Overview

Chemical warfare is not a popular topic but is one of great concern to civilians, emergency physicians, and prehospital providers given the potential devastation.

As General Pershing noted after World War I, "...the effect is so deadly to the unprepared that we can never afford to neglect the question."

The US government has determined that the likelihood of a terrorist attack within the United States is increasing. This has prompted governmental domestic preparedness training initiatives targeted at physicians and other healthcare providers in the areas of nuclear, biological, and chemical warfare.

Decontamination is time consuming and requires resources. Nerve agents and substances causing injury to the skin and tissue are easily soluble in and penetrate many different types of material. If chemical warfare (CW) agents have penetrated sufficiently deep, then toxic gases can be released from the material for long periods. By adding substances that increase the viscosity of a CW agent, its persistence time and adhesive ability can be increased. These thickened agents will thus be more difficult to decontaminate with liquid decontaminants since they adhere to the material and are difficult to dissolve.

The need for decontamination can only be established by means of detection. If detection is not possible, then decontamination must be done solely on suspicion of contamination.

This article focuses on the initial approach to the chemical warfare victim. Detailed information on patient decontamination and the appropriate use of personal protective equipment is described elsewhere on this web site (see CBRNE - Chemical Decontamination for more information).

For excellent patient education resources, visit eMedicine's Bioterrorism and Warfare Center. Also, see eMedicine's patient education articles Chemical Warfare and Personal Protective Equipment.

Triage of Chemical Casualties

Patient triage only should be carried out in the hospital setting by those wearing appropriate personal protective equipment. In the simplest form of triage, patients or casualties are separated into 3 groups. The first group consists of those for whom the degree of medical care required exceeds that which is available. In this setting, medical assets are insufficient relative to the presenting illness, or an experienced triage officer deems the patient's injuries irreversible. The medical focus is on comfort care, and the casualty is triaged as expectant. A casualty's classification may change as assets become available or when later reevaluation demonstrates that the casualty's condition was not as serious as first anticipated.

The second group consists of casualties who require immediate intervention for survival. In a conventional situation (ie, noncontaminated environment), these casualties usually have treatable injuries affecting the airway, breathing, or circulation (ABCs). Other examples of immediate interventions include administration of antidotes or spot decontamination.
The third group consists of casualties who have injuries that place them in no immediate danger of loss of life. Medical care is needed but not immediately. For example, casualties in this group may include those with minor injuries who merely require laceration repair or those with extensive injuries necessitating long-term hospitalization who are presently stable.

The triage system commonly used by US military medical departments and by civilian medical systems contains the above categories (immediate, delayed, and expectant) and incorporates a fourth category (minimal). In this scheme, minimal injuries are those that require a quick intervention, do not require a physician or evacuation, and result in a rapid return to duty. Occasionally, as in the North Atlantic Treaty Organization (NATO) Emergency War Surgery Handbook, a fifth category, urgent, is added to denote a casualty who requires intervention within minutes to survive. Also, in some schemes, the term “chemical immediate” is used for casualties who require immediate administration of antidotes for survival (as in nerve agent or cyanide poisoning).[1]

Decontamination is the process by which particulate, vapor, and liquid materials are safely removed from an exposed person without further contaminating the casualty, the environment, or rescuers. Decontamination is an important part of all disasters involving hazardous materials and weapons of mass destruction.

Two main goals exist in setting up decontamination at a medical facility:

- To protect the facility and its personnel from becoming contaminated, and thus further casualties
- To facilitate the treatment and triage of contaminated patients as rapidly as possible

Analysis of hazardous materials accidents has shown that up to 85% of the victims arrive at a healthcare facility without prehospital treatment or decontamination. Terrorist events, with their larger number of patients, unknown substances, and large numbers of “worried well,” increase the possibility of casualties arriving at a facility contaminated or potentially contaminated.

**Immediate support measures**

Monitor patients who potentially are exposed to chemical warfare agents for changes in their clinical status and provide appropriate supportive measures. First address the patient's ABCs and coexisting life-threatening or limb-threatening injuries. Reassess these injuries throughout the patient's course.

