CBRNE Rapid Reference Guide

August 2021

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Some of the information presented in this guide was adapted from the following sources:

- Borden Institute. Medical Management of Chemical Casualties Handbook. 2014.
- Stanek SA, Saunders D. (eds) USAMRIID's Medical Management of Biologic Casualties Handbook, 9th edition, 2020.
- Goans RE. Armed Forces Radiobiology Research Institute. Medical Management of Radiological Casualties. 2013.
- CDC. Radiation Emergencies. Information for Clinicians.

General Principles

- Appropriate PPE must be utilized according to the role being performed:
    - Management of victims within the contaminated site of a chemical or biologic release requires Level A fully encapsulated protection with a supplied air source
    - Decontamination of victims within a warm zone requires Level B protection
    - Decontamination of chemical-exposed victims at a hospital generally requires Level C protection
- Clothing removal is estimated to remove between 75 to 90% of the contaminant.
    - Contaminated clothing and other personal belongings should be double bagged and labelled.
- Once decontamination has been accomplished, standard work uniform generally suffices.
- If a victim exposed to a biologic agent presents with illness, the exposure occurred days earlier and decontamination is not needed.
- Virtually all of the biologic agents present initially with a flu-like syndrome including fever, chills, malaise, headache and myalgias. Until a formal diagnosis is made, all patients should be managed as if they were infectious and capable of spreading the illness person-to-person.
- The biologic toxins are some of the most toxic agents known (even greater than the nerve agents)
- The biologic toxins are non-volatile so they do not pose a vapor threat or a threat of secondary or person-to-person transmission, and with the exception of the mycotoxins, are not dermally active.
- When managing victims of an, as yet, undiagnosed biologic illness and possible attack, look at the primary clinical syndrome:
    - Pulmonary syndrome, think:
        - Anthrax, pneumonic plague, tularemia
    - Neurologic syndrome, think:
        - Botulism, encephalitides
    - Gastrointestional syndrome, think:
        - Brucellosis, SEB, salmonella, shigella, E. coli, cholera, cryptosporidium
    - Dermatologic manifestations, think:
        - Smallpox, anthrax, tularemia, glanders, bubonic plague, VHF, T-2 mycotoxins
- Victims of radiation exposure with associated trauma should have life-threatening trauma prioritized before the radiation concern as the traumatic injury is an immediate life-threat.

CHEMICAL AGENTS

NERVE AGENTS

Agents: Tabun (GA), Sarin (GB), Soman (GD), GF, VX, Novichok

Mechanism of Action: Inhibit acetylcholinesterase (acetylcholine accumulates and target organs are overstimulated). Two types of cholinergic receptors: nicotinic and muscarinic.

Clinical Effects:

Muscarinic Effects

"DUMBBELS"
D – Diarrhea
U – Urination
M – Miosis
B – Bradycardia
B - Bronchorrhea, Bronchospasm
E – Emesis
L - Lacrimation
S - Salivation, Secretions, Sweating

"SLUDGE"
Salivation
Lacrimation
Urination
Defecation/Diarrhea
Gastrointestinal Distress
Emesis

Nicotinic Effects

"M,T,W,H,H,F"
Mydriasis
Tachycardia
Weakness
Hypertension
Hyperglycemia
Fasciculations

Time of symptom onset:

- Vapor exposure – seconds to a few minutes
- Liquid exposure – small amount – up to 18 hours
  - large amount – 1 to 30 minutes

Exposure Signs, Symptoms, and Treatment:

Decontamination:

Vapor Exposure – remove clothing to remove any trapped vapor
Liquid Exposure – remove clothing and perform decontamination

Mild Vapor Exposure

- Eyes: miosis, dim vision, headache
- Nose: rhinorrhea
- Mouth: salivation
- Lungs: dyspnea (tightness in the chest)

Treatment

- One Mark I Kit
  OR
- One Duodote
  OR
- Atropine 2 mg IV or IM
  and
  2-PAM (Pralidoxime) 1 gram IV

Severe Vapor Exposure

All of the above, plus

- Severe breathing difficulty or cessation of respiration
- Generalized muscular twitching, weakness, or paralysis
- Convulsions
- Loss of consciousness
- Loss of bladder and bowel control

Treatment

- Three Mark I Kits

OR

- Three Duodotes

OR

- Atropine 6 mg and IV or IM and 2-PAM (Pralidoxime) 1 gm IV and Diazepam 5 to 10 mg IV

Mild to Moderate Liquid Exposure

- Muscle twitching at site of exposure
- Sweating at site of exposure
- Nausea, vomiting
- Feeling of weakness

Treatment

- One to two Mark I Kits

OR

- One to two Duodotes

OR

- Atropine 2 to 4 mg IV or IM and 2-PAM (Pralidoxime) 1 gram IV

Severe Liquid Exposure

All of the above, plus

- Severe breathing difficulty or respiratory arrest
- Generalized muscle twitching, weakness, or paralysis
- Seizures
- Loss of consciousness
- Loss of bladder and bowel control

Treatment

- Three Mark I Kits
- Three Duodotes

OR

- Atropine 6 mg and IV or IM and 2-PAM (Pralidoxime) 1 gm IV and Diazepam 5 to 10 mg IV

Additional Treatment:

Airway management and ventilation may be necessary for high airway resistance

Atropine - How much to give?

- Until secretions are drying or dry
- Until ventilation is "easy"
- Until a conscious casualty is breathing comfortably

NOTE 1:

- Mark 1 Kit contains 2 individual autoinjectors
  - Autoinjector 1 – Atropine 2 mg
  - Autoinjector 2 – 2-PAM (Pralidoxime) 600 mg
- Duodote autoinjector contains both atropine and pralidoxime in one autoinjector

NOTE 2:

Atropine treats the muscarinic effects

- 2-PAM (Pralidoxime) treats the nicotinic effects (in the absence of aging)
    - Aging – after a period of time, the bond between the nerve agent and the muscarinic receptor site becomes permanent and 2-PAM will no longer be effective.

        Aging Time:

        Soman (GD): 2 minutes
        Sarin (GB): 4-5 hours
        VX: 60 hours

    Since the clinical presentation of all nerve agents will be similar and the specific agent will be unknown, 2-PAM (Pralidoxime) should be given to all victims.

