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PROTECT THYSELF AND STAFF

At a chemical release site, only properly trained HAZMAT personnel wearing Level A* PPE may enter the contaminated "Hot Zone" for patient rescue. All patients and rescuers in the Hot Zone must be decontaminated in a restricted "Warm Zone" prior to patient transport. Properly trained HAZMAT and First Responder personnel in this zone must wear Level B PPE.†

Medical personnel in the Cold Zone, medical transport personnel and emergency department and intensive care unit personnel should wear splash-resistant clothing and boots, nitrile or SilverShield® gloves, eye protection, and an air-purifying respirator with appropriate cartridge§ during patient handling to prevent secondary exposure from off-gassing via exhalation, body secretions, and from contaminated patients who self-transported or were inadequately decontaminated.

PATIENT DECONTAMINATION

All victims of toxic chemical exposures require rapid decontamination. Contaminated clothing can trap hazardous vapors and continue to cause harm. If the patient's clothing was exposed to toxic liquid, quickly remove *all* clothing, from

head to toe, before washing or showering the entire body with copious amounts of tepid water. After a vapor exposure, remove outer clothing before washing. Discard all contaminated clothing as hazardous waste.

The following are appropriate irrigation procedures:

- Eyes: copious irrigation with saline, water, or eye solutions for 15 minutes.
- Skin: copious flush with water, followed by 2 soap and water wash/shampoos, 2 rinses. Do not abrade skin.
- Wounds: flush with surgical irrigation solution.

For exposure to dry, pure metals and strong corrosives, gently brush or vacuum large particles before flushing. Following exposure to gas or vapors, persons without skin or eye irritation do not need additional decontamination and are not likely to be a secondary contamination risk.

Decontaminate equipment, eyeglasses, and hard surfaces with 5% hypochlorite (undiluted household bleach) for all military-grade chemicals. For ammonia and unknown contaminants, use soap and water.

TREATMENT

Treatment is based on identifying the chemical from an exposure history, or from recognizing a constellation of signs and symptoms that indicate a chemical family when the chemical agent is unknown. Initial treatment for all victims includes "ABCs," rapid decontamination and fresh air. Treatment protocols may vary depending on training and the availability of antidotes. Call Oregon Poison Control (800/222-1222) for medical advice.

ALERT

Report the following immediately to the Oregon Poison Center and your local health department:

- clusters of sudden onset of respiratory, neurological, gastrointestinal or dermatological symptoms;
- clusters with unusual age distribution (e.g. child chemical exposures),
- unexplained deaths in young or healthy people.

SHELTERING

Assign a staff person to provide patient information on what to do in case of a chemical attack, and how to shelter in place (<http://www.cdc.gov/planning/shelteringfacts.asp>). Find out how to prepare your office to shelter in place at www.nicsinfo.org/SIP%20Center.htm.



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PREPARING FOR A CHEMICAL TERRORIST EVENT — A PRIMER

THE POTENTIAL terrorist use of biological agents has several times been discussed in these pages. However, the recent instances of chemical agents used in terror attacks worldwide reminds us that chemical weapons are also terrifying and lethal. This issue of the *CD Summary* is a primer on potential agents of chemical terrorism.

GHOSTS FROM THE PAST

The use of chemicals to kill and terrorize was reported as early as 423 BC in the Peloponnesian War, when sulfur smoke was used to conquer a fort. The 7th-century Greeks combined naphtha, lime, sulfur, pitch and saltpeter in their naval operations. Venetian soldiers placed poisons in explosive mortar shells and also poisoned wells, crops and animals in the 15th and 16th centuries. Although during the 1800s there was much debate over proposals to use poisonous gases in wars, the use of chemicals as an "acceptable" military weapon really began with the notorious 1915 release of chlorine gas from 6000 cylinders in Belgium, which killed 800 in one afternoon. Chemical use was later expanded in WWI by the release of phosgene and chloropicrin by the British, and mustard gas by the Germans. U.S. chemical defoliants cleared tunnels and bunkers in Vietnam and Laos, and the Soviets used chemicals in Afghanistan. Iraq's use of mustard, tabun and sarin gases in the Iranian war and cyanide against the Kurds triggered the latest anxiety about military use of chemical weapons against populations.