**Emergency department management**

*Potential for secondary contamination*

Victims who were exposed only to gas or vapor and have no gross deposition of the material on their clothing or skin are not likely to carry significant amounts of chemical beyond the hot zone and are not likely to pose risks of secondary contamination to hospital personnel. However, victims whose skin or clothing are covered with liquid or solid chemical or victims who have condensation of chemical vapor on their clothes or skin may contaminate hospital personnel and the ED by direct contact or by off-gassing vapor. If the victim has ingested a chemical, toxic vomitus may also pose a danger through direct contact or off-gassing vapor.

*Decontamination area*

Previously decontaminated patients and patients exposed only to gas or vapor who have no evidence of skin or eye irritation may be transferred immediately to the critical care area. Other victims will require decontamination as described below.

*ABC reminders*

Evaluate and support airway, breathing, and circulation. Intubate the trachea in cases of respiratory compromise. If the patient's condition precludes intubation, surgically create an airway.

Treat patients who have bronchospasm with aerosolized bronchodilators; use these and all catecholamines with caution because of the possible enhanced risk of cardiac dysrhythmias.

Patients who are comatose, hypotensive, or have seizures or ventricular dysrhythmias should be treated in the conventional manner.

*Basic decontamination*

Patients who are able and cooperative may assist with their own decontamination. Remove and double-bag contaminated clothing and personal belongings.

Flush exposed or irritated skin and hair with plain water for 3-5 minutes. For oily or otherwise adherent chemicals, use mild soap on the skin and hair. Rinse thoroughly with water.
Flush exposed or irritated eyes with plain water or saline for at least 5 minutes. Remove contact lenses if present and easily removable without additional trauma to the eye. If a corrosive material is suspected or if pain or injury is evident, continue irrigation while transferring the patient to the critical care area.

In cases of ingestion, do not induce emesis. Administer 4-8 ounces of water to dilute stomach contents if the patient is conscious and able to swallow. Immediately transfer the patient to the critical care area.

**Critical care area**

Be certain that appropriate decontamination has been carried out.

**ABC reminders**

Evaluate and support airway, breathing, and circulation. Establish intravenous access in seriously ill patients. Continuously monitor cardiac rhythm.

Patients who are comatose, hypotensive, or have seizures or ventricular dysrhythmias should be treated in the conventional manner.

**Inhalation exposure**

Administer supplemental oxygen by mask to patients who have respiratory complaints. Treat patients who have bronchospasm with aerosolized bronchodilators; use these and all catecholamines with caution because of the potential or possible enhanced risk of cardiac dysrhythmias.

**Skin exposure**

If chemical burns are present, treat as thermal burns.

**Eye exposure**

Ensure that adequate eye irrigation has been completed. Test visual acuity. Examine the eyes for corneal damage using a magnifying device or a slit lamp and fluorescein stain. For small corneal defects, use ophthalmic ointment or drops, analgesic medication, and an eye patch. Immediately consult an ophthalmologist for patients who have severe corneal injuries.

**Ingestion exposure**

Do not induce emesis. If the patient is alert and charcoal has not been given previously, administer a slurry of activated charcoal. If a corrosive material is suspected, administer 4-8 ounces of water; do not give a slurry of activated charcoal. Consider endoscopy to evaluate the extent of gastrointestinal tract injury. If a large dose has been ingested and the patient's condition is evaluated within 30 minutes after ingestion, consider gastric lavage.

**Laboratory tests**

Routine laboratory studies for all exposed patients include CBC, glucose, and electrolyte determinations. Additional studies for patients exposed to an unidentified chemical include ECG monitoring, renal function tests, and liver function tests. Chest radiography and pulse oximetry (or ABG measurements) are recommended for severe inhalation exposure.

**Disposition and follow-up**

Consider hospitalizing patients who have suspected serious exposures and persistent or progressive symptoms.

**Delayed effects**

When the chemical has not been identified, the patient should be observed for an extended period or admitted to the hospital.