NOTE 3:

When obtaining antidote from the federal or military supplies, Duodote autoinjectors may be labelled as "Antidote Treatment Nerve, Auto-Injector"

NOTE 4:

When obtaining antidote from the federal or military supplies, Diazepam autoinjectors may be labelled as "Convulsive Antidote, Nerve Agent" (CANA)

PULMONARY AGENTS

Agents: Ammonia, Chlorine, Phosgene

Mechanism of Action: The pulmonary agents react with water to form either acid (hydrochloric acid in the case of exposure to chlorine and phosgene) or alkali (ammonium hydroxide in the case of exposure to ammonia). In addition, phosgene may act directly to damage proteins, enzymes, and cell membranes in the alveoli of the lungs.

Clinical Effects: The clinical effects of the pulmonary agents depend upon the degree of water solubility of the chemicals. Ammonia is highly water soluble and acts on the upper respiratory tract. Chlorine is moderately water soluble and acts on both the upper and lower respiratory tract. Phosgene is poorly water soluble and is inhaled into the lower respiratory tract and alveoli.

Upper Respiratory Tract Agents: Symptoms and signs include nasopharyngeal irritation, hoarseness, painful swallowing, laryngeal inflammation, cough, stridor, laryngospasm, wheezing, and chest pain.

Lower Respiratory Tract Agents: Symptoms and signs may include irritation of the eyes and mucous membranes of the nose and throat, although the complaints are usually not as severe as with the upper respiratory tract agents and often not reported at all. Gradually worsening shortness of breath will develop, coughing, and pulmonary edema with potentially large amounts of clear, frothy sputum.

Time of Symptom Onset:

Upper Respiratory Tract Agents: Symptoms and signs typically begin immediately or shortly after exposure to these agents because of the degree of water solubility.

Lower Respiratory Tract Agents: Typically, there is a delay in onset of symptoms. The latent period varies between 1 and 72 hours although most victims with significant exposure will develop symptoms within 24 hours.

Treatment:

- Remove from the contaminated environment.
- Decontamination for any liquid exposure and clothing removal to minimize trapped vapor exposure.
- Assure an adequate airway particularly in patients with hoarseness or stridor.
- Prevent and treat hypoxia with supplemental oxygen administration. Continuous positive airway pressure, intubation, and ventilation may be required to maintain adequate oxygenation and treat pulmonary edema.
- Prevent and treat hypotension by appropriate fluid administration. Hypotension may result from the large amount of fluid lost into the alveoli from the pulmonary edema.

- Manage secretions by suctioning and positioning for drainage.
- Manage bronchospasm with bronchodilators. Parenteral (intravenous) steroids may also be used for bronchospasm in patients with reactive airway disease.
- Enforce rest, stretcher evacuation, and no exertion. Any physical activity or exertion may enhance the effect of lower respiratory tract agents, shorten the latent period, and increase the severity of the damage.

VESICANT (BLISTER) AGENTS

Agents: Sulfur mustard, Lewisite

Mechanism of Action: Mustard and Lewisite are both vapor and liquid hazards. Mustard penetrates rapidly through intact skin, mucous membranes, and through inhalation. Penetration is enhanced by moisture and warmth. Mustard binds within minutes to cellular proteins, enzymes, and damages DNA leading to inflammation and cellular death. It also has mild cholinergic effects. Lewisite is also rapidly absorbed through skin, mucous membranes and the respiratory tract. The exact mechanism by which it causes damage is not known.

Clinical Effects: The clinical effects of both agents vary from mild irritation to severe damage and necrosis.

Eyes: Irritation of the conjunctiva, blepharospasm, corneal damage and opacification occur.

Skin: The skin damage resembles that of a thermal burn with erythema, blister formation, and potentially full-thickness necrosis. It is important to note that the blister fluid of mustard exposed victims does not contain any active mustard and poses no threat to care-givers. The initial skin damage from Lewisite is a grey appearing epithelial injury that then progresses to resemble the effect of mustard.

Pulmonary: Irritation of the mucous membranes of the nose and pharynx. Cough, laryngitis, and laryngospasm occur as the concentration of agent increases. Necrosis of the mucous membranes of the airway may result leading to pseudomembrane formation. Shortness of breath may result from lower airway damage. Lewisite exposure may lead to pulmonary edema.

Gastrointestinal: Nausea, vomiting and diarrhea may result from the cholinergic effects of mustard. GI effects such as vomiting and diarrhea are more common with Lewisite.

Hematologic: Mustard in large amounts is toxic to stem cells in bone marrow leading to marrow suppression and pancytopenia. Lewisite, in contrast, does not affect the bone marrow.

Time of Symptom Onset:

Sulfur Mustard: Mustard has a latent period during which the exposed individual is asymptomatic and, as a result, may not be aware of the exposure. This latent period varies between 1 to 24 hours.

Lewisite: Lewisite, unlike mustard, causes immediate discomfort, irritation and pain upon exposure.

Treatment:

- Remove from the contaminated environment.
- Decontaminate with copious amounts of soap and water for any liquid exposure and clothing removal to minimize trapped vapor exposure. Reactive Skin Decontamination Lotion (RSDL) may be used for small areas of localized exposure.
    - NOTE: Decontamination will not change the clinical course of patients exposed to mustard since the damage occurs within minutes and the victim is often unaware of the exposure, but it will minimize the risk of secondary contamination of caregivers.
- Assure an adequate airway particularly in patients with hoarseness or stridor.
- Prevent and treat hypoxia with supplemental oxygen administration. Continuous positive airway pressure, intubation, and ventilation may be required to maintain adequate oxygenation and treat pulmonary edema.
- Bronchodilators may be administered for wheezing.
- Antibiotics should not be given prophylactically but only for documented infection.
- Anticholinergic eye drops (i.e., homatropine) may be administered to help prevent synechiae formation along with topical antibiotics.
- Vaseline may be applied to eyelid edges to prevent them from adhering together.
- Prevent and treat hypotension by appropriate fluid administration. Maintain fluid and electrolyte balance by administration of IV fluids as appropriate.
- Management of skin injury is similar to that of a thermal burn.
- Granulocyte colony stimulating factor has been shown to be of benefit in animal studies for bone marrow suppression and may be indicated in these cases.
- Lewisite, unlike mustard, has a specific antidote available – British AntiLewisite (BAL) or dimercaprol which may be given via intramuscular injection.

ASPHYXIANT AGENTS

Agents: Cyanide, Cyanogen Chloride, Hydrogen Sulfide

Mechanism of Action: Cyanide is primarily a vapor hazard as the liquid rapidly volatilizes. Cyanide binds to the cytochrome a3 enzyme in cell mitochondria and blocks intracellular oxygen utilization therefore inhibiting aerobic metabolism. As a result, anaerobic metabolism takes over leading to lactic acidosis. Hydrogen sulfide also inhibits cytochrome a3, leading to anaerobic metabolism.

