

SEDGWICK COUNTY EMERGENCY MEDICAL SERVICES



NBC FIELD

TREATMENT GUIDELINES



Statement from the Medical Director

The prospect of a nuclear, biological, or chemical attack on one or more communities in the United States brings the depravity of man into frightening focus. The need for this reference book is to prepare for a cataclysmic event that we hope will not occur. If it does, there will be widespread morbidity and mortality, which we cannot prevent. But, we can help to limit the result of such madness.

The guidelines presented here are to be used only upon the direct authorization of the medical director, his physician designate of record; or if the medical director cannot be reached or is incapacitated by the attack, by EMS 1, EMS 2, or EMS 3, who are herein authorized to serve as the designate to the medical director in the event of such an attack.

Ernest L. McClellan, MD
3/20/2000

Use & Reproduction of NBC Field Treatment Guidelines

The medical guidelines listed in this reference book are for use by pre-hospital care paramedics in the care and transportation of persons involved in nuclear, biological, or chemical attack. The treatment guidelines act as a reference in the event of a domestic attack. **Use of the guidelines may be obtained by 10-89 contact with the medical director or his designate, as specified above, upon suspicion of NBC involvement.** These treatment guidelines are **NOT** intended for day to day use.

Use or reproduction of these protocols is limited to Sedgwick County, KS EMS (SCEMS) paramedics. Use of these protocols may be granted to other advanced life support ambulance service paramedic personnel who are operating by SCEMS request to support SCEMS operations. Use of these guidelines by advanced life support agencies other than SCEMS must be granted at the time by 10-89 contact with the SCEMS medical director or his designate as specified above. Reproduction in any form of these guidelines is prohibited without the expressed written consent of Sedgwick County EMS.



CHEMICAL EXPOSURES

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Chlorine Background

Background

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 about two times heavier than air.

Terroristic Threat

No current chlorine weaponry known. May be used as a chemical asphyxiant. Dispersion must be in large concentration. May use projectiles, mortars, bombs, and large cloud release.

Health Effects

Chlorine exposure causes an immediate severe irritation of 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 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.

General

Exposures irritate eyes and central (upper) airways within minutes. Low doses produce some cough and choking sensation. Moderate doses may also produce a sense of suffocation, hoarseness, and substernal pain. High doses may also produce a severe dyspnea, with pulmonary edema, N/V, headache, syncope also is seen. Very high doses may produce sudden death without an obvious pulmonary lesion, possibly via laryngospasm. Treat all exposures with a minimum of observation!

Chlorine Treatment Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. Remove contaminated clothing and decontaminate patient.
2. Flush eyes with copious amounts of water for both gas and liquid exposure.
3. Flush skin with copious amounts of water for liquid exposures
4. ABCDE
 - a. High flow oxygen.

C. Breathing

1. If apneic: CPR with intubation. Laryngospasm may be present with intense exposures, making intubation difficult. Tracheostomy may be required.
2. If stridorous/hoarse: Consider intubation under direct vision since laryngospasm may be imminent. Be wary of sedation!
3. If dyspnea/cough/chest tightness: Consider intubation (be wary of sedation!) and be prepared to treat pulmonary edema. Bronchospasm may be present. Diminished air exchange may preclude wheezes. Remember that wheezes are a less reliable indicator in children.

D. If bronchospasm: Provide aggressive bronchodilation using SCEMS standing orders and protocols.

1. Albuterol per SCEMS Standing Orders and Authorized Range & Use.
2. SoluMedrol per SCEMS Authorized Range and Use

E. If asymptomatic, direct to medical observation for at least 1 hour. If becomes symptomatic, treat as above. If asymptomatic after 1 hour, lesser observation for 12 hours.

F. If pulmonary edema occurs, treat as non-cardiac pulmonary edema with PEEP 5-7 cm and/or intubation.

1. Use BVM to assist patient with respirations.
2. Do NOT use Lasix to treat chlorine induced pulmonary edema!

G. Transport to appropriate facility per SCEMS TPM.

Hydrocyanic Acid (AC) Background;

[Hydrogen Cyanide and Cyanogen Chloride (CK)]

Background

Hydrocyanic Acid (AC) and Cyanogen Chloride (CK) are liquids, but will vaporize (evaporate) at about 73°F (58°C), so they will be found in a 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. CK 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.

Terroristic Threat

AC has limited value because the amount needed is large, and because AC, being lighter than air, drifts away quickly. CK would be more effective due to its heavier than air characteristics. There is a potential that some cyanide munitions were abandoned at sites around the US. Iraq was believed to have used cyanide munitions against the Kurds in 1988.

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 oxygen deficiency. High concentrations of vapor may cause a brief increase in rate and depth of breathing (in 15 seconds), seizures (30 seconds), and cessation of breathing (3-5 minutes) and cardiac activity (4-10 minutes), and death. A smaller concentration will cause headache, flushing, lightheadedness, 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, because of hypoxia.

General

Patient should be moved from toxic environment immediately. 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; wash liquid off skin. 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. 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. Past military experience with respirators indicate some may not be protective past 30 minutes. Caution is RECOMMENDED!

Hydrocyanic Acid (AC) Protocol

[Hydrogen Cyanide and Cyanogen Chloride (CK)]

- A. Scene Size Up**
1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 2. What is the mechanism of illness/injury? Is it what it seems?
 3. How many patients are involved in this incident?
 4. What additional resources do I need to handle this patient?
- B. Primary Assessment**
1. If exposure was other than gas, remove contaminated clothing and decontaminate patient.
 2. **ABCDE**
 - a. **High flow oxygen** with appropriate adjunct.
- C. Mild Exposure** (Conscious, breathing):
1. Observe with oxygenation.
- D. Severe Exposure** (Unconscious, not breathing):
1. Continue high flow oxygen with BVM.
 2. Monitor EKG and SpO₂. Saturation will remain normal even in severe casualty until terminal stage; however, additional oxygen may assist in therapy. Of note is that pulse oximetry is completely unreliable in the presence of methemoglobinemia, which is induced by amyl nitrite or sodium nitrite therapy.
 3. If cyanide kit is available, follow directions to administer amyl nitrite crushed ampule in BVM every few minutes until IV access can be established.
 4. Establish IV access.
 5. If cyanide kit is available, administer 300 mg (10 ml) of sodium nitrite IV over 5 minutes. Flush line. Pediatric dose is 0.2—0.3 ml/kg, or 6-9 mg/kg of the 3% solution. No separate recommendation for infants.
 6. If cyanide kit is available, follow with 12.5 grams (50 ml) of sodium thiosulfate IV. Pediatric dose is 0.4 mg/kg, or 1.65 ml/kg of the 25% solution. No separate recommendation for infants. Use with care in patients with hypertension or heart disease. Repeat 1/2 dose in 20 minutes if no improvement.
 7. If patient remains apneic, intubate and continue oxygen delivery.
 8. Consider Sodium Bicarbonate for acidosis.
- E. Transport** to appropriate facility per SCEMS TPM.

Lewisite (L) Background

Background

Lewisite is a vesicant that damages the eyes, skin, and airways by direct contact. After absorption, it causes an increase in capillary permeability to produce hypovolemia, shock, and organ damage. Exposure to Lewisite causes immediate pain or irritation, although lesions require hours to become full-blown. Management of a Lewisite casualty is similar to management of a mustard casualty, although a specific antidote, British-Anti-Lewisite (BAL; dimercaprol) will alleviate some effects. Lewisite is an oily, colorless liquid with the odor of geraniums. It is more volatile than mustard.