**Categories for Triage of Chemical Casualties**

**Immediate**

**Nerve agents**

A casualty of nerve agents who is in severe distress is classified as immediate. The patient may be unconscious, may be in severe respiratory distress, or may have become apneic minutes before reaching the facility. He or she may be convulsing or immediately postictal. Often the contents of 3 MARK I kits (or more) plus diazepam and possibly
short-term ventilatory assistance is all that is required to save a life. In addition, classify a casualty with signs in 2 or more systems (eg, neuromuscular, gastrointestinal [GI], respiratory excluding eyes and nose) as immediate and administer the contents of 3 MARK I kits and diazepam.

**Cyanide**

A casualty of cyanide who is convulsing or who became apneic minutes before reaching the medical facility and has adequate circulation (still has a pulse) is in the immediate group, assuming that cyanide antidotes are available. If the ABCs remain adequate, the administration of antidote may be all that is required for complete recovery. Since death usually occurs within 4-5 minutes of exposure to a lethal amount of cyanide unless treatment is immediate, this type of casualty is unlikely to be seen at the medical facility.

**Phosgene and vesicants**

Place casualties of phosgene or vesicant agents who have moderate or severe respiratory distress in the immediate group, where intense ventilatory and other respiratory support measures are immediately available.

**Incapacitating agents**

Those with cardiovascular collapse or severe hyperthermia are placed in the immediate category following exposure to agents such as 3-quinuclidinyl benzilate (BZ) or Agent 15.

**Delayed**

**Nerve agents**

Place casualties who require hospitalization but have no immediate threat to life in the delayed group. This generally is limited to a casualty who has survived a severe nerve agent exposure, is regaining consciousness, and has resumed spontaneous respiration. This casualty requires further medical care for the time necessary for recovery.

**Vesicants**

Triage those with skin injuries of greater than 5% total body surface area but less than 50% as delayed. Other criteria include severe eye involvement or pulmonary symptoms with an onset of more than 4 hours postexposure.

**Cyanide**

Triage victims of cyanide vapor exposure who are alive after 15 minutes as minimal or delayed.

**Pulmonary agents**

Following exposure, triage patients with delayed onset of respiratory distress (>4 h) as delayed.

**Incapacitating agents**

Severe or worsening signs after exposure warrant patient triage to the delayed category.

**Minimal**

**Nerve agents**

Patients who are walking and talking can be assessed for the degree of miosis and its potential to interfere with performance prior to return to duty.

**Vesicants**

Patients with small exposures (< 5% total body surface area) or minor eye irritation can be triaged as minimal.

Table 1. Categories for Triage of Chemical Casualties (Open Table in a new window)
Chemical Warfare Agents

Nerve Agents

Nerve agents are organophosphates and therefore cause the characteristic cholinergic toxidrome. Organophosphates inhibit acetylcholinesterase, which results in an excess accumulation of acetylcholine at muscarinic and nicotinic receptors. Clinically, these agents cause increased bodily secretions (SLUDGE; salivation, lacrimation, urination, defecation, emesis), miosis, bronchospasm, increased airway secretions, sudden loss of consciousness, seizures, apnea, muscle fasciculations that progress to flaccid paralysis, and death. The primary cause of death in patients exposed to nerve agents is respiratory failure caused by respiratory muscle weakness, bronchoconstriction, and increased respiratory secretions. Maintain a high index of suspicion of organophosphate and/or nerve agent poisoning whenever presented with a patient who has pinpoint pupils with seizures, muscle fasciculations, or flaccid paralysis.

Depending on the patient's clinical status, treatment with atropine and pralidoxime may be feasible. Atropine dries secretions and relaxes smooth muscle via antagonism of acetylcholine at its muscarinic receptors. Clinically, this results in decreased SLUDGE and improved ventilation by decreasing bronchoconstriction and bronchorrhea. Pralidoxime improves the patient's muscle strength by removing the nerve agent from acetylcholinesterase. The acetylcholinesterase, when freed from the nerve agent, then resumes breakdown of acetylcholine. Pralidoxime's effectiveness decreases with time, because the enzyme/nerve agent complex can age and form an irreversible bond. The time course for this depends on the specific agent and can vary from minutes to hours.

Exposure to nerve agents occurs from either vapor or liquid forms. A patient's vapor or liquid exposure can be classified as mild, moderate, and severe based on clinical criteria.