Clinical Effects:

Exposure	Signs and Symptoms
Moderate (low concentration)	Dizziness, nausea, vomiting, headache, transient increase in rate and depth of breathing. Symptoms may progress if exposure continues.
Severe (high concentration)	Transient increase in rate and depth of breathing, seizures, respiratory arrest, cardiac arrest

Time of Symptom Onset:

Inhaled:

Low concentration: within minutes

High concentration: within seconds, death within 6 to 8 minutes

Ingested: approximately 10 minutes, death within approximately 30 minutes

Treatment:

- Utilize appropriate P.P.E.
- Remove from the contaminated environment.
- Decontaminate with soap and water for any liquid exposure and clothing removal to minimize trapped vapor exposure.
- If cyanide was ingested, DO NOT induce emesis. Administer activated charcoal. If the patient vomits, collect and contain all vomitus to prevent secondary exposure from cyanide off-gassing.
- Assure adequate airway and ventilation.
- Sodium bicarbonate may be administered for victims with cardiac dysrhythmias or profound acidosis.
- Benzodiazepines may be administered for seizures.
- Administer antidote:
    - Hydroxocobalamin is the preferred antidote for cyanide. It binds with the cyanide ion to form cyanocobalamin (vitamin B12). The adult dose is 5 grams given intravenously over 15 minutes.

- If hydroxocobalamin is not available, a cyanide antidote kit may be used as an alternative.
  - Step 1:
    - If IV access is available, administer sodium nitrite 10 ml of 3% solution over 5 to 10 minutes.
    - If IV access is not available, administer amyl nitrite by inhalation.
      - One amyl nitrite perle should be broken onto a gauze pad and held under the nose, placed under the lip of a facemask, or over the bag-mask intake and the patient should inhale for 30 seconds of each minute. A new perle should be utilized every three minutes if intravenous sodium nitrite infusions will be delayed.
      - Nitrite displaces cyanide from the cytochrome system by creating methemoglobin which preferentially binds the cyanide.
      - Hypotension may develop if nitrite is administered too quickly.
  - Step 2:
    - After nitrite administration, administer sodium thiosulfate 50 mL of a 25% solution over 10 to 20 minutes, which converts the cyanide to thiocyanate that is then excreted in the urine.
- For hydrogen sulfide exposure, mild to moderate toxicity is treated primarily with supportive care. For victims with severe toxicity, aggressive airway management including intubation, oxygen supplementation and crystalloid/pressor support is appropriate. In addition, sodium nitrite may be administered as for cyanide toxicity based upon animal data. Thiosulfate and hydroxocobalamin are not useful.

NOTE: Cyanide has often been described as having the odor of bitter almonds and hydrogen sulfide smells like rotten eggs.

- The sense of smell should never be considered a reliable indicator of exposure.
- Fifty percent of the population is genetically unable to detect cyanide.
- Both cyanide and hydrogen sulfide induce olfactory fatigue and after a period of time the odor can no longer be detected.
- If the odor of these two agents is detected, exposure has occurred and immediate evacuation from the location is indicated.

BIOLOGIC AGENTS

Bacteria

ANTHRAX

Agent: Bacillus anthracis (gram + spore forming bacteria)

Mode of Exposure:
- Inhalation (most likely in intentional release) – inhalational anthrax
- Ingestion – gastrointestinal anthrax
- Inoculation – cutaneous anthrax

Pathogenesis: Upon inhalation of the spores, the spores are attacked by macrophages. The macrophages, with the spores, then travel to the regional lymph nodes in the mediastinum. The spores germinate and produce vegetative bacteria which produce 3 toxins:
- Edema Factor – which causes tissue swelling
- Lethal Factor – which causes cell necrosis
- Protective Factor – allows Edema Factor and Lethal Factor to attack the cell

Incubation Period:
- Inhalational anthrax: Usually 1 to 6 days, rarely may be longer
- Gastrointestinal anthrax: Usually 1 to 6 days
- Cutaneous anthrax: Usually 5 to 7 days (maybe as short as 1 day or as long as 12 days)

Clinical Presentation:
- Inhalational Anthrax: Initially patient presents with fever, myalgias, cough, mild chest discomfort, and fatigue (flu-like syndrome) typically lasting 2 to 5 days. Physical examination is non-specific. In some cases the patient may then have a short period of apparent improvement. Patients then develop severe respiratory distress, sepsis, shock and death ensues in 24 to 36 hours. Inhalational anthrax has, in the past, been associated with hemorrhagic meningitis in up to 50% of patients and gastrointestinal bleeding in as many as 80% of patients. Traditional teaching is that inhalational anthrax causes an acute mediastinitis without pneumonia, however pulmonary infiltrates were identified in many of the victims of the 2001 letter attack.
- Gastrointestinal Anthrax: Infection may result in the oropharynx or the intestinal tract and is thought to result from ingestion of vegetative bacteria and not the spores.
    - Oropharyngeal anthrax presents with fever and pharyngitis. Ulcers which then develop pseudomembranes develop, typically of the tonsils.

Dysphagia and regional lymphadenopathy are usually present. The resulting edema can lead to airway compromise. The infection may progress to sepsis.
    - Intestinal anthrax presents with fever, nausea, vomiting, and abdominal pain. These complaints can progress to GI hemorrhage, ascites, and sepsis.
- Cutaneous Anthrax: This form of anthrax is the most common, naturally occurring anthrax and is found in individuals working with infected livestock. The disease begins as a painless or pruritic papule at the exposure site. Within 24 hours it enlarges and ulcerates. It is usually associated with surrounding edema. As the ulcer dries, it forms a black scab resembling a piece of coal which separates over the next several weeks. Regional lymphadenopathy may be found, and systemic infection occurs in 10 to 20% of patients.

Mortality:
- Inhalational anthrax: Historic case fatality rate (CFR) is greater than 85%. However, the CFR after the letter attack in 2001 was 45%, likely due to improvements in treatment regimens and ICU management.
- GI anthrax: Historic case fatality rate is greater than 50%.
- Cutaneous anthrax: Untreated CFR is 10 to 20%; treated CFR is less than 1%.

Transmissibility: Anthrax is not spread person-to-person.

Infection Control: Standard precautions

Diagnosis: Blood gram stain and culture (note that once antibiotics are initiated, culture will be negative). Gram stain of CSF may reveal hemorrhage and organisms. Chest x-ray and CT scan should be performed to look for widened mediastinum, enlarged mediastinal lymph nodes, and pleural effusions.

