation period, which may require from 10 days to 3 weeks, the onset of Q fever symptoms may be sudden with chills, a headache behind the eyes, weakness, malaise, and severe sweats; or the onset may be insidious and present as a "fever of unknown origin." Pneumonia is present in some cases, but pulmonary symptoms are usually not prominent. Person-to-person transmission rarely, if ever, occurs. Case fatality rates are usually below 1%.

2. Treatment:

a. Evaluate patient for dehydration and shock (which would suggest an alternate diagnosis). If effects are mild, it might be practical to send the patient for medical care via private conveyance; hospitalization may not be necessary.

b. IV fluids are not usually necessary, but if the patient's condition suggests dehydration or the possibility of some other diagnosis, obtain IV access and run lactated Ringer's at a rate sufficient to correct volume loss and replace fluids.

c. Universal precautions should be practiced with respect to body fluids.

d. Q fever is generally a self-limited illness even without treatment. Tetracycline (500 mg q 6 hr) and doxycycline (100 mg q 12 hr) are the treatments of choice and are given orally for 5 to 7 days starting between the 8th to 12th day postexposure. Q fever endocarditis (rare) is much more difficult to treat.

e. Before transporting the patient, check for additional victims.

f. Transport the patient to the most appropriate medical facility as directed by medical consultation.

g. Patients who are exposed to Q fever by aerosol do not present a risk for secondary contamination or re-aerosolization of the organism. Decontamination is accomplished with soap and water or by the use of weak (0.5%) hypochlorite solutions. Wash the ambulance interior if necessary and wipe with dilute (0.5%) chlorine bleach or other appropriate disinfectant. Decontamination is not absolutely necessary before the next run unless there has been unusually heavy contamination.

FACT SHEET

Salmonella

Description of Agent: Several distinct bacteria within the group Salmonella cause diarrheal illnesses, sometimes with a septicemia. In 1984, *Salmonella typhimurium*, which causes a diarrheal illness in humans, was used by terrorists in Oregon to contaminate foods in restaurants: 720 people became ill as a result. *Salmonella* illnesses are not rare, and cannot be distinguished on the basis of clinical signs from other causes of diarrhea. The illness would typically be less profound than with cholera. Infants are at the greatest risk of severe illness and death.

Signs and Symptoms: Acute onset of headache, abdominal pain, bloody diarrhea, nausea, and sometimes vomiting 6 to 72 hours after exposure to contaminated food; incubation is usually 12-36 hours. Fever is usually present. Diarrhea and anorexia often last several days. Dehydration may be severe, especially in infants.

Diagnosis: Fecal Gram stain and culture; serologic tests are not useful. Salmonella is a commonly occurring disease in the U.S. with an estimated 5 million annual cases.

Treatment: For uncomplicated cases, oral rehydration therapy alone is indicated. IV fluids may be needed with severe dehydration. Antibiotics may prolong the Carrier State, but should be considered with infants, the elderly, or those with underlying illnesses. Ciprofloxacin 500 mg q 12 hr x 3 days is effective.

Prophylaxis: No immunization available.

Decontamination: Enteric precautions should be practiced. Hypochlorite and/or soap and water is effective. Destroy any remaining contaminated food. Wear gloves for patient contact and specimen handling.

TREATMENT PROTOCOL

Salmonella

1. General:

Several distinct bacteria within the group Salmonella cause diarrheal illnesses, sometimes with a septicemia (where organisms are also multiplying in the blood and other tissue). In 1984, *Salmonella typhimurium*, which causes a diarrheal illness in humans, was used by terrorists in Oregon to contaminate foods in restaurants: 720 people became ill as a result. *Salmonella* illnesses are not rare, and cannot be distinguished on the basis of clinical signs from other causes of diarrhea. The illness would typically be less profound than with cholera. Infants are at the greatest risk of severe illness and death. Signs and symptoms include the acute onset of headache, abdominal pain, bloody diarrhea, nausea, and sometimes vomiting 6 to 72 hours after exposure to contaminated food; incubation is usually 12-36 hours. Fever is usually present. Diarrhea and anorexia often last several days. Dehydration may be severe, especially in infants.