Terrorists shocked our sense of security with the 1995 Tokyo subway station sarin gas attack, killing 11 and injuring more than 5,000. Just last year, a Mexican tractor-trailer loaded with 10 tons of cyanide was hijacked, muriatic acid bombs were found in Florida mailboxes, and a terrorist plot to release cyanide gas in the London subway system was foiled.

TODAY'S SPECTER

Intentional events involving stolen or illicit purchases of commercial or industrial chemicals could target groups of people, building ventilation systems, or drinking water systems. Food sources can be targeted, as in the January 2003 poisoning of 70 Michiganders through pesticide-laced hamburger.

Anyone responding to an event, assisting or treating casualties, or providing ancillary medical services needs to be familiar about the effects and treatments of chemical exposures, appropriate decontamination procedures, and protective clothing and equipment to wear to protect oneself from a secondary exposure. Here's a quick readiness guide for you and your staff.

PREPARATION AND RECOGNITION

There are thousands of commercially used chemicals and a limited number of military-grade agents. The easiest approach to identify chemicals for diagnosis, appropriate decontamination and medical treatment, is by grouping those agents that present with similar symptoms, or are treated with similar protocols. Chemicals of greatest concern can be grouped as follows: nerve agents, systemic asphyxiants, blister/vesicant agents, pulmonary irritants, and lacrimator (riot-control) agents.

CHEMICAL TYPES

Nerve cholinesterase inhibitors

(sarin, tabun, soman, VX) and pesticides manufactured and distributed for agricultural or home use (such as malathion and diazinon organophosphates or methyl-carbamates). These chemicals cause overstimulation of cholinergic muscarinic sites resulting in increased body fluid secretion, and of cholinergic neuromuscular sites resulting in fasciculations, weakness and muscular paralysis, and CNS symptoms. Nerve agents are readily absorbed through eyes, lungs and skin, and result in systemic poisoning. One drop of concentrated sarin on the skin can be lethal. Nerve agents are volatile at ambient temperatures, and exposure to high levels of vapor can produce symptoms within seconds.

Systemic asphyxiants

(cyanides, sulfides, azides) are rapidly absorbed by inhalation and mucosal and dermal surfaces. They interfere with cellular oxygen utilization within seconds of exposure.

Blister/vesicants

(sulfur and nitrogen mustards, Lewisite, phosgene oxime) are rapidly absorbed through eyes, lungs and skin. They cause skin blisters and damage to eyes, mucosa, respiratory tract, and internal organs. Lewisite causes liver toxicity, and may cause systemic arsenic toxicity (contains trivalent arsenic). The onset of symptoms is generally immediate, although mustards can have a delayed onset.

Pulmonary irritants

(ammonia, chlorine, hydrogen fluoride, oxides of nitrogen, vinyl chloride, phosgene) are irritating and corrosive to the respiratory tract and cause severe pulmonary edema, eye and skin irrita-

* Level A: positive pressure self-contained breathing apparatus (SCBA) and fully encapsulating, chemical protective suit

† SCBA, hooded chemical-resistant suit, butyl outer/SilverShield® inner gloves, chemical resistant boots.

§ Or a full-face, air-purifying, canister-equipped respirator *De rigeur* for the security-conscious fashionista.