Vapor exposure

Mild vapor exposure results in miosis and rhinorrhea. Patients also may develop eye pain, which can be treated with topical homatropine. Patients with only a mild vapor exposure do not require treatment with atropine or pralidoxime. Observe patients with mild vapor exposure and those who are asymptomatic but claim to have been exposed to a nerve agent vapor for 1 hour for development of muscle weakness, respiratory symptoms, or GI symptoms. If these symptoms develop, classify the patient as moderate; he or she requires treatment with atropine and pralidoxime. Severe vapor exposure results in loss of consciousness, seizures, flaccid paralysis, and apnea.
Liquid exposure

Mild liquid nerve agent exposure results in localized fasciculations and sweating. A mild liquid nerve agent exposure requires 1 dose of atropine and pralidoxime. Patients with mild liquid exposure, those who are asymptomatic but claim to have been exposed to a liquid nerve agent, and those with an unknown form of exposure require an observation period of 18 hours. If patients develop respiratory or GI symptoms, classify them as moderate. Loss of consciousness, seizures, flaccid paralysis, and apnea are manifestations of a severe exposure.

Antidotes

Treatment of nerve agent exposure is covered in detail elsewhere (see CBRNE - Nerve Agents, G-series: Tabun, Sarin, Soman, CBRNE - Nerve Agents, Binary: GB2, VX2, CBRNE - Nerve Agents, V-series: Ve, Vg, Vm, Vx).

Cyanide

Levels of exposure

Cyanide binds to ferric state iron found in mitochondria, which prevents the mitochondria from using oxygen. Because of the subsequent anaerobic metabolism, patients develop lactate accumulation with severe acidosis and cellular asphyxiation.

Cyanide can be found in vapor, liquid, and solid salt forms. Cyanide toxicity from chemical warfare typically occurs by inhalation of vapor, but it also may occur by ingestion or dermal contact with a large amount of the liquid form.

Cyanide poisoning at low concentrations produces anxiety, hyperventilation, headache, dizziness, and vomiting. Patients also may develop a cherry red skin discoloration, although this is rare. Since the body normally metabolizes cyanide into thiocyanate, which then is excreted in the urine, patients with mild exposures should improve when removed from the source of exposure and may not require any other intervention.

Cyanide poisoning at higher concentrations begins to produce respiratory distress. Suspect significant cyanide poisoning whenever confronted with a patient who is experiencing respiratory distress with normal oxygen concentration of the blood. This occurs because cyanide prevents the body from using oxygen. Toxicity screens for cyanide are not timely enough to be useful in patients with toxicity. A high venous oxygen level and lactic acidosis may help confirm suspicion of cyanide poisoning. Patients with this intermediate level of exposure may require treatment with amyl nitrite, sodium nitrite, and sodium thiosulfate.

Cyanide exposure at even higher concentrations can produce dramatic results. Patients can progress through anxiety and hyperpnea to seizures within 30 seconds. Within 3-5 minutes of exposure, patients may stop breathing. Finally, patients may experience asystole and death within 6-10 minutes.

Treatment

Treatment of cyanide involves removing cyanide from the cells, detoxifying cyanide, and providing supportive measures aimed at minimizing toxic effects. Treatment details are discussed elsewhere (see CBRNE - Cyanides, Cyanogen Chloride).

Vesicants

Vesicants are chemical agents that cause blistering of the skin. Lewisite and sulfur mustard are the 2 main agents used in chemical warfare. Exposure to either agent can come from a vapor or liquid form. Both agents produce irritation and damage to the eyes, skin, and airway. Chemical decontamination within 30 minutes of exposure is of primary importance, as that is when most of the injury to the patient occurs. Decontamination after that point still is required to prevent cross-contamination of healthcare providers.

Effects of exposure

Eyes: Eye exposure can produce mild conjunctivitis up to corneal perforation depending on the level of exposure. Treat eye exposures beyond decontamination in conjunction with ophthalmology.

Skin: Skin exposures result in vesicles, which coalesce into bullae. Bullae require surgical irrigation, debridement, and a topical antibiotic.