Terroristic Threat

Lewisite was first synthesized in 1918 by Dr. Wilford Lee Lewis, but production was too late for its use in WW I. It has not been used in warfare, although it may be stockpiled by some countries. Lewisite is sometimes mixed with mustard to achieve a lower freezing point of the mixture for ground dispersal and aerial spraying.

Health Effects

Unlike mustard, Lewisite vapor or liquid causes immediate pain or irritation. A person with a droplet of Lewisite on his skin will note the burning and will immediately take steps to try to remove it. The vapor is so irritating that a person will seek to mask or to leave the contaminated area. 5 minutes after exposure to liquid Lewisite a grayish area of dead epithelium will result. Erythema and blister formation follow over the course of 12 to 18 hours and result in tissue necrosis and tissue sloughing. Eyes are painful with blepharospasm on contact. Edema of conjunctiva and lids follows, often swelling shut within the hour. Iritis and corneal damage may follow if dose is high. Liquid Lewisite causes severe eye damage within minutes of contact. Respiratory tract damage is dose related. Pulmonary edema may occur following inhalation. Systemic effects include permeability of systemic capillaries with resulting intravascular fluid loss, hypovolemia, shock, and organ congestion.

General

Triage the same as for mustard victims. Differential diagnosis is made by the fact that mustard does not have immediate pain from contact, whereas Lewisite does.

Lewisite Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?
- B. **Primary Assessment**
 - 1. For liquid or gas exposures, remove contaminated clothing and decontaminate patient with 0.5% hypochlorite solution, soap and cool water, or thoroughly flushed with water alone.
 - 2. ABCDE
 - a. High flow oxygen with appropriate adjunct.
- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam without delay of treatment.
- D. **Eyes:**
 - 1. Irrigate eye exposures with copious amounts of water.
- E. **Field Treatment**
 - 1. Field treatment is generally supportive after decontamination, as signs/symptoms do not appear until later.
 - 2. Patients with chemical pneumonitis or pulmonary edema:
 - a. Albuterol per SCEMS standing order and protocol.
 - b. Consider need for intubation.
 - c. Consider BVM for positive pressure ventilation.
 - 3. Patients presenting in hypovolemic shock should be treated per SCEMS standing orders/protocols for hypovolemic patients.
- F. **Transport** to appropriate facility per SCEMS TPM.

Methyl Isocyanate / Methylene Bisphenyl Isocyanate / Methylene Diisocyanate Background

Background

Methylene Bisphenyl Isocyanate (MDI) 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.

Terroristic Threat

Very large quantities of MDI are produced, transported, and used annually in the US. They are used in the production and usage of foams, lacquers, and sealants. MDI is a commonly used precursor in the industrial production of insecticides and laminating materials. There are no military designations or military unique uses listed in the reference material.

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 may result in permanent respiratory problems. Repeated or long-term exposure of the skin may cause a skin 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.

General

MDI is found as a solid, which has a melting point of 98.6° F (37° C). Vapor exposures occur with liquids containing dissolved solid. Gas exposures may occur with high-temperature volatilization. Thermal decomposition produces carbon monoxide and oxides of nitrogen. Sensitivity to this substance (eye, nose irritation) occurs at concentrations five times higher than OSHA limits (0.2 mg/m³), hence toxic exposures may go unrecognized.

Methyl Isocyanate / Methylene Bisphenyl Isocyanate / Methylene Diisocyanate Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?
- B. **Primary Assessment**
 - 1. For liquid or solid exposures, remove contaminated clothing and decontaminate patient with soap and water.
 - 2. ABCDE
 - a. High flow oxygen with appropriate adjunct.
- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam without delay of treatment.
- D. **Eyes:**
 - 1. Irrigate eye exposures with copious amounts of water.
- E. **Swallowing:**
 - 1. Liquids/solids should be removed by induced vomiting or lavage.
- F. **Breathing:**
 - 1. If apneic, start CPR
 - 2. Intubate patient if apneic. Consider intubation under direct vision if patient is stridorous/hoarse.
 - 3. If dyspnea/cough/chest tightness: Consider intubation for impending pulmonary edema or laryngospasm. Also consider possible bronchospasm which may limit air movement sufficient to preclude wheezing. Remember that infant and children anatomical differences make wheezing a less reliable indicator.
 - 4. If bronchospasm, treat aggressively! If present, treat with Albuterol, Solu Medrol, and Aminophylline per SCEMS standing orders and protocols. Note: Coughing indicates that the bronchospasm has most likely NOT been treated sufficiently.
 - 5. If Pulmonary Edema: Treat as non cardiac related pulmonary edema. Use Positive Pressure with BVM to assist patient respirations.
- G. **Transport** to appropriate facility per SCEMS TPM.

Mustard / (Sulfur Mustard) Background

Background

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, but 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.

Terroristic Threat

Mustard was used extensively in WW I and was the largest chemical casualty producer in that war. Mustard was used by Iraq against Iran in the 1980's. The US 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. Terroristic threat would be in the form of gas or liquid dispersal.

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 and 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 the stem cells with cessation of production of white cells, red cells, and platelets). There is no antidote. Epidemiologic 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.

General

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

Mustard / (Sulfur Mustard) Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?
- B. **Primary Assessment**
 - 1. For liquid or gas exposures, remove contaminated clothing and decontaminate patient with 0.5% hypochlorite solution, soap and cool water, or thoroughly flushed with water alone.
 - 2. ABCDE
 - a. High flow oxygen with appropriate adjunct.
- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam without delay of treatment.
- D. **Eyes:**
 - 1. Irrigate eye exposures with copious amounts of water.
- E. **Field Treatment**
 - 1. Field treatment is generally supportive after decontamination, as signs/symptoms do not appear until later.
 - 2. Patients with chemical pneumonitis:
 - a. Albuterol per SCEMS standing order and protocol.
 - b. Consider need for intubation.
 - c. Consider BVM for positive pressure ventilation.
- F. **Transport** to appropriate facility per SCEMS TPM.

Nerve Agents - Background

Tabun (GA), Sarin (GB); Soman (GD). GF and VX

Background

Nerve agents are very toxic organophosphorus compounds that have biological activity similar to that of many insecticides. Their volatilities range 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.

Terroristic Threat

Nerve agents were first synthesized pre-WW II, but were not used in that war. They were used by Iraq in its war with Iran. The US has a large stockpile of GA and VX in weapons; these are being destroyed. Terroristic deployment would be in gas, liquid, or powder form.

Health Effects

Nerve agents are the most toxic chemical agents. Initial effects from small amounts of agent differ depending on route of exposure. After a small vapor exposure, there is the immediate onset of effects in the eyes (small or pinpoint pupil (miosis), redness, eye pain, 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.

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. This results in a syndrome titled SLUDGE (Salivation, Lacrimation, Urination, Diaphoresis, Gastrointestinal Cramps, and Emesis).

Nerve Agents - Protocol

Tabun (GA), Sarin (GB); Soman (GD). GF and VX

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. For liquid or vapor exposures, remove contaminated clothing and decontaminate patient with soap and cool water.
2. ABCDE
 - a. High flow oxygen with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam without delay of treatment.
3. Determine in History & Physical if exposure to vapor (effects start within seconds to 1 or 2 minutes.)
4. Determine in History & Physical if exposure to liquid (effects start within minutes to 18 hours).

D. Field Treatment for Mild to Moderate Exposure

1. Dyspnea (following vapor exposure) and GI effects (following liquid exposure) should be treated with Atropine, 2 mg IM, or as specified in Mark I kit.
2. Repeat Atropine doses at 5 to 10 minute intervals until improvement or signs of Atropinization is noted.
3. DO NOT treat miosis or rhinorrhea unless severe .