Chemical Type	Signs and Symptoms	Medical Treatment Summary	Useful Labs
<p>Nerve Agents: sarin, tabun, soman, VX, organophosphate and methyl-carbamate pesticides.</p> <p>Immediate decontamination is critical to prevent further exposure to patient and health care workers. Onset: Vapor—seconds to minutes. Liquid—minutes to hours</p>	<p>Note: Not all signs need to be present.</p> <p><i>Muscarinic:</i> diaphoresis, salivation, nausea, abdominal cramps, vomiting, diarrhea, urination, lacrimation, miosis, blurred vision, bradycardia, bronchorrhea, dyspnea, chest tightness.</p> <p><i>Nicotinic:</i> fasciculations, muscular weakness, muscle paralysis, hypertension, tachycardia.</p> <p><i>CNS:</i> confusion, restlessness, anxiety, ataxia, headaches, fatigue, loss of consciousness, respiratory depression, seizures, coma.</p> <p><i>Note:</i> children may exhibit different symptoms (CNS, stupor, flaccidity, dyspnea).</p>	<p>Inhalation and dermal absorption Treatment must counteract cholinergic excess in all three receptor sites. Maintain ventilation, oxygenation, ABCs.</p> <p>Antidotes: Atropine sulfate for hypersecretions, pulmonary edema, or bradycardia. Parenteral atropine should not be used to control rhinorrhea or to treat miosis. Adult: 1–2 mg IV/IM initial dose. 1–2 mg IV/IM may be continued q 5–10 mins. until bronchial secretions are dried, bradycardia resolved. If symptoms return, repeat dosing. Pediatric: 0.02 mg/kg IV (IM okay if no IV access); use at least 0.1 mg; not to exceed adult dose q 5 to 60 mins until symptoms subside (bronchosecretions dry; bradycardia resolved); if symptoms return, repeat dosing scheme. Pralidoxime (2-PAM) for muscle weakness, fasciculations, severe toxicity. Adult: 1–2 g. IV or IM. IV dose can be mixed in 100 mL NS and infused over 15–30 mins. If symptoms recur or continue, repeat dose in one hour or start continuous infusion at 200-500 mg/hour. Pediatric: 25 to 50 mg/kg up to 1 g IV or IM. IV dose can be infused over 15 –30 mins. If symptoms recur or continue, repeat dose in one hour or start continuous infusion at 5–10 mg/kg/hr. May repeat in 1 hour if weakness not resolved, then again every 6 hours, or by continuous IV infusion at 5–10 mg/kg/hr. Diazepam or equivalent dosing of another Benzodiazepine for seizures, agitation: Adult: 5–10 mg IV/ IM. Pediatric: 0.1 mg/kg IV/ IM</p>	<p>Treat patient immediately. Do not wait for laboratory confirmation.</p> <p>Plasma pseudocholinesterase and red blood cell acetylcholinesterase for organophosphate and methyl carbamate chemicals.</p>
<p>Systemic Asphyxiants: cyanides, sulfides, azides.</p> <p>Rapid decontamination. Onset: Cyanides—seconds.</p>	<p>Headaches, dizziness, nausea, vomiting, drowsiness, hallucinations, gasping for air, loss of consciousness, brady/tachycardia, seizures, respiratory/cardiac arrest.</p> <p>Liquid cyanogen chloride may cause eye and respiratory tract irritation and chemical burns on exposed skin.</p>	<p>Inhalation and dermal absorption Maintain ventilation, oxygenation, ABC’s.</p> <p>Cyanide: Cyanide Antidote Kit Adult: 10 mL of 3% sodium nitrite solution (300 mg) IV over 3 minutes; followed by sodium thiosulfate 12.5 grams IV (50 mL of a 25% solution), over 10 to 20 min. Pediatric: 0.33 mL/kg of 10% sodium nitrite IV (10 mg/kg) over 3 to 5 mins. followed by 1.65 mL/kg of 25% sodium thiosulfate (400 mg/kg) over 10 mins. Other asphyxiants: No antidote; provide supportive care. Supportive: oxygen, correct acidosis, treat eye and skin injuries as for burns.</p>	<p>Cyanide: high anion gap metabolic acidosis, elevated lactate methemoglobin, and urinary thiocyanate levels.</p>
<p>Vesicants: sulfur and nitrogen mustards, Lewisite, phosgene oxime</p> <p>Rapid decontamination Onset: Lewisite—immediate Mustard—effects may be delayed 2–24 hours. Phosgene oxime—immediate</p>	<p>Pain initially, followed by erythema, blisters, and a chemical burn, severe itching, necrosis. Mustards produce groups of small blisters over erythematous areas. Lewisite blisters expand, taking up to 4 days to cover entire erythematous areas. Conjunctivitis, corneal opacity, blepharitis. Dry cough, nose, throat, lung irritation to marked airway damage. Epistaxis. Nausea, vomiting, diarrhea, abdominal pain, hyperexcitability, convulsions.</p>	<p>Inhalation and dermal absorption No antidotes. Maintain ventilation, oxygenation, ABC’s, eye and burn care. May consider using topical BAL.</p>	<p>Leukocytosis during first day of exposure. Chemical pneumonitis within first 2 to 3 days after inhalation exposure. Thiodiglycol urinary metaolite of sulfur mustard. Urinary speciated arsenic to identify Lewisite.</p>
<p>Pulmonary Irritants: ammonia, chlorine, hydrogen fluoride, oxides of nitrogen, phosgene.</p> <p>Rapid decontamination. Onset: Immediate. Phosgene can be delayed 24 hours.</p>	<p>Eye and airway irritation, dyspnea, chest tightness, rapid breathing, coughing, wheezing, rales, hemoptysis, stridor, frothy secretions (2–24 hrs), cyanosis, upper airway swelling, pulmonary edema, lung collapse; tachycardia, initial hypertension, hypotension, possible cardiovascular collapse; nausea, vomiting; skin burns, blisters.</p>	<p>Inhalation No antidotes. Manage secretions, maintain ventilation, oxygenation, monitor heart, renal, liver functions. Treat pulmonary edema with PEEP to maintain PO₂ above 60 mm Hg. Hydrogen fluoride: Pneumonitis from inhaled HF may be treated with oxygen and nebulized 2.5% calcium gluconate (mix 1.5 mL of 10% calcium gluconate with 4.5 mL sterile water). Skin burns may be treated with topical calcium gluconate. Do not inject or use calcium chloride for treating skin burns. For hydrogen fluoride systemic toxicity, serum calcium, magnesium and potassium levels must be rapidly corrected with IV boluses of calcium gluconate and magnesium sulfate.</p>	<p>Hydrogen fluoride: EKG and serum potassium, calcium and magnesium concentrations q30 min until stable. No laboratory tests for other pulmonary irritants.</p>
<p>Lacrimators/Riot Control: CN, CS tear gas, chloropicrin.</p> <p>Rapid decontamination. Onset: Seconds.</p>	<p>Eye, nose, throat, respiratory tract irritation, tearing, rhinorrhea, sneezing, coughing, bronchospasm, wheezing, vomiting, pulmonary edema, skin burning sensation, erythema. High concentrations—chemical skin burns. Exposure in confined spaces—severe airway damage possible. Particles can get embedded in cornea or conjunctiva to cause tissue damage.</p>	<p>No specific therapy. Eye flushing, remove particles. Ophthalmic exam.</p>	<p>No laboratory tests.</p>