Management:
- Current recommendation is the administration of an intravenous fluoroquinolone with one or two additional antibiotics depending on the presence or absence of associated hemorrhage meningitis.
    - The latest CDC recommendations should be reviewed as antibiotic regimens are subject to change.
- ABThrax (raxibacumab) and Anthim (obiltoxaximab) are FDA approved monoclonal antibodies against Protective Factor in combination with antibiotic therapy.
- Supportive care to assure adequate airway and ventilation and maintain circulatory status.

Post-exposure Prophylaxis:

- Anthrax vaccine (Biothrax®) should be administered at 0, 2 and 4 weeks post-exposure in combination with an oral fluoroquinolone or doxycycline or clindamycin.
- ABThrax (raxibacumab) and Anthim (obiltoxaximab) may be utilized when other treatments are contraindicated.

PLAGUE

Agent: Yersinia pestis (gram negative, non-spore forming, non-motile bacillus)

Mode of exposure:
- Inhalation (most likely in intentional release) – pneumonic plague
- Inoculation – bubonic plague (most common naturally occurring form)

Pathogenesis: Upon either inhalation or inoculation as a result of an infected flea bite, the bacteria are ingested by macrophages which travel to the regional lymph nodes producing a lymphadenitis. Bubonic plague results from the infected flea bite and the resulting lymphadenopathy (buboes). Primary pneumonic plague results from the inhalation of the bacteria. Secondary pneumonic plague can result from bacteremic spread due to the bubonic form. Septicemic plague can occur with either bubonic or pneumonic plague or as the primary manifestation of infection.

Incubation period:
- Pneumonic plague - 1 to 6 days (in most cases 2 to 4 days)
- Bubonic plague – 2 to 8 days

Clinical presentation:
Both forms of plague begin abruptly with a non-specific flu-like syndrome including high fever, chills, malaise, headache, and myalgias. Nausea, vomiting, and abdominal pain may occur.
- Pneumonic plague: Within 24 hours of the onset of symptoms, tachypnea, dyspnea, and cough with hemoptysis develop. The respiratory distress rapidly progress to respiratory and circulatory failure.
- Bubonic plague: At the same time or shortly after the non-specific symptoms occur, an enlarged, painful, infected node (bubo) develops. Bacteremia is common.
- Septicemic plague: In those patients that progress to septicemia, thrombi may occur in distal acral vessels leading to necrosis of appendages such as the fingers, toes, tip of the nose, ears, and the penis in males.

Mortality:
- Pneumonic plague: 100% if unrecognized and untreated or delayed for more than 24 hours after symptom onset, approximately 50% if treated promptly
- Bubonic plague: 60% if untreated, <5% if treated promptly
- Septicemic plague: 100% if untreated, 30 to 50% treated.

Transmissibility:
Pneumonic plague may spread person-to-person.

Infection control:

- Pneumonic plague: Strict isolation with droplet precautions for minimum of 48 hours after initiation of antibiotic treatment or until sputum cultures are negative in confirmed cases
- Bubonic plague: Standard precautions
- Septicemic plague: Standard precautions

Diagnosis:

- Bubonic plague clinical diagnosis: Clinical suspicion in a patient with fever, malaise, painful swollen lymph node after recent travel to endemic area or possible rodent exposure and flea bite.
- Pneumonic plague clinical diagnosis: Presentation of large numbers of patients with rapidly progressive, severe pneumonia with hemoptysis (suggesting an intentional release of plague)
- Laboratory diagnosis: Finding of coccobacillus using Wright, Giemsa, or methylene blue stain or specific immunofluorescence antibody stain smear from bubo needle aspirate, sputum, blood or CSF samples. Blood cultures should be obtained, ideally prior to antibiotic administration, and commonly take 48 to 72 hours. NOTE: Treatment should not be withheld pending culture results.

Management:

- Immediate initiation of empiric antibiotic therapy
    - Streptomycin (preferred), gentamicin acceptable alternative
    - Alternatives
        - Doxycycline
        - Ciprofloxacin
        - Chloramphenicol
        - Levofloxacin
    - The latest CDC recommendations should be reviewed as antibiotic regimens are subject to change.
- Supportive care to assure adequate airway and ventilation and maintain circulatory status.

Post-exposure prophylaxis

- Antibiotics:
    - Doxycycline
    - Alternatives:
        - Ciprofloxacin
        - Chloramphenicol
- Vaccine – no vaccine or immune globulin is available.

BRUCELLOSIS

Agent: Brucella species (B. abortus, B. canis, B. melitensis, B. suis)

Mode of exposure:
- Inhalation of contaminated particles (most likely in intentional release)
- Contact with infected animal tissues, blood, urine, semen
- Ingestion of raw milk and other dairy products from infected animals
- Inoculation through open skin

Pathogenesis:
- Inhalation of as few as 10 Brucella organisms is enough to cause disease
- Brucella organisms invade the reticuloendothelial system, especially in the liver, spleen and bone marrow. Over time granulomas form from which persistent bacteremia may result.

Incubation period: Few days to several months, usually 3 to 4 weeks.

Clinical presentation:
- Fever (typically intermittent), malaise, sweats, myalgias/arthalgias
- Fatigue, chills, back pain
- Headache, irritability and depression
- Anorexia, vomiting, abdominal pain, diarrhea, constipation
- Examination often reveals hepatomegaly, splenomegaly, arthritis (usually monoarticular but may be polyarticular), adenopathy, weight loss, bursitis, tenosynovitis, osteomyelitis, sacroiliitis, discitis

Mortality: Rare

Transmissibility:
- Generally not transmissible person-to-person
- Bacteria may be passed through breast milk
- May be spread through sexual contact.

Infection control: Standard precautions

Diagnosis: Definitive diagnosis includes:
- Isolation of Brucella sp. from a clinical specimen;
- > a fourfold rise in Brucella sp. agglutination titer between acute and convalescent sera obtained > 2 weeks apart and performed at the same lab;
- demonstration by immunofluorescence of Brucella sp. in a clinical specimen.

Management:

- Antibiotic therapy
    - Doxycycline plus streptomycin or gentamicin
    - Doxycycline plus rifampin
- Antibiotic therapy (cont'd)
    - Fluoroquinolone plus rifampin
    - TMP-SMX plus rifampin
    - Triple antibiotic therapy is often needed for complicated brucellosis (skeletal disease, endocarditis)
    - The latest CDC recommendations should be reviewed as antibiotic regimens are subject to change.
    - NOTE: Relapse rates of 5 to 10% have been reported.