E. Field Treatment for Severe Exposure:

1. Administer Atropine, 2 mg IM or IV, or as specified in Mark I kits. IM administration is better than IV in hypoxic patients. IM administration should be considered over IV in patients where hypoxia has NOT been at least partially corrected. Hypoxic patients can suffer ventricular fibrillation.
2. Treat seizures with Valium per SCEMS standing orders and protocols.
3. Repeat Atropine 2 mg IM or IV at 3-5 minute intervals until improvement or signs of Atropinization is noted.

F. Transport to appropriate facility per SCEMS TPM.

Phosgene (CX) Background

Background

Phosgene oxime (CX) is an urticant or nettle agent that causes a corrosive type of skin and tissue lesion. It is not a true vesicant, since it does not cause blisters. The vapor is extremely irritating, and both the vapor and liquid cause almost immediate tissue damage upon contact. There is very scanty information on phosgene oxime. CX is a solid at temperatures below 95°F, but the vapor pressure of the solid is high enough to produce symptoms. Traces of many metals cause it to decompose. However, it corrodes most metals.

Terroristic Threat

There is no current assessment of the potential of phosgene oxime as a military threat agent.

Health Effects

Skin: Phosgene oxime liquid or vapor causes pain on contact which is followed in turn by blanching with an erythematous ring in 30 seconds, a wheal in 30 minutes, and necrosis later. The extreme pain may persist for days. **Eyes:** Phosgene oxime is extremely painful to the eyes. The damage is probably similar to that caused by Lewisite. **Pulmonary:** Phosgene oxime is very irritating to the upper airways. This agent causes shortness of breath and pulmonary edema after inhalation and after skin application. **Other:** Some animal data suggest that phosgene oxime may cause hemorrhagic inflammatory changes in the gastrointestinal tract.

General

Phosgene effects cause immediate pain and irritation to all exposed skin and mucous membranes. The time course of damage to other tissue probably parallels that of damage to the skin. In making a differential diagnosis, other causes of urticaria and skin necrosis must be considered. Common urticants do not cause the extreme pain that phosgene oxime does.

Phosgene (CX) Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?
- B. **Primary Assessment**
 - 1. For liquid or gas exposures, remove contaminated clothing and decontaminate patient with soap and cool water, or thoroughly flushed with water alone.
 - 2. ABCDE
 - a. High flow oxygen with appropriate adjunct.
- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam without delay of treatment.
- D. **Eyes:**
 - 1. Irrigate eye exposures with copious amounts of water for at least 15 minutes.
- E. **Field Treatment**
 - 1. Field treatment is generally supportive after decontamination, as signs/symptoms do not appear until later.
 - 2. Patients with chemical pneumonitis:
 - a. Albuterol per SCEMS standing order and protocol.
 - b. Consider need for intubation.
 - c. Consider BVM for positive pressure ventilation.
- F. **Rapid Transport** to appropriate facility per SCEMS TPM.

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BIOLOGICAL EXPOSURES

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Definition of Weaponization: *This is the process of refining the bacteria/virus/toxin to amplify its effects, increase its virulence, and develop a vaccine for the aggressor.*

Anthrax Background

Background

Bacillus anthracis, the causative agent of Anthrax, is a rod-shaped, gram-positive, sporulating organism with the spores constituting the usual infective form. Anthrax is primarily a zoonotic disease of herbivores, with cattle, sheep and horse being the usual domesticated hosts, but other animals may be infected. Human disease may be contracted by handling contaminated hair, wool, hides, flesh, blood and the excreta of infected animals and from manufactured products such as bone meal, as well as by purposeful dissemination of spores. Infection is introduced through scratches or abrasions of the skin, wounds, inhalation of spores, eating insufficiently cooked meat, or by flies. The spores are very stable and may remain viable for many years in soil and water. They will resist sunlight for varying periods.

Terroristic Threat

Anthrax spores were weaponized by the US in the 1950's and 1960's before the old US offensive program was terminated. Other countries have weaponized this agent or are suspected of doing so. Anthrax bacterium is easy to cultivate and spore production is readily induced. Anthrax's resistance to sunlight, heat and disinfectants make it advantageous for use by terrorists. This agent can be produced in either a wet or dried form, stabilized for weaponization, and delivered as an aerosol cloud or as a point source from a spray device.

Health Effects

Anthrax presents as three distinct clinical syndromes in man: cutaneous, inhalational, and gastrointestinal disease. The cutaneous form occurs most frequently on the hands and forearms of persons working with infected livestock. It begins with a papule followed by formation of a blister-like fluid-filled vesicle. If the local infection becomes systemic it is often fatal. Endemic inhalational anthrax is a rare infection contracted by inhalation of the spores. It occurs mainly among workers handling infected hides, wool, and furs. The intestinal form, which is also rare in humans, is contracted by ingestion. The mortality of cutaneous anthrax in man is up to 25%. Inhalation and ingestion forms carry almost a 100% fatality rate.

General

Of note is that almost all inhalation anthrax cases in which treatment was begun after patients were significantly symptomatic have been fatal, regardless of treatment. Anthrax has an incubation period of 1-6 days, presumably dependent upon the dose and strain of inhaled organisms. Onset is gradual and nonspecific.

Anthrax - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - b. Don N-100 HEPA Mask and a cutaneous covering.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. For vapor, liquid or solid exposures, remove contaminated clothing and decontaminate patient with soap and cool water.
 - a. Use reverse isolation by covering the patient as possible.
2. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat symptoms utilizing SCEMS standing orders/protocols.

E. Transport to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Brucellosis Background

Background

The Brucellae are a group of gram-negative cocco-bacillary organisms, of which four species are pathogenic in humans. Abattoir and laboratory worker infections suggest that *Brucella* spp. Are highly infectious via the aerosol route. It is estimated that inhalation of only 10 to 100 bacteria is sufficient to cause disease in man. The relatively long and variable incubation period (5-60 days) and the fact that many infections are asymptomatic under natural conditions has made it a less desirable agent for weaponization, although large aerosol doses may shorten the incubation period and increase the clinical attack rate. Brucellosis infection has a low mortality rate (5% of untreated cases) with most deaths caused by endocarditis or meningitis. It is an incapacitating and disabling disease in its natural form.

Terroristic Threat

Not suited well for weaponization due to long and unpredictable incubation period. Mortality rate is low. It is unknown if large aerosol doses will overcome these drawbacks to using brucellosis as a weapon. Dispersal would most likely occur in a vapor form with low detection probability.

Health Effects

S/S for sub acute and acute brucellosis are non-specific and include: irregular fever, headache, profound weakness and fatigue, chills, sweating, arthralgias, and myalgias. Depression and mental status changes occur. Osteoarticular findings (i.e., sacroiliitis, vertebral osteomyelitis) also occur. Cough and pleuritic chest pain may occur in up to twenty percent of cases, but these are usually not associated with acute pneumonitis. GI symptoms occur in 70% of adult cases, and less frequently in children. Fatalities are uncommon.

General

Brucellosis appears as a nonspecific febrile illness which resembles influenza. Responders should protect themselves from airborne exposure, as well as BSI. Draining lesions allow for person to person transmission. The predominant use of brucellosis is as a fear weapon.

Brucellosis - Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - b. Don N-100 HEPA Mask.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?

- B. **Primary Assessment**
 - 1. For vapor, liquid or solid exposures, remove contaminated clothing and decontaminate patient with soap and cool water.
 - 2. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam.

- D. **Field Treatment**
 - 1. Treat symptoms utilizing SCEMS standing orders/protocols.

- E. **Transport** to appropriate facility per SCEMS TPM.