Respiratory: Airway injury initially damages the upper airway, resulting in epistaxis, pharyngitis, laryngitis, and bronchitis. With a high enough level of exposure, damage may progress to the lower airway. Damage to the lower respiratory tract produces dyspnea from bronchiolitis, which can progress to respiratory failure and death. Treatment of respiratory symptoms is supportive, with use of bronchodilators, oxygen, and mechanical ventilation if necessary.

Mustard also can produce bone marrow suppression beginning 3-5 days after exposure, which can contribute to a delayed cause of death. Time to onset of symptoms from exposure to lewisite is immediate. Conversely, mustard
exposure does not produce symptoms until 2-48 hours after exposure. For lewisite exposures, a specific antidote of dimercaprol (British antilewisite agent) can be administered in a dose of 2.5-5 mg/kg IM.

### Pulmonary Intoxicants

Pulmonary intoxicants cause lung injury via inhalation of a vapor. Chemical warfare agents in this group include phosgene and chlorine. Phosgene exposure causes a mild transient irritation of the eyes, sinuses, pharynx, and bronchi upon initial exposure. Patients then are asymptomatic until 2-4 hours postexposure, when they begin to develop noncardiogenic pulmonary edema, which should be treated with supplemental oxygen and mechanical ventilation as needed. These patients also may develop hypovolemic shock from loss of fluid into the alveolar space and may require intravenous fluids for hypotension. Since the pulmonary edema is noncardiogenic and the patients are hypovolemic, do not use diuretics.

Chlorine exposure has a pronounced, immediate irritant effect on the upper and lower respiratory tract, which may produce respiratory failure shortly after exposure. If the patient survives the initial exposure, he or she may develop noncardiogenic pulmonary edema and hypovolemic shock within 12-24 hours, similar to that caused by phosgene.

### Differential Diagnosis of Chemical Casualties

#### Table 2. Clinical Syndromes and Potential Chemical Etiologies* (Open Table in a new window)

<table>
<thead>
<tr>
<th>Category</th>
<th>Clinical Syndrome</th>
<th>Potential Chemical Etiology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholinergic crisis</td>
<td>• Salivation, diarrhea, lacrimation, bronchorrhea, diaphoresis, and/or urination</td>
<td>• Nicotine†</td>
</tr>
<tr>
<td></td>
<td>• Miosis, fasciculations, weakness, bradycardia or tachycardia, hypotension, or hypertension, and/or seizures</td>
<td>• Organophosphate insecticides†</td>
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<tr>
<td></td>
<td></td>
<td>• —decreased acetylcholinesterase activity</td>
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<td></td>
<td></td>
<td>• Carbamate insecticides</td>
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<tr>
<td></td>
<td></td>
<td>• Medicinal carbamates (eg, physostigmine)</td>
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<tr>
<td>Generalized muscle rigidity</td>
<td>• Seizurelike, generalized muscle contractions or painful spasms (neck and limbs) and usually tachycardia and hypertension</td>
<td>• Strychnine</td>
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<td></td>
<td></td>
<td>• —intact sensorium</td>
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<tr>
<td>Oropharyngeal pain and ulcerations</td>
<td>• Lip, mouth, and pharyngeal ulcerations and burning pain</td>
<td>• Paraquat†</td>
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<td></td>
<td></td>
<td>• — dyspnea and hemoptysis secondary to pulmonary edema or hemorrhage; can progress to pulmonary fibrosis over days to weeks</td>
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<td></td>
<td></td>
<td>• Diquat</td>
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<td></td>
<td></td>
<td>• Caustics (ie, acids and alkalis)</td>
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<td></td>
<td></td>
<td>• Inorganic mercuric salts</td>
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<td></td>
<td></td>
<td>• Mustards (eg, sulfur)</td>
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<tr>
<td>Cellular hypoxia</td>
<td>• Mild - Nausea, vomiting, and headache</td>
<td>• Cyanide† (eg, hydrogen cyanide gas, sodium cyanide)</td>
</tr>
<tr>
<td></td>
<td>• Severe - Altered mental status, dyspnea, hypotension, seizures, and metabolic acidosis</td>
<td>• —bitter almond odor§</td>
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<td></td>
<td></td>
<td>• Sodium monofluoroacetate (SMFA)†</td>
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<td></td>
<td></td>
<td>• —hypocalcemia or hypokalemia</td>
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<tr>
<td></td>
<td></td>
<td>• Carbon monoxide</td>
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<tr>
<td></td>
<td></td>
<td>• Hydrogen sulfide</td>
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<tr>
<td></td>
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<td>• Sodium azide</td>
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<td></td>
<td></td>
<td>• Methemoglobin-causing agents</td>
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<tr>
<td>Peripheral neuropathy and/or neurocognitive effects</td>
<td>• Peripheral neuropathy signs and symptoms - Muscle weakness and atrophy, &quot;glove and stocking&quot; sensory loss, and depressed or absent deep tendon reflexes</td>
<td>• Mercury (organic)†</td>
</tr>
<tr>
<td></td>
<td>• Neurocognitive effects - Memory loss,</td>
<td>• —visual disturbances, paresthesias, and/or ataxia</td>
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<tr>
<td></td>
<td></td>
<td>• Arsenic (inorganic)†</td>
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<tr>
<td></td>
<td></td>
<td>• —delirium and/or peripheral neuropathy</td>
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<tr>
<td>Chemical</td>
<td>Symptoms</td>
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<tr>
<td>Thallium</td>
<td>Delirium, ataxia, and/or encephalopathy</td>
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<tr>
<td>Lead</td>
<td>Delirium and/or peripheral neuropathy</td>
<td></td>
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<tr>
<td>Acrylamide</td>
<td>Encephalopathy and/or peripheral neuropathy</td>
<td></td>
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<tr>
<td>Severe gastrointestinal illness, dehydration</td>
<td>Abdominal pain, vomiting, profuse diarrhea (possibly bloody), and hypotension, possibly followed by multisystem organ failure</td>
<td></td>
</tr>
<tr>
<td>Arsenic†</td>
<td>Inhalation an additional route of exposure; severe respiratory illness possible</td>
<td></td>
</tr>
<tr>
<td>Ricin†</td>
<td>Colchicine</td>
<td></td>
</tr>
<tr>
<td>Barium</td>
<td>Hypokalemia common</td>
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</tr>
</tbody>
</table>