Post-exposure prophylaxis

- Antibiotic therapy
    - Doxycycline plus rifampin
- Vaccine – no vaccine or immune globulin is available

Q FEVER

Agent: Coxiella burnetii (obligate intracellular gram negative bacterium)

Mode of exposure:
- Inhalation of infectious particles (most likely in intentional release)
- Ingestion
- Sexual contact (extremely rare)

Pathogenesis: The bacteria multiply in the lungs and then invade the blood stream to infect other organs.

Incubation period: 2 to 3 weeks

Clinical presentation:
- Historically, as many as 60% of infections are asymptomatic (may not be the case in an intentional release)
- Flu-like syndrome – Fever (can last two weeks in untreated patients), headache (retro-orbital) malaise, myalgias/arthalgias
- Pneumonia
- Hepatitis
- Rare – pericarditis, myocarditis, endocarditis, meningoencephalitis, peripheral myelitis, rash

Mortality: Rare

Transmissibility:
- Generally not transmissible person-to-person, rare cases via blood transfusion or aerosol generating procedures

Infection control: Standard precautions

Diagnosis:
- Case pattern – geographic area or short time period
- Indirect immunofluorescence
- Polymerase Chain Reaction (PCR) detection (serum, blood, tissue biopsy)

Management:
- Antibiotic therapy
    - Doxycycline, Alternative: Moxifloxacin, ciprofloxacin
    - Pregnant women: trimethoprim/sulfamethoxazole
    - The latest CDC recommendations should be reviewed as antibiotic regimens are subject to change.

Post-exposure prophylaxis

- Antibiotic therapy - Doxycycline
- Vaccine – no vaccine or immune globulin is available

TULAREMIA

Agent: Francisella tularensis (aerobic non-motile gram negative coccobacillus)

Mode of exposure:
- Inhalation (most likely in intentional release)
- Ingestion
- Inoculation through skin

Incubation period: Typically 3 to 6 days (range 1 to 21 days)

Clinical presentation:
- Typhoidal Tularamia:
    - Flu-like syndrome – Fever, headache, malaise, myalgias/arthralgia's, cough
    - Nausea, vomiting, diarrhea, abdominal pain
    - Pneumonia (most likely after an intentional release)
    - Severe disease may include meningitis, pericarditis, endocarditis, sepsis, renal or hepatic injury.
- Ulceroglandular Tularemia: Most often after skin or mucous membrane inoculation
    - Systemic symptoms as described above
    - Painful papule at the site of inoculation which progresses to pustule and then ulcer
        - Associated with lymphadenopathy which can become fluctuant and drain

Mortality: 30 to 60% if untreated, 1 to3% if treated

Transmissibility: No documented cases of person-to-person transmission

Infection control: Standard precautions

Diagnosis:
- Case pattern – geographic area or short time period
- Direct immunofluorescence assay
- Polymerase Chain Reaction (PCR) detection

Management:
- Antibiotic therapy
    - Streptomycin or gentamicin
    - Alternatives: Doxycycline, Ciprofloxacin, Chloramphenicol
    - The latest CDC recommendations should be reviewed as antibiotic regimens are subject to change.

Post-exposure prophylaxis

- Antibiotic therapy – Doxycycline, Ciprofloxacin
- Vaccine – no vaccine or immune globulin is available in the U.S.

Virus

SMALLPOX

Agent: Orthopoxvirus Variola (major, minor) – DNA virus

Mode of exposure:
- Inhalation

Pathogenesis: After inhalation, a viremia occurs. The virus then disseminates to lymphoid tissues, liver, spleen, bone marrow, and lung and a secondary viremia results.

Incubation period: 12 days average (range 7 to 19 days)

Clinical presentation:
- Initially a flu-like syndrome – Fever (as high as 104°F), rigors, headache, malaise, nausea, vomiting, prostration. Approximately 15% of patients become delirious.
- Within 48 to 72 hours of the onset, the characteristic rash develops, first on the face, hands and forearm and then it spreads to the lower extremities and then centrally over the trunk. The rash begins as small, erythematous macules which become papular and then vesicular. The lesions are predominantly located on the face and extremities and are synchronous in their progression.
- After 8 to 14 days, the lesions scab and after separating leave depressed, depigmented scars in the skin.

Mortality: Historically 30% if unvaccinated, 3% if vaccinated

Transmissibility: Person-to-person spread is likely.
- All patients should be considered infectious from the time the rash appears until all scabs have separated and should remain isolated.
- Contacts of smallpox patients should be isolated for a minimum of 17 days.

Infection control: Contact and airborne precautions and patient isolation.

Diagnosis:
- Clinical recognition of characteristic rash

Management:
- Isolation and contact and airborne precautions
- Antiviral treatment
    - Tecovirimat (TPOXX)
    - Brincidofovir (Tembexa)
    - Cidofovir

- Supportive care – maintenance of fluid status, treatment of secondary infection

Post-exposure prophylaxis

- Vaccine – Two vaccines as well as immune globulin are available in the U.S.
    - Vaccine – given as soon as possible after exposure
        - ACAM2000 – Live virus (replicating)
        - JYNNEOS – Live virus (non-replicating)
- Vaccinia Immune Globulin – indicated for passive immunoprophylaxis for the following vaccine complications:
    - Eczema vaccinatum
    - Progressive vaccinia
    - Severe generalized vaccinia
    - Vaccinia infections in individuals who have skin conditions such as burns, impetigo, varicella-zoster, or poison ivy; or in individuals who have eczematous skin lesions

VIRAL HEMORRHAGIC FEVERS (VHF)

Agents: Arenaviridae, Bunyaviridae, Filoviridae, and Flaviviridae.

- Arenaviridae
  - Lassa Fever
  - Argentine hemorrhagic fever
  - Bolivian hemorrhagic fever
  - Brazilian hemorrhagic fever
  - Venezuelan hemorrhagic fever
- Bunyaviridae
  - Hantavirus genus
    - Hemorrhagic fever with renal syndrome (HFRS)
  - Nairovirus genus
    - Crimean-Congo hemorrhagic fever (CCHF)
  - Phlebovirus genus
    - Rift Valley fever
- Filoviridae
  - Ebola
  - Marburg
- Flaviviridae
  - Dengue
  - Yellow Fever
  - Omsk hemorrhagic fever
  - Kyasanur Forest disease

Mode of exposure: Varies depending upon specific agent, including exposure to blood and body fluids, inhalation, and transmission from animal to human via a vector, inhalation, or ingestion of rodent secretions or excretions.

Pathogenesis: The exact pathogenesis for many of the VHF disease is still being determined but the target organ is the vascular bed and system. The etiology of the coagulopathy is thought to be multifactorial including a consumptive coagulopathy and primary bone marrow dysfunction.