- F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Cholera Background

Background

Vibrio cholerae is a short, curved, motile, gram-negative, non-sporulating rod. There are two serogroups that have been associated with cholera in humans. The organisms are facultative anaerobes, growing best at a pH of 7.0, but able to tolerate an alkaline environment. The entire clinical syndrome is caused by the action of the toxin on the intestinal epithelial cell. Fluid loss in cholera originates in the small intestine with the colon being relatively insensitive to the toxin. The large volume of fluid produced in the upper intestine overwhelms the capacity of the lower intestine to absorb. Transmission is made through direct or indirect fecal contamination of water or foods, and by heavily soiled hands or utensils. All populations are susceptible, while natural resistance to infection is variable. Recovery from an attack is followed by a temporary immunity which may furnish some protection for years. The organism is easily killed by drying. It is not viable in pure water, but will survive up to 24 hours in sewage, and as long as 6 weeks in certain types of relatively impure water containing organic matter. It is readily killed by dry heat at 117 degree C, by steam and boiling, by short exposure to ordinary disinfectants and by chlorination of water. The rate of symptomatic to asymptomatic cases is 1:400.

Terroristic Threat

Has purportedly been investigated in the past as a biological weapon. Cholera does not easily spread from person to person. To be effective in a terroristic sense, major drinking water supplies would need to be heavily contaminated.

Health Effects

After an incubation period varying from 4 hours to 5 days, presumably dependent upon the dose of ingested organisms, onset is usually rather sudden, although the clinical manifestations range from an asymptomatic carrier state to severe illness. Cholera is characterized by sudden onset with nausea, vomiting, profuse watery diarrhea with 'rice water' appearance, the rapid loss of body fluids, toxemia, and frequent collapse. Mortality can range as high as 50% in untreated cases.

General

Treatment of cholera is primarily dependent upon replacement of fluids and electrolytes.

Cholera - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat symptoms utilizing SCEMS standing orders/protocols.
 - a. SCEMS orders for hypotension should be followed if patient exhibits S/S of shock.

E. **Transport** to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Glanders Background

Background

The causative agent of Glanders is *Burkholderia* (formerly *Pseudomonas*) *mallei*, a gram negative bacillus primarily noted for producing disease in horses, mules, and donkeys. In the past man has seldom been infected, despite frequent and often close contact with infected animals. This may be due to exposure to low concentrations of organisms from infected sites in sick animals and the fact that strains virulent for equids are often less virulent for man. There are four basic forms of disease in horses and man. Human cases have occurred primarily in veterinarians, horse and donkey caretakers, and abattoir workers. The organism spreads to man by invading the nasal, oral and conjunctival mucous membranes, by inhalation into the lungs, and by invading abraded or lacerated skin. Aerosols from cultures have been observed to be highly infectious to laboratory workers. Despite the rarity of contagion to man from infected horses and donkeys, the attack rates caused by laboratory aerosols have been as high as 46% and cases have been severe. Since aerosol spread is efficient, and there is no available vaccine or really dependable therapy, *B. mallei* has been viewed as a potential BW agent.

Terroristic Threat

Is viewed as a potential biowarfare/terroristic option. Spread will most likely be in an aerosol form that goes unnoticed at the time of dispersal.

Health Effects

Incubation period ranges from 10—14 days after inhalation. Inhalational exposure produces fever, rigors, sweats, myalgia, headache, pleuritic chest pain, cervical adenopathy, splenomegaly, and generalized papular/pustular eruptions. Almost always fatal without treatment.

General

Healthcare workers should practice standard precautions. Person to person airborne transmission is unlikely, although secondary cases may occur through improper handling of infected secretions. Hypochlorite solution is effective for decontamination using a 0.5% solution.

Glanders - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat symptoms utilizing SCEMS standing orders/protocols.

E. Transport to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Plague Background

Background

Yersinia pestis, a rod-shaped, non-motile, non-sporulating, gram-negative, bipolar staining, facultative, anaerobic bacterium causes plague, normally a zoonotic disease of rodents. Fleas that live on the rodents can sometimes pass the bacteria to human beings, who then suffer from the bubonic form of plague. The pneumonic form of the disease would be seen as the primary form after purposeful aerosol dissemination of the organisms. The bubonic form would be seen after purposeful dissemination through the release of infected fleas. All human populations are susceptible. Recovery from the disease may be followed by temporary immunity. The organism will probably remain viable in water and moist meals and grains for several weeks.

Terroristic Threat

Dispersal by aerosol or through the release of infectious fleas. Aerosol dispersal is theoretical at this point in time.

Health Effects

Plague incubates in 2-3 days. High fever, chills, headache, hemoptysis, and toxemia, progressing rapidly to dyspnea, stridor, and cyanosis. Death from respiratory failure, circulatory collapse, and a bleeding diathesis. Bubonic plague incubates in 2– 10 days. Malaise, high fever, and tender lymph nodes (buboes); may progress spontaneously to the septicemic form, with spread to CNS, lungs, etc.

General

Untreated pneumonic plague in man carries a mortality rate of 100%. Untreated bubonic plague, acquired from flea bites, has an approximately 50% mortality rate.

Technicians should use standard precautions when exposed to bubonic plague, and droplet precautions when exposed to pneumonic plague. Heat, disinfectants (2-5% hypochlorite) and exposure to sunlight renders bacteria harmless.

Plague - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat symptoms utilizing SCEMS standing orders/protocols.
2. Usual supportive therapy required includes IV crystalloids and hemodynamic monitoring per SCEMS orders.

E. **Transport** to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Tularemia Background

Background

Francisella tularensis, the causative agent of tularemia, is a small, aerobic non-motile, gram-negative cocco-bacillus. Tularemia (also known as rabbit fever and deer fly fever) is a zoonotic disease which humans typically acquire after contact of their skin or mucous membranes with tissues or body fluids of infected animals, or from bites of infected deerflies, mosquitoes, or ticks. Less commonly, inhalation of contaminated dusts or ingestion of contaminated foods or water may produce clinical disease. Respiratory exposure by aerosol would cause typhoidal or pneumonic tularemia. *F. tularensis* can remain viable for weeks in water, soil, carcasses, and hides, and for years in frozen rabbit meat. It is resistant for months to temperatures of freezing and below. It is rather easily killed by heat and disinfectants.

Terroristic Threat

Tularemia was weaponized by the US during its biowarfare program. Other countries are suspected to have weaponized this agent. This agent can be dispersed in either a wet or dry form.

Health Effects

Ulceroglandular tularemia presents with a local ulcer and regional lymphadenopathy, fever, chills, headache and malaise. Typhoidal tularemia presents with fever, headache, malaise, substernal discomfort, prostration, weight loss and a non-productive cough. Pneumonia may be associated with any form, but is most common in typhoidal tularemia.

General

Standard precautions are recommended for healthcare workers. Infection generally occurs through inoculation of the skin or mucous membranes with infectious matter.

Tularemia - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat symptoms utilizing SCEMS standing orders/protocols.

E. Transport to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Q Fever Background

Background

The endemic form of Q fever is a zoonotic disease caused by a rickettsia, *Coxiella burnetii*. Its natural reservoirs are sheep, cattle and goats, and grows to especially high concentrations in placental tissues. Exposure to infected animals at parturition is an important risk factor for endemic disease. The organisms are also excreted in animal milk, urine, and feces. Humans acquire the disease by inhalation of aerosols contaminated with the organisms. Farmers and abattoir workers are at greatest risk occupationally. A biological warfare attack with Q fever would cause a disease similar to that occurring naturally. Q fever is also a significant hazard in laboratory personnel who are working with the organism.