2. Treatment:

a. Evaluate the patient for dehydration and shock. If the patient has only mild effects, it might be practical to send him/her for medical care via private conveyance; hospitalization may not be necessary.

b. Obtain IV access with a large-bore needle and run lactated Ringer's at a rate sufficient to correct volume loss and replace fluids.

c. Telemetered EKG may provide information on electrolyte balance.

d. Protect yourself and others from contact with diarrheal fluids; they are highly infectious.
(1) Gloves, aprons, and other protective garments should beworn.
(2) Try to contain the patient's stools and to minimize contamination of the ambulance. Blanket rolls may be used to create a dike and plastic or other sheeting may be used to contain fluid within the dike.
(3) Change contaminated clothing and wash hands thoroughly.

e. For uncomplicated cases, oral rehydration therapy alone is indicated. IV fluids may be needed with severe dehydration. Antibiotics may prolong the Carrier State, but should be considered with infants, the elderly, or those with underlying illnesses. Ciprofloxacin 500 mg q 12 hr x 3 days is effective.

f. Before transporting the patient, check for additional victims.

g. Transport the patient to the most appropriate medical facility as directed by medical consultation.

h. Enteric precautions should be practiced. Hypochlorite and/or soap and water is effective. Destroy any remaining contaminated food. Wash the ambulance interior if necessary and wipe with a 70% alcohol, dilute chlorine bleach, or other disinfectant. If practical, complete the decontamination before the next run.

FACT SHEET

Staphylococcal Enterotoxin B

Description of Agent: Staphylococcus enterotoxin B (SEB) is one of several toxins produced by the bacteria *Staphylococcus aureus*. SEB is a common contributor to staphylococcal food poisoning but can also be disseminated as an aerosol and inhaled.

Signs and Symptoms: From 3-12 hours after aerosol exposure, there is the sudden onset of fever, chills, headache, myalgia, and nonproductive cough. Some patients may develop shortness of breath and retrosternal chest pain. The fever may last 2 to 5 days, and the cough may persist for up to 4 weeks. Patients may also present with nausea, vomiting, and diarrhea if they swallow toxin. Higher exposure levels can lead to pulmonary edema, and rarely, death.

Diagnosis: Diagnosis is clinical. Patients present with a febrile respiratory syndrome without CXR abnormalities. Large numbers of people presenting with typical symptoms and signs of SEB pulmonary exposure would suggest an intentional attack with this toxin.

Treatment: Treatment is limited to supportive care. Artificial ventilation might be needed for very severe cases, and attention to fluid management is important.

Prophylaxis: Use of protective mask. There is currently no human vaccine available to prevent SEB intoxication.

Decontamination: Hypochlorite (bleach) and/or soap and water. Destroy any food that may have been contaminated.

TREATMENT PROTOCOL

Staphylococcus Enterotoxin B

1. General:

Staphylococcus enterotoxin B (SEB) is a substance produced by Staphylococcus aureus. SEB is common contributor to foodborne enteritis outbreaks but can also be disseminated as an aerosol and inhaled. Symptoms usually follow inhalation by 3 to 12 hours and would include sudden onset of fever, headache, chills, pain in the muscles, and a nonproductive cough. Nausea, vomiting, and watery diarrhea may be accompanied by heavy fluid losses and a feeling of profound malaise leading to incapacitation; higher doses can lead to a toxic shock syndrome and death. Reddening of the eyes is common. Overall, the mortality rate from an attack would be lower than that from many other biological agents.

2. Treatment:

a. Evaluate the patient for dehydration and shock.

b. Obtain IV access with a large-bore needle and run lactated Ringer's at a rate sufficient to correct volume loss and replace fluids.

c. Telemetered EKG may provide information on electrolyte balance.

d. Diarrheal fluids are not dangerous, but you may not know whether you are dealing with SEB or cholera or Salmonellosis. Therefore, treat diarrheal fluids as highly infectious.

(1) Don gloves and aprons or other protective garments.

(2) Try to contain stools, to minimize contamination of the ambulance. Blanket rolls may be used to create a dike, and plastic or other sheeting may be used to contain fluid within the dike.

(3) Change contaminated clothing and wash hands thoroughly.

e. Treatment is limited to supportive care. Artificial ventilation might be needed for very severe cases, and attention to fluid management is important.

f. Before transporting the patient, check for additional victims.

g. Transport the patient to the most appropriate medical facility as directed by medical consultation.

h. Decontaminate with hypochlorite (bleach) and/or soap and water. Destroy any food that may have been contaminated. Wash the ambulance interior if necessary and wipe with a 70% alcohol, dilute chlorine bleach, or other disinfectant. If practical, complete the decontamination before the next run.

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