Incubation period: Varies depending upon the specific agent, generally 7 to 14 days but may be as short as 3 days for Rift Valley fever, Congo-Crimean HF, Marburg, Ebola, Yellow fever, Dengue, Kyasanur Forest disease and Omsk HF. Hantavirus may take as long as 35 days to manifest.

Clinical presentation:

- Initially a flu-like syndrome – Fever, headache, malaise, myalgias.

- Depending upon the specific agent, the patient may have very mild symptoms (most cases of naturally occurring Lassa) or develop severe VHF syndrome findings including capillary lead, bleeding diathesis, and shock leading to end organ failure.

Mortality:
- Arenaviridae
    - Lassa Fever – 1 to 2%
    - Argentine hemorrhagic fever – 30%
    - Bolivian hemorrhagic fever – 25 to 35%
    - Brazilian hemorrhagic fever – 10 to 30%
    - Venezuelan hemorrhagic fever – 10 to 30%
- Bunyaviridae
    - Hantavirus genus
        - Hemorrhagic fever with renal syndrome (HFRS) – 5%
    - Nairovirus genus
        - Crimean-Congo hemorrhagic fever – 30%
    - Phlebovirus genus
        - Rift Valley fever - <1%
- Filoviridae
    - Ebola – 40 to 90%
    - Marburg – 23 to 70%
- Flaviviridae
    - Dengue
    - Yellow Fever – 3 to12%, up to 50% if the hemorrhagic form develops
    - Omsk hemorrhagic fever – 0.2 to 3%
    - Kyasanur Forest disease – 3 to 10%

Transmissibility: The VHFs are generally highly transmissible.

Infection control: Standard, contact, and droplet precautions and strict patient isolation.

Diagnosis:
- A VHF should be suspected with an appropriate travel history, a severe febrile illness, and signs of vascular involvement.
- Definitive diagnosis depends upon determination of the specific virus including ELISA analysis. Viral culture requires a minimum of 3 days and in many cases much longer.

Management:
- Isolation and contact and airborne precautions
- Supportive care – maintenance of fluid status, treatment of secondary infection
- Severe hemorrhage requires appropriate replacement therapy

- Inmazeb (combination of 3 monoclonal antibodies) and Ebanga (single monoclonal antibody) have been approved for the treatment of Ebola.
- ZMAPP (combination of 3 monoclonal antibodies) has shown promise for the treatment of Ebola but is considered experimental at the time of this writing.
- Ribavirin may be used under an IND protocol or EUA for the treatment of CCHF, Lassa, and HFRS.

Pre-Exposure Prophylaxis

- Yellow fever vaccine – live attenuated vaccine for prevention of yellow fever in travelers
- Ervebo - rVSVΔG-ZEBOV-GP Ebola vaccine is licensed for adults responding to an Ebola outbreak to prevent infection.

Post-Exposure Prophylaxis

- Individuals with percutaneous or mucocutaneous exposure to blood, body fluids, secretions, or excretions from a patient with suspected VHF should immediately wash the exposed skin surfaces with soap and water and irrigate mucous membranes with copious amounts of water or saline solution.

BOTULINUM TOXIN

Agent: Botulinum neurotoxin Types A through G

Bacterial Source: Clostridium botulinum (Types A through G), Clostridium butyricum (Type E), Clostridium baratii (Type F), Clostridium argentinense (Type G)

Mode of exposure:
- Inhalation
- Ingestion

Pathogenesis:
- Botulinum toxin acts to inhibit acetylcholine release at the presynaptic terminal, thus blocking neuromuscular impulse transmission at both muscarinic and nicotinic receptors.
- Recovery requires regeneration of presynaptic nerve endings which can take 30 to 120 days or longer.

Onset of Symptoms: 12 to 36 hours average, may take several days if low dose exposure

Clinical presentation:
- Progressive, descending paralysis in an awake, alert, afebrile patient.
    - Cranial nerves initially affected. Findings include mydriasis, ptosis, diplopia, dysarthria, dysphonia, and dysphagia.
    - Flaccid skeletal muscle paralysis ensues in a descending fashion.
        - Oropharyngeal muscle weakness can lead to airway difficulties
        - Weakness and paralysis of the muscles of respiration can lead to respiratory failure
- Anticholinergic signs and symptoms include decreased secretions and dry mouth, constipation, urinary retention, and ileus.
- Deep tendon reflexes may be decreased or absent.
- Typically, there are no abnormal sensory findings.

Mortality: Approximately 3 to 5% (naturally occurring botulism)

Transmissibility: Botulism does not spread person-to-person.

Infection control: Standard precautions

Diagnosis:

- Botulism is a clinical diagnosis.
- Mouse neutralization is the definitive laboratory test but takes several days and a lab capable of performing the study.

Management:

- Supportive care
- Respiratory failure must be managed with airway control and ventilatory assistance.
- Botulinum antitoxin – Heptavalent Botulinum Antitoxin, a despeciated equine antitoxin, is available from the CDC.
    - Treatment with the antitoxin will help prevent progression of the disease but will not reverse existing findings or remove toxin already bound to nerve terminals.
    - Treatment should be initiated without delay based upon the clinical presentation consistent with botulism.

Post-exposure prophylaxis

- The Heptavalent Botulinum Antitoxin may be considered in extremely rare cases involving high risk exposure to botulinum toxin (e.g. laboratory mishap).

RICIN

Agent: Ricin

Source: Ricinus communis (Castor bean plant)

Mode of exposure:
- Inhalation
- Ingestion
- Inoculation (Injection)

Pathogenesis: Cellular protein cytotoxin, inhibition of protein synthesis leading to cell death and tissue necrosis

Onset of Symptoms:
- Inhalation: 4 to 8 hours
- Ingestion: 4 to 6 hours
- Injection: Generally within 12 hours

Clinical presentation:
- Inhalation: Fever, chest discomfort, cough, progressively worsening dyspnea, alveolar necrosis, acute respiratory distress syndrome with respiratory failure, death.
- Ingestion: Nausea, vomiting, diarrhea, necrosis of gastrointestinal epithelium, hemorrhage, and liver, kidney and spleen necrosis
- Inoculation (injection): Muscle necrosis, necrosis of regional lymph nodes, liver and kidney damage, may progress to multisystem organ failure

Mortality:
- Inhalation – unknown
- Ingestion – 2 to 6%
- Injection - unknown

Transmissibility: Ricin does not spread person-to-person.