Terroristic Threat

Q fever is very infectious. A single inhaled organism may produce clinical illness. For this reason, Q fever could be used in a biowarfare setting as an incapacitating agent.

Health Effects

Incubation period of 10—40 days. S/S include fever, cough, and pleuritic chest pain that may occur as early as ten days after exposure. Patients are not generally critically ill, and the illness lasts from 2 days to 2 weeks. Pneumonia occurs in half of all patients, but only half of these, or 25% of all patients, will have a cough or rales.

General

Standard precautions are recommended for healthcare workers. Direct transmission from person to person is rare.

Q Fever - Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?

- B. **Primary Assessment**
 - 1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam.

- D. **Field Treatment**
 - 1. Treat symptoms utilizing SCEMS standing orders/protocols.

- E. **Transport** to appropriate facility per SCEMS TPM.

- F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Smallpox Background

Background

Variola virus causes smallpox. It is an orthopox virus and occurs in at least two strains, variola major and the milder disease, variola minor. Despite the global eradication of smallpox and continued availability of a vaccine, the potential weaponization of variola continues to pose a military threat. This threat can be attributed to the aerosol infectivity of the virus, the relative ease of large-scale production, and an increasingly *Orthopoxvirus* naïve populace. Although the fully developed cutaneous eruption of smallpox is unique, earlier stages of the rash could be mistaken for varicella (Chicken Pox). Secondary spread of infection constitutes a nosocomial hazard from the time of onset of a smallpox patient's exanthem until scabs have separated. Quarantine with respiratory isolation should be applied to secondary contacts for 17 days post exposure.

Terroristic Threat

Because vaccination has not occurred since the 1980's, populations are at risk for smallpox. It is suspected that this virus has been weaponized.

Health Effects

Incubation period is an average of 12 days. Clinical manifestations begin acutely with malaise, fever, rigors, vomiting, headache, and backache. 15% of patients developed delirium. Approximately 10% of light skinned patients exhibited an erythematous rash during this phase. Two to three days later, an enanthem appeared concomitantly with a discrete rash about the face, hands and forearms.

General

Droplet and airborne precautions for a minimum of 16 to 17 days following exposure for ALL contacts. Patients should be considered infectious until all scabs separate. The potential for airborne spread to other than close contacts is controversial, but should be considered a real possibility. In general, close person to person proximity is required for transmission to reliably occur.

Smallpox - Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - b. Don N-100 HEPA mask.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?

- B. **Primary Assessment**
 - 1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam.

- D. **Field Treatment**
 - 1. Treat symptoms utilizing SCEMS standing orders/protocols.

- E. **Transport** to appropriate facility per SCEMS TPM.

- F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Venezuelan Equine Encephalitis Background (VEE)

Background

Venezuelan equine encephalitis (VEE) virus is an arthropod-borne alphavirus that is endemic in northern South America, Trinidad, Central America, Mexico, and Florida. Eight serologically distinct viruses belonging to the VEE complex have been associated with human disease; the two most important of these pathogens are designated subtype I, variants A/B and C. These agents also cause severe disease in horses, mules, burros and donkeys (Equidae). Natural infections are acquired by the bites of a wide variety of mosquitoes. Equidae serve as amplifying hosts and source of mosquito infection. In natural human epidemics, severe and often fatal encephalitis in Equidae always precedes disease in humans. The virus is rather easily killed by heat and disinfectants.

Terroristic Threat

VEE was weaponized by the US in its biowarfare program. Other countries are suspected to have weaponized this virus as well. This virus could theoretically be produced in either a wet or dry form and potentially stabilized for weaponization.

Health Effects

Incubation period is 2-6 days with a sudden onset of generalized malaise, spiking fevers, rigors, severe headache, photophobia, and myalgias. Nausea, vomiting, cough, sore throat, and diarrhea may follow. The case fatality rate is less than 1 %, although is somewhat higher in the very young or aged. Nearly 100% of those infected suffer an overt illness.

General

Standard precautions are recommended for healthcare workers. Person to person transmission may theoretically occur by means of respiratory droplet infection.

VEE - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat symptoms utilizing SCEMS standing orders/protocols.

E. Transport to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Viral Hemorrhagic Fevers Background

Background

The viral hemorrhagic fevers are a diverse group of human illnesses that are due to RNA viruses from several different viral families: the Filoviridae, which consists of Ebola and Marburg viruses; the Arenaviridae, including Lassa fever, Argentine and Bolivian hemorrhagic fever viruses; the Bunyaviridae, including various members from the Hantavirus genus, Congo-Crimean hemorrhagic fever virus from the Nairovirus genus, and Rift Valley fever from the Phlebovirus genus; and Flaviviridae, such as Yellow fever virus, Dengue hemorrhagic fever virus, and others. The viruses may be spread in a variety of ways, and for some there is a possibility that humans could be infected through a respiratory portal of entry. Although evidence for weaponization does not exist for many of these viruses, many are included in this handbook because of their potential for aerosol dissemination or weaponization, or likelihood for confusion with similar agents which might be weaponized.

Terroristic Threat

All of these diseases are potential biowarfare/terrorist agents. With the exception of the dengue virus, all of these viruses are infectious by aerosol or fomites. Since most patients are viremic, there is a potential for nosocomial transmission to patients, medical staff, and particularly laboratory personnel.

Health Effects

S/S of viral hemorrhagic fevers are febrile illnesses which can be complicated by easy bleeding, petechiae, hypotension and even shock, flushing of the face and chest, and edema. Constitutional symptoms such as malaise, myalgias, headache, vomiting, and diarrhea may occur in any of the hemorrhagic fevers. Common presenting complaints are fever, myalgia, and prostration; clinical examination may reveal only conjunctival injection, mild hypotension, flushing, and petechial hemorrhages. Full-blown VHF typically evolves to shock and generalized mucous membrane hemorrhage and often is accompanied by evidence of neurologic, hematopoietic, or pulmonary involvement.

General

Contact precautions for healthcare workers. Decontamination is accomplished with hypochlorite or phenolic disinfectants. Isolation measures and barrier nursing procedures are indicated (Described in appendix). VHF should be suspected in any patient presenting with a severe febrile illness and evidence of vascular involvement who has traveled to an area where the virus is known to occur, or where intelligence information suggests a biological warfare threat.

Viral Hemorrhagic Fevers - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat signs/symptoms utilizing SCEMS standing orders/protocols.
2. Viral Hemorrhagic Fevers may produce profound shock. Be prepared to treat shock with SCEMS standing orders/protocols.

E. Transport to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Botulinum Background

Background

Botulinum toxins are a group of seven related neurotoxins produced by the bacillus *Clostridium botulinum*. These toxins, types A through G, could be delivered by aerosol over concentrations of troops. When inhaled, these toxins produce a clinical picture very similar to foodborne intoxication, although the time to onset of paralytic symptoms may actually be longer than for foodborne cases, and may vary by type and dose of toxin. The clinical syndrome produced by one or more of these toxins is known as “botulism”.

Terroristic Threat

It is feasible to deliver botulinum toxins as a biological weapon, and other countries have weaponized or are suspected to have weaponized one or more of this group of toxins. Iraq admitted to a UN inspection team in August of 1991 that it had done research on the offensive use of botulinum toxins prior to the Persian Gulf War, which occurred in January and February of that year. Iraq has not only researched the use of the toxin as a weapon, but has filled and deployed over 100 munitions with botulinum toxin.

Health Effects

Onset of symptoms will vary from 24 to 36 hours, to several days following exposure. Lower doses have a longer incubation period than larger doses in recent primate studies. S/S include 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.