Please obtain treatment information from the Oregon Poison Center 503.494.8968 or 800.222.1222

For CDC fact sheets on more chemicals, go to <http://www.bt.cdc.gov>

For a larger, 11”x17” downloadable version of this chart, go to <http://www.dhs.state.or.us/publichealth/bioterrorism/provider.cfm>

tion. Ammonia and chlorine liquids can result in frostbite. Symptom onset is immediate, although phosgene’s full effects may be delayed 12 to 24 hours.

Lacrimators

(CN, CS, tear gas) are generally used for riot control, and cause respiratory, eye and skin irritation, and possibly pulmonary edema and vomiting. Symptom onset is immediate.

Preparation for the above also includes insuring that your facility has the necessary decontamination and personal protective equipment (PPE) ready, and that staff are trained in

their use and in triage of mass casualties. You’ll need ready access to antidotes such as atropine, pralidoxime (2-PAM), and cyanide kits.

EARLY RECOGNITION CLUES

If the chemical is unknown, if it’s a mass casualty event, or if antidotes may be indicated, contact Oregon Poison Control (800/222-1222) for immediate assistance. Treatment must almost always be administered before the diagnosis can be confirmed. The few lab tests available for chemical exposures are only for later confirmation.

The *odor* of a chemical residue may give clues, but most of these classes of chemicals have a variety of odors or have no odor. Odors of nerve agents range from no odor to fruity or camphor. Bitter almond and mild garlic odors may be sensed with systemic asphyxiants like cyanide, but olfactory fatigue sets in quickly. Vesicants have a variety of odors, including garlicky, fishy, soapy, fruity, or no odor. Lewisite is said to smell like geraniums, and mustard gas like garlic or horseradish. Chlorine and ammonia are distinctive, but phosgene smells like irritating, new-mown hay. Lacrimators have a variety of odors.

Color is problematic, as most of these toxic gases are colorless, and liquids can vary through a spectrum from colorless to shades of yellow, green and brown.

Signs and symptoms depend on the chemical agent, dose, and exposure routes. Sensitive individuals, those with pre-existing conditions such as cardiovascular and respiratory diseases and hypertension, and children are at higher risk. Children may exhibit different signs and symptoms and are more vulnerable because of their small body weight, relatively greater lung surface areas, and higher inhalation rates. Their small airways are especially sensitive to corrosive chemicals.