Exposure control:
- Level B protection for active release or entry into contaminated area
- Standard universal precautions for hospital treatment of patients

Diagnosis:
- Ricin poisoning is a clinical diagnosis based upon any known threat or in the context of multiple casualties presenting with similar clinical findings at the same time from the same general geographic area

- There are no validated assays for detection of ricin in healthcare clinical laboratories
- ELISA and immunohistochemical tests may be available through the CDC

Management:

- Supportive care
- Respiratory failure must be managed with airway control and ventilatory assistance.
- Gastric lavage and charcoal administration may be performed for ingestion less than 1 hour

Post-exposure prophylaxis

- No antidote or prophylaxis medications are available.
- A vaccine is in development (Rivax) but has not yet been approved for use.

STAPHYLOCOCCAL ENTEROTOXIN B (SEB)

Agent: Staphylococcal Enterotoxin B

Source: Staphylococcus aureus

Mode of exposure:
- Inhalation
- Ingestion

Pathogenesis: Release of cytokines with an intense inflammatory response that injures target tissues

Onset of Symptoms:
- Inhalation: typically 3 to 12 hours
- Ingestion: typically 1 to 6 hours

Clinical presentation:
- Inhalation: Fever, chills, chest discomfort, non-productive cough, headache, malaise, myalgias, shortness of breath. Severe intoxication can lead to pulmonary edema, acute respiratory distress syndrome, toxic shock and death.
- Ingestion: Nausea, vomiting, diarrhea, abdominal cramping and pain

Mortality: Rare

Transmissibility: Does not spread person-to-person.

Exposure control: Standard precautions for hospital treatment of patients

Diagnosis:
- SEB poisoning is a clinical diagnosis based upon any known threat or in the context of multiple casualties presenting with similar clinical findings at the same time from the same general geographic area
- There are no validated assays for detection of SEB in healthcare clinical laboratories
- ELISA and PCR tests may be available through the CDC or reference labs.

Management:
- Supportive care
- Respiratory compromise must be managed with airway control as necessary and ventilatory assistance.
- Most patients with ingested SEB improve in 1 to 2 days; most patients with inhalational SEB improve within 5 to 7 days.

Post-exposure prophylaxis

- There is no approved vaccine although a vaccine candidate is in development.

T-2 MYCOTOXINS

Agent: Trichothecene mycotoxins (Yellow rain)

Source: Fungi (mold) metabolites

Mode of exposure:
- Inhalation
- Ingestion
- Transdermal

Pathogenesis: Inhibition of cellular protein synthesis, particularly on actively proliferating cells (skin, bone marrow, and gastrointestinal tract), inhibition of mitochondrial respiration.

Onset of Symptoms: Within minutes of exposure

Clinical presentation:
- Symptoms of exposure involve all three routes concurrently.
- Inhalation: Nasal pruritus, sneezing, epistaxis, rhinorrhea, nasal discomfort, cough, bloody sputum, dyspnea, wheezing.
- Ingestion: Nausea, vomiting, anorexia, bloody diarrhea, abdominal cramping and pain
- Transdermal: Burning skin pain, erythema, tenderness, blistering, skin necrosis
- Systemic toxicity includes weakness, dizziness, prostration, ataxia, tachycardia, hypotension and death. Late sign of systemic toxicity is bone marrow suppression and pancytopenia.

Mortality: unknown. Case fatality rates from several historical accidental ingestion incidents ranged from 10 to 60%.

Transmissibility: Contact precautions until decontamination is accomplished, then standard precautions.

Exposure control: Standard precautions for hospital treatment of patients

Diagnosis:
- T-2 mycotoxin poisoning is a clinical diagnosis based upon any known threat, presence of yellow or other pigmented oily residue, or in the context of multiple casualties presenting with similar clinical findings at the same time from the same general geographic area
- There are no rapid assays for detection of T-2 mycotoxin in healthcare clinical laboratories

- ELISA and chromatography tests may be available through reference labs.

Management:

- Decontamination as soon as possible after exposure to include clothing removal and washing with soap and water.
    - Decontamination reduces skin damage even if delayed for 4 to 6 hours post exposure.
- Reactive Skin Decontamination Lotion (RSDL) may be used to decontaminate small, localized areas of exposure.
- Supportive care as for a thermal burn
- Activated charcoal has been recommended to bind residual toxin in the oropharynx and GI tract.
- Respiratory and ventilatory support as necessary
- Serial lymphocyte counts may help identify bone marrow suppression.
    - The role of colony stimulating factors is unknown.

Post-exposure prophylaxis: No specific therapy or interventions are available.

RADIATION / NUCLEAR EXPOSURE

Types of Radiation

- Radiation illness involves exposure to energized particles or rays.
  - Alpha Particles – ionized particles that are limited in their travel distance. Can be shielded by dead skin, a piece of paper or clothing. Alpha particles are a risk if internalized, most commonly either through inhalation or open wounds.
  - Beta Particles – energized electrons that can penetrate a short distance into tissues. Beta particles are a risk if large amounts are deposited externally resulting in skin damage or internalized. Protection is afforded by a thin layer of plastic.
  - Gamma Rays – highly energized photons that pass through organic matter easily. Gamma radiation can result in partial or whole body exposure with resulting multi-organ damage. Gamma-emitting radionuclides are a risk when internalized. Protection typically involves dense materials such as lead.
  - Neutrons – uncharged particles that are highly energized and very penetrating. Typically, neutron radiation results from nuclear fission or fusion reactions. Neutrons have as much as 20 times greater risk of future effects compared to gamma rays and can make something radioactive.

Mode of Exposure:

- Simple Radiologic Device (Radiation Exposure Device) – Radioactive material placed in a location where nearby individuals can be exposed to the radiation. Casualties would result from radiation exposure.
- Radiologic Dispersal Device – Radioactive material attached to a conventional explosive such that radioactive material is dispersed over a large area. Casualties would result from trauma because of the explosion as well as exposure to radioactive material either through surface contamination, inhalation of radioactive particles, or ingestion.
- Improvised Nuclear Device – Device designed to produce a partial or full yield nuclear detonation with resulting traumatic injuries, burns, and radiation exposure.

Pathogenesis: Damage to cells from radiation and radioactive materials happens within microseconds of exposure. The exposure to radiation and high energy particles damages cell membranes, cellular proteins, and DNA, particularly in cells with high replication rates. This leads to organ damage and over longer periods of time the potential for carcinoma development.

Onset of Symptoms: Within minutes to hours of exposure depending on the dose of radiation received.