General

Standard precautions for healthcare workers. Toxin is not dermally active and secondary aerosols are not a hazard from patients. In cases of dermal exposure to infectious material, hypochlorite (0.5% for 10-15 minutes) and/or soap and water for decontamination.

Botulinum - Protocol

- A. **Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?

- B. **Primary Assessment**
 - 1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

- C. **Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam.

- D. **Field Treatment**
 - 1. Treat signs/symptoms utilizing SCEMS standing orders/protocols.
 - 2. Be prepared to assist patient in ventilations with BVM and endotracheal intubation.

- E. **Transport** to appropriate facility per SCEMS TPM.

- F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Staphylococcal Enterotoxin B Background

Background

Staphylococcus aureus produces a number of exotoxins, one of which is Staphylococcal enterotoxin B, or SEB. Such toxins are referred to as exotoxins since they are excreted from the organism; however, they normally exert their effects on the intestines and thereby are called enterotoxins. SEB is one of the pyrogenic toxins that commonly causes food poisoning in humans after the toxin is produced in improperly handled foodstuffs and subsequently ingested. SEB has a very broad spectrum of biological activity. This toxin causes a markedly different clinical syndrome when inhaled than it characteristically produces when ingested. Significant morbidity is produced in individuals who are exposed to SEB by either portal of entry to the body.

Terroristic Threat

Although this toxin would not be likely to produce significant mortality on the battlefield, it could render up to 80% or more of exposed personnel clinically ill and unable to perform their duties for 1-2 weeks. Therefore, SEB is not generally thought of as a lethal agent. It may severely incapacitate a population, thereby making it an extremely important toxin to consider. This toxin could be used (theoretically) in a terrorist mode to sabotage food or small volume water supplies.

Health Effects

S/S appear from 3 to 12 hours after aerosol exposure. Sudden onset of fever, chills, headache, myalgia, and nonproductive cough occur. Some patients may develop shortness of breath and retrosternal chest pain. Fever may last 2 to 5 days, and cough may persist for up to 4 weeks. Patients may also present with nausea, vomiting, and diarrhea if they swallow the toxin. Presumably, higher exposure can lead to septic shock and death.

General

Standard precautions for healthcare workers. Hypochlorite (0.5% for 10-15 minutes) and/or soap and water. Destroy any food that may have been contaminated.

Staphylococcal Enterotoxin B (SEB) - Protocol

- A. Scene Size Up**
 - 1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - 2. What is the mechanism of illness/injury? Is it what it seems?
 - 3. How many patients are involved in this incident?
 - 4. What additional resources do I need to handle this patient?

- B. Primary Assessment**
 - 1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

- C. Secondary Assessment:**
 - 1. Monitor SpO₂ and EKG.
 - 2. Complete history & physical exam.

- D. Field Treatment**
 - 1. Treat signs/symptoms utilizing SCEMS standing orders/protocols.
 - 2. Be prepared to assist patient in ventilations with BVM and endotracheal intubation.
 - 3. Attention to fluid management using SCEMS standing orders/protocols is important.

- E. Transport** to appropriate facility per SCEMS TPM.

- F.** If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Ricin Background

Background

Ricin is a potent protein toxin derived from the beans of the castor plant (*Ricinus communis*). Castor beans are ubiquitous worldwide, and the toxin is fairly easily produced. Ricin is therefore a potentially widely available toxin. When inhaled as a small particle aerosol, this toxin may produce pathologic changes within 8 hours and severe respiratory symptoms followed by acute hypoxic respiratory failure in 36—72 hours. When ingested, ricin causes severe gastrointestinal symptoms followed by vascular collapse and death. This toxin may also cause disseminated intravascular coagulopathy, microcirculatory failure and multiple organ failure if given intravenously in laboratory animals.

Terroristic Threat

Ricin's potential biological warfare significance relates in part to its wide availability. Ricin has been used in an assassination through injection of a small pellet. This toxin could be distributed in a variety of forms.

Health Effects

Weakness, fever, cough and pulmonary edema occur 18-24 hours after inhalation exposure, followed by severe respiratory distress and death from hypoxemia in 36—72 hours. Non inhalation routes of exposure do not cause lung irritation.

General

Standard precautions for healthcare workers. Secondary aerosols should generally not be a danger to health care providers. Weak hypochlorite solutions (0.1% sodium hypochlorite) and/or soap and water can decontaminate skin surfaces. Ricin is one of the most toxic substances known to man.

Ricin - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - b. If known contamination, decontaminate patient and rescuers.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat signs/symptoms utilizing SCEMS standing orders/protocols.
2. Treat pulmonary edema utilizing SCEMS standing orders/protocols.
3. Be prepared to assist patient in ventilations with BVM and endotracheal intubation.
4. Attention to fluid management using SCEMS standing orders/protocols is important.
5. Gastrointestinal intoxication is best managed by vigorous gastric decontamination with activated charcoal using SCEMS standing orders/protocols.

E. Transport to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

T-2 Mycotoxins Background

Background

The trichothecene mycotoxins are low molecular weight, nonvolatile compounds produced by filamentous fungi (molds) of the genera *Fusarium*, *Myrotecium*, *Trichoderma*, *Stachybotrys* and others. The structures of approximately 150 trichothecene derivatives have been described in the literature. These substances are relatively insoluble in water but are highly soluble in ethanol, methanol and propylene glycol. The trichothecenes are extremely stable to heat and ultraviolet light inactivation. Heating to 1500° F for 30 minutes is required for inactivation, while brief exposure to NaOCl destroys toxic activity. The potential for use as a BW toxin was demonstrated to the Russian military shortly after WW II when flour contaminated with species of *Fusarium* was unknowingly baked into bread that was ingested by civilians. Some developed a protracted lethal illness called alimentary toxic aleukia.

Terroristic Threat

Mycotoxins allegedly have been used in aerosol form (“yellow rain” or “yellow powder”) to produce lethal and non-lethal casualties in Laos, Kampuchea, and Afghanistan. Attack should be suspected if an aerosol attack occurs in the form of “yellow rain” with droplets of yellow fluid contaminating clothes and the environment.

Health Effects

Exposure causes skin pain, pruritus, redness, vesicles, necrosis and sloughing of epidermis. Effects on the airway include nose and throat pain, nasal discharge, itching and sneezing, cough, dyspnea, wheezing, chest pain and hemoptysis. Toxin also produces effects after ingestion or eye contact. Severe poisoning results in prostration, weakness, ataxia, collapse, shock, and death.

General

Standard precautions for healthcare workers. The only defense against exposure to T-2 Mycotoxins is to wear a protective mask and clothing during an attack. No specific immunotherapy exists and there is no specific antidote.

T-2 Mycotoxin - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - b. If known contamination, decontaminate patient and rescuers.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat signs/symptoms utilizing SCEMS standing orders/protocols.
2. Be prepared to assist patient in ventilations with BVM and endotracheal intubation.
4. Attention to fluid management using SCEMS standing orders/protocols is important.
5. Gastrointestinal intoxication is best managed by vigorous gastric decontamination with activated charcoal using SCEMS standing orders/protocols.