*Not intended as a complete differential diagnosis for each syndrome or a list of all chemicals that might be used in a covert chemical release.

†Potential agents for a covert chemical release based on historic use (ie, intentional or inadvertent use), high toxicity, and/or ease of availability.

§ Unreliable sign.

Chemical agents that may cause rapid onset of respiratory symptoms include nerve agents, cyanide, mustard, lewisite, and phosgene. Additionally, rapid onset of neurologic symptoms is observed following significant nerve agent or cyanide exposures. Treatment considerations in patients with rapid onset of respiratory or neurologic symptoms include the use of atropine, pralidoxime, and diazepam (typically by Autoinjector) followed by the administration of cyanide antidotes if the patient fails to respond to nerve agent antidotes.

Delayed onset of respiratory symptoms may be observed following mustard, lewisite, or phosgene exposure. Care in this setting is discussed in greater detail in other articles (see CBRNE - Vesicants, Mustard: Hd, Hn1-3, H, CBRNE - Vesicants, Organic Arsenicals: L, ED, MD, PD, HL, CBRNE - Lung-Damaging Agents, Phosgene).

**Summary**

Triage of casualties of chemical agents is based on many of the same principles as the triage of conventional casualties. Members of the triage team try to provide immediate care to those who need it for survival; they temporarily set aside or delay treatment of those who have minor injuries or do not need immediate medical intervention; and they do not use limited medical assets on the hopelessly injured. Initial triage and treatment of the chemical-exposed patient pose unique medical challenges, since early care must be rendered within the constraints of personal protective clothing. In addition, decontamination, which may be a time-consuming process, must be carried out before the casualty receives more definitive care. Triage is a matter of judgment on the part of the triage team. Ideally, base this judgment upon knowledge of medical assets, the casualty load, and most importantly, an understanding of the toxicology of the chemical agent involved and potential complications of exposure.

For excellent patient education resources, visit eMedicine's Bioterrorism and Warfare Center. Also, see eMedicine's patient education article Chemical Warfare.

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References


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