E. Transport to appropriate facility per SCEMS TPM.

F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

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NUCLEAR EXPOSURES

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Nuclear Exposure

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Nuclear Background

Background

Ionizing radiation presents in a number of different forms. Alpha, Beta, and Gamma level radiation, as well as neutrons. Alpha particles are slow moving, low energy particles that usually can be stopped by such things as clothing and paper. When they contact the skin, they only penetrate a few cells deep. They can produce serious effects if ingested or inhaled. Beta particles are smaller than alpha particles and are higher in energy. They can be stopped by aluminum and similar materials. They generally cause less local damage than alpha particles, but can be harmful if ingested or inhaled. Gamma rays are more highly energized and penetrating than alpha and beta particles. The origin of gamma rays is related to that of X-rays. Gamma radiation is extremely dangerous, carrying high levels of energy capable of penetrating thick shielding. They easily pass through clothing and the entire body, inflicting extensive cell damage and cause indirect damage by causing internal tissue to emit alpha and beta particles. Neutrons are more penetrating than the other types of radiation (3 to 10 times that of gamma rays), but less dangerous than the internal hazard associated with ingestion of alpha and beta particles. Exposure to neutrons causes direct tissue damage. Not normally a problem for paramedics, as they generally occur only near a reactor core.

Terroristic Threat

Considered less than that of chemical or biological possibilities due to the expense of and limited availability of radioactive materials. Likely dispersal would be by conventional explosion which does not create a nuclear reaction, but does spread radioactive materials resulting in contamination.

Health Effects

See Page 52

General

Dosing is a function of Time, Distance, Shielding & Source. Technicians should gain this information in the scene size up phase of response if possible. Bunker gear provides some level of protection in the case of alpha and beta radiation. Dust/Mist/Fume masks are sufficient to block radioactive dust.

RAD - Radiation Absorbed Dose: Unit of local tissue energy deposition.

REM - Roentgen Equivalent in Man: Provides a gauge of the likely injury to the irradiated part of an organism.

Nuclear - Protocol

A. Scene Size Up

1. Assess scene safety - Do not enter without proper protection.
 - a. Remove patient from hazardous environment before TX.
 - b. If external contamination, decontaminate patient and rescuers.
 - c. Use dosimeters and survey instruments when available.
 - d. Use bunker gear to provide limited protection against radiation.
 - e. Use particulate mask to protect your airway.
2. What is the mechanism of illness/injury? Is it what it seems?
3. How many patients are involved in this incident?
4. What additional resources do I need to handle this patient?

B. Primary Assessment

1. ABCDE
 - a. Oxygen as indicated with appropriate adjunct.

C. Secondary Assessment:

1. Monitor SpO₂ and EKG.
2. Complete history & physical exam.

D. Field Treatment

1. Treat signs/symptoms utilizing SCEMS standing orders/protocols.
 - a. Externally radiated patients pose little danger to pre-hospital personnel.
 - b. Internally contaminated patients (ingested or inhaled) pose little danger to rescue personnel.
2. If radioactive particle inhalation is suspected, swab nasal passages and save swabs in bag for hand off to ER.
3. Externally contaminated patients require decontamination prior to treatment.
4. Avoid cross contamination of open wounds.

E. Transport to appropriate facility per SCEMS TPM.

- F. If outbreak/epidemic has not been recognized/declared and you suspect such, notify ER personnel and SCEMS Operations staff of suspicions.

Dose-Effect Relationships to Ionizing Radiation

Whole Body Exposure Dose (RAD)	Effect
5-25	Asymptomatic. Blood studies are normal.
50-75	Asymptomatic. Minor depressions of white blood cells and platelets in a few patients.
75-125	May produce anorexia, nausea, and vomiting, and fatigue in approximately 10-20% of patients within two days.
125-200	Possible nausea and vomiting. Diarrhea, anxiety, tachycardia. Fatal to less than 5% of patients.
200-600	Nausea and vomiting, diarrhea in the first several hours, weakness, fatigue. Fatal to approximately 50% of patients within six weeks without prompt medical attention.
600-1,000	Severe nausea and vomiting, diarrhea in the first several hours. Fatal to 100% of patients within two weeks without prompt medical attention.
1,000 or more	"Burning sensation" within minutes, nausea and vomiting within 10 minutes, confusion ataxia, and prostration within one hour, watery diarrhea within 1-2 hours. Fatal to 100% within short time without prompt medical attention.
Localized Exposure Dose (RAD)	Effect
50	Asymptomatic
500	Asymptomatic (usually). May have risk of altered function of exposed area.
2,500	Atrophy, vascular lesion, and altered pigmentation.
5,000	Chronic ulcer, risk of carcinogenesis.
50,000	Permanent destruction of exposed tissue.

Referenced from Brady Paramedic Emergency Care, Third Edition.

APPENDIX

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Appendix A: **Patient Isolation Precautions**

Standard Precautions (Apply as possible in pre-hospital setting)

- Hand washing after patient contact.
- Use of gloves when touching blood, body fluids, secretions, excretions and contaminated items.
- Use of mask, eye protection, and gown during procedures likely to generate splashes or sprays of blood, body fluids, secretions or excretions
- Handle contaminated patient-care equipment and linen in a manner that prevents the transfer of microorganisms to people or equipment.
- Practice care when handling sharps and use a mouthpiece or other ventilation device as an alternative to mouth-to-mouth resuscitation when practical.
- Place the patient in a private area when feasible if they may contaminate the environment.

Airborne Precautions (Apply as possible in pre-hospital setting)

Standard Precautions plus:

- Place the patient in a private area that has negative air pressure, at least six air changes/hour, and appropriate filtration of air before it is discharged from the room.
- Use of respiratory protection when entering the room.
- Limit movement and transport of the patient. Use a mask on the patient if they need to be moved.

Droplet Precautions (Apply as possible in pre-hospital setting)

Standard Precaution plus:

- Place the patient in a private area or with someone with the same infection. If not feasible, maintain at least 3 feet between patients.
- Use of a mask when working within 3 feet of the patient.
- Limit movement and transport of the patient. Use a mask on the patient if they need to be moved.

Contact Precautions (Apply as possible in pre-hospital setting)

Standard Precautions plus:

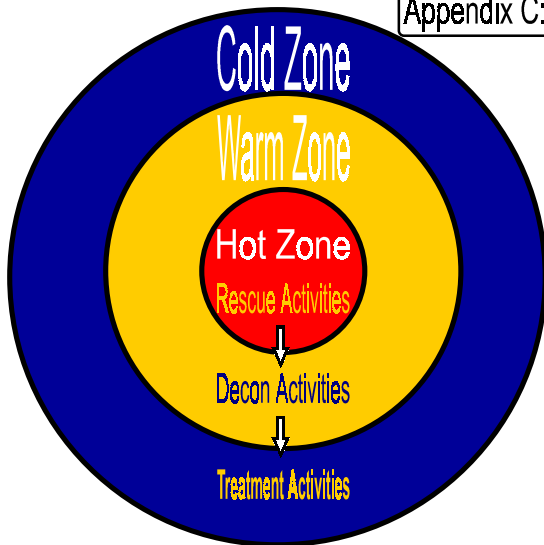
- Place the patient in a private area or with someone with the same infection if possible.
- Use of gloves when entering the area. Change gloves after contact with infective material.
- Use of gown when entering the area if contact with patient is anticipated or if the patient has diarrhea, a colostomy or wound drainage not covered by a dressing.
- Limit the movement or transport of the patient from the area.
- Ensure that patient-care items, bedside equipment, and frequently touched surfaces receive daily cleaning.
- Dedicate use of non-critical patient-care equipment to a single patient, or cohort of patients with the same pathogen. If not feasible, adequate disinfection between patients is necessary.

Appendix B:

Differential Diagnosis of Chemical Nerve Agent, Botulinum Toxin and SEB Intoxication following Inhalation Exposure

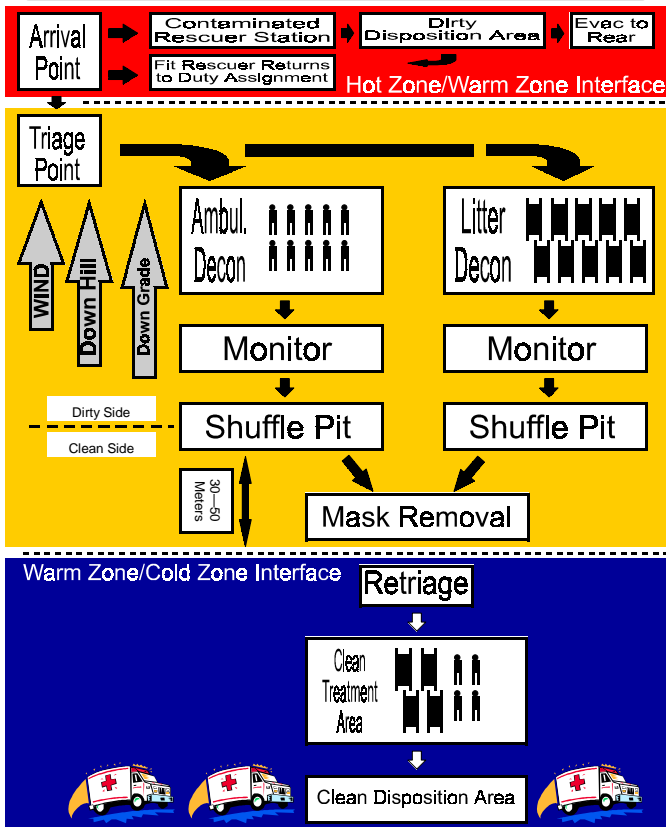
	<u>Chemical Nerve Agent</u>	<u>Botulinum Toxin</u>	<u>SEB</u>
Time to Symptoms	Minutes	Hours (12-48)	Hours (1-6)
Nervous	Convulsions, Muscle twitching	Progressive paralysis	Headache Muscle aches
Cardiovascular	Slow heart rate	Normal rate	Normal or Rapid Heart Rate
Respiratory	Difficult breathing, airways constriction	Normal, then progressive paralysis	Nonproductive cough Severe cases; chest pain/difficult breathing
Gastrointestinal	Increased motility, pain, diarrhea	Decreased motility	Nausea, vomiting and/or diarrhea
Ocular	Small pupils	Droopy eyelids	May see "red eyes" (conjunctiva infection)
Salivary	Profuse, watery Saliva	Normal; difficulty swallowing	May be slightly increased quantities of saliva
Death	Minutes	2-3 days	Unlikely
Response to Atropine/2PAM-CI	Yes	No	Atropine may reduce gastrointestinal symptoms

Appendix C: Zones

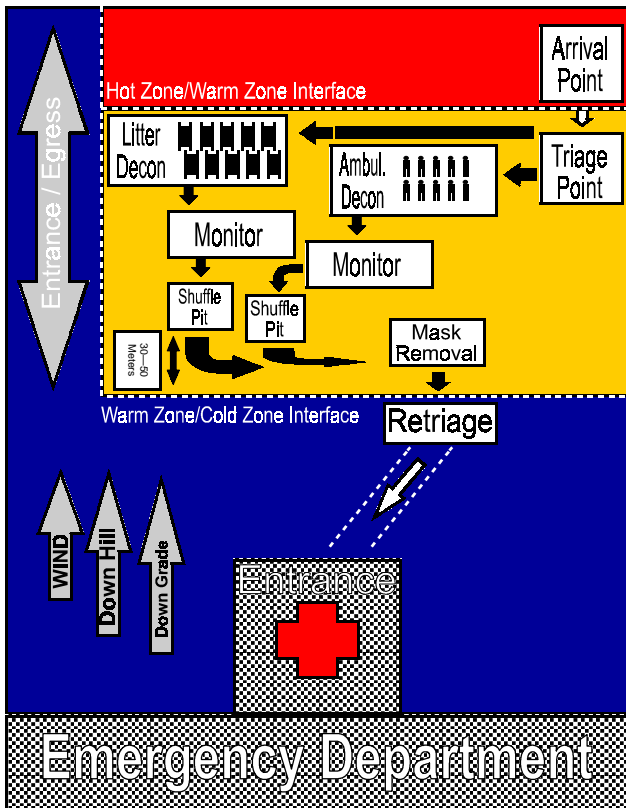


Zone	Actions	Personnel
Hot	Rescue	Haz Mat Techs
Warm	Decontamination	Fire/Rescue
Cold	Treatment	EMS

Appendix D: CASUALTY DECON

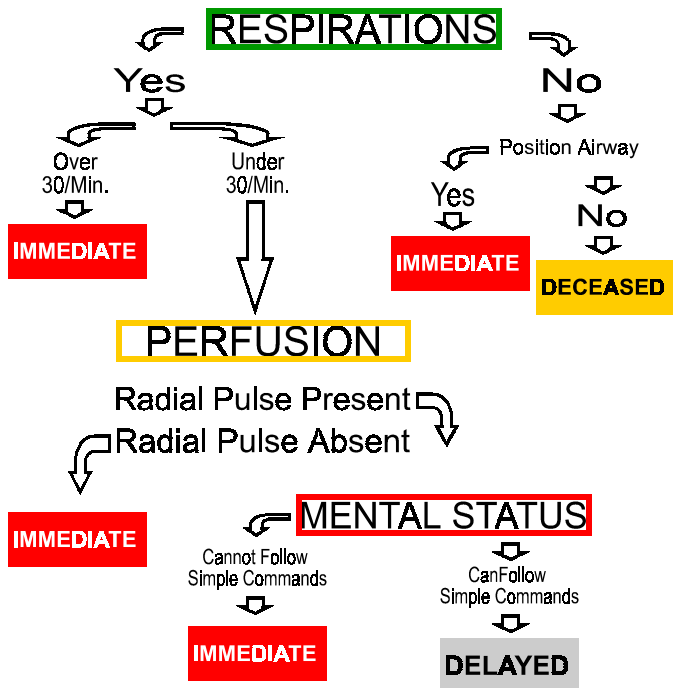


Appendix E: HOSPITAL CASUALTY DECON



Appendix F: START Triage

****For use in Mass Casualty Situations****



Appendix G: Pralidoxime Chloride (2-PAMCl)

Actions: An anticholinesterase antagonist that reactivates cholinesterase inhibited by phosphate esters. A chemical reaction with anticholinesterases and depolarization at the neuromuscular junction also takes place. Rapidly absorbed and well dispersed throughout body fluids. Most of a single dose is excreted within 6 hours in the urine.

Indications and Uses: Antidote for anticholinesterase drug or chemical overdose or poisoning. Primarily useful for many phosphate ester insecticide poisons with anticholinesterase activity (e.g., parathion).

Usual Dose: 600 mg IM from Mark 1 kit autoinjector. This follows the 2 mg IM dose of atropine from the Mark 1 kit autoinjector. If S/S continue, additional doses can be given with extreme caution. Atropine must be given before pralidoxime, but after adequate ventilation has been established. Ventricular fibrillation can occur if oxygenation is inadequate. This is more likely to occur when given IV than when given IM.

Dose Adjustments: Reduce dose in renal impairment.

Contraindications: None when indicated. Increases toxicity of Sevin (Carbamate insecticide). Effects in pregnancy are not known. Use only if clearly needed in pregnant patients.

Drug /Lab Interactions: Morphine, theophylline, (aminophylline), succinylcholine, reserpine compounds, and phenothiazines are contraindicated. May defeat effectiveness. Potentiates barbituates.

Side Effects: Usually minor and transient: blurred vision, diplopia, dizziness, headache, impaired accommodation, laryngospasm, muscle rigidity, nausea, pharyngeal pain, tachycardia.

Appendix H: Reference Listing

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