- The amount of energy deposited in tissue by ionizing radiation is quantified as the absorbed dose.
  - Units of measurement:

        - RAD – radiation absorbed dose
        - Gray (Gy) – 1 gray equals 100 RAD
- Vomiting is an early sign of exposure. The time to vomiting is a gross indicator of the dose of radiation received.
    - Vomit >4 hours post exposure – dose <2 Gy
    - Vomit 1 to 4 hours post exposure – dose 2 to 6 Gy
    - Vomit <1 hour post exposure – dose >6 Gy

Clinical presentation:

- The Acute Radiation Syndrome represents a constellation of illnesses that follow a generally predictable course over hours to days to weeks following radiation exposure.
    - Prodrome
        - Anorexia, nausea, vomiting, mild fever, diarrhea
    - Latent period
        - Period of apparent improvement lasting a few days to a few weeks. During this time, critical cell populations may be decreasing depending upon the dose received.
    - Overt Illness
        - Hematopoietic Syndrome – Occurs with exposure of >2 Gy. Damage to the bone marrow results in decreasing WBCs, platelets, and RBCs. Beginning upon presentation and repeated every 6 to 12 hours for a minimum of 48 hours post-exposure, the absolute lymphocyte count should be monitored:
            - If the count remains > 1200, a lethal dose is unlikely
            - If the count falls to between 300 to 1200, a significant exposure occurred
            - It the count falls to <300, a critical exposure has occurred
        - Gastrointestinal Syndrome – Occurs with exposure ≥6 Gy. Findings include severe diarrhea, fluid and electrolyte disturbances, potential opportunistic infection and sepsis, and bloody diarrhea.
        - Neurovascular Syndrome - Occurs with exposure of >12 Gy. Findings include fever, hypotension, prostration, ataxia, confusion, seizures, and increased intracranial pressure. Death is inevitable.
        - Cutaneous Syndrome – Initial skin changes can include erythema, swelling, blistering. Later changes include hair loss, nail changes, ulcers and areas of skin necrosis, and sloughing. If large areas are involved, usually signifies a poor prognosis due to large radiation exposure.
    - Recovery or Death
- Victims of a nuclear explosion will present with trauma and burns, in addition to the radiation concerns.

Mortality: Varies depending upon the dose of radiation received.

Transmissibility: Surface contamination with radioactive materials may be spread by contact.

Exposure control:

- Level C protection is recommended if performing decontamination activities
    - Decontamination of staff is indicated after treating contaminated patients
- Contaminated victims do not pose a significant health risk to medical care providers
- Standard precautions typically suffice once decontamination has been performed
    - Irradiated patients pose no risk to healthcare personnel (just as patients who have undergone x-rays do not pose a risk)
- Respirators should be used if care is required prior to formal decontamination.
- Personal dosimeters should be used to monitor individual provider exposure.

Diagnosis: Radiation poisoning is a diagnosis based upon the clinical presentation, any known threat and the identified presence of radioactive material at a scene or on the patient.

Management:

- Treatment of trauma and burns takes precedence over treatment of radiation exposure.
    - If surgical operation is required, ideally it should be performed within the first 48 hours of exposure or delayed until after hematopoietic recovery to minimize the risk of post-operative infection.
    - Remove particulate remnants from open wounds.
- External radiation survey should be conducted using a Geiger-Mueller counter to determine the presence of external contamination.
- If external contamination is found, decontaminate with soap and water.
- Monitor the patient for fluid and electrolyte imbalance and evidence of volume depletion and hemodynamic compromise
- Perform sequential CBCs (at least daily) with differential to assess for progressive decline in lymphocyte count.
- Consider colony stimulating (hematopoietic growth) factors and medications (cytokines) for evidence of bone marrow suppression.
- Consider early administration of radionuclide-specific decorporation agents as appropriate:

DECORPORATION AGENT	USED FOR
DTPA	Americium
	Berkelium
	Californium

	Curium
	Plutonium
	Uranium (within 4 hours)
BAL (Dimercaprol	Polonium
Prussian Blue (Radiogardase)	Cesium 137
	Thallium
Forced Water Intake and Diuresis	Tritium
Bicarbonate Diuresis	Uranium
Potassium Iodide	Radioactive Iodine (I125, I131) ASAP within 4 hours

    - Potassium iodide (KI) must be given as soon as possible after exposure and within 4 hours to block uptake of radioactive iodine (I125, I131) by the thyroid gland.
        - After 12 hours post exposure, there is no benefit to KI administration.
- Provide supportive and symptomatic care with focus on infection prevention, including administration of antibiotics.
- Ondansetron (Zofran) may be used for radiation-induced vomiting
- Patients with moderate or severe radiation exposure (>2 to 3 Gy) should be placed in isolation rooms.

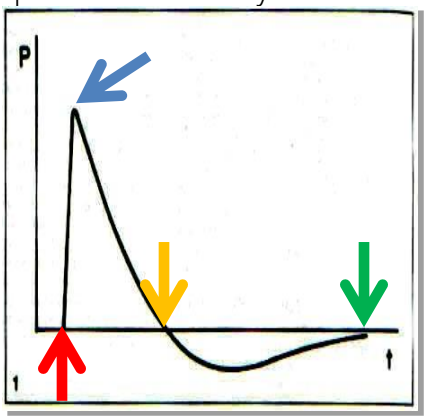
EXPLOSIVES

Agent:

- The detonation of an explosive involves the sudden conversion of an explosive material (solid or liquid) into a gas under high temperature and pressure
    - High order explosives – detonate faster than the speed of sound
        - Dynamite, nitroglycerin, C-4, Semtex, ammonium nitrate-fuel oil (ANFO), TATP, PETN
    - Low order explosives – react slower than the speed of sound in a process of deflagration (burning)
        - Gunpowder, black powder, pipe bombs, Molotov cocktails (petroleum based)

Blast Physics:

- The detonation of high order explosive results in a blast and pressure wave that is represented by the Friedlander Curve. The degree of maximum pressure is termed the blast overpressure and only occurs with high order explosives.



    - The red arrow indicates the moment of detonation
    - The blue arrow indicates the maximum generated pressure (blast overpressure)
    - The pressure curve from the red arrow to the orange arrow is the positive pressure phase
    - The pressure curve from the orange arrow to the green arrow is the negative pressure phase

Pathophysiology:

- The extent of damage from exposure to a high order explosive depends upon:
    - The magnitude of the blast overpressure
    - The duration of the blast overpressure
    - The medium in which the detonation occurred
    - The distance from the blast wave

- Whether the explosion took place in an open or enclosed space which can reflect or focus the blast wave

Pathophysiology (cont'd)

- Exposure to the blast overpressure wave damages primarily air containing organs and structures.
  - Pounds/sq inch associated with injury
    - 2 - 5 psi: TM rupture.
    - 15 psi: Lung damage threshold.
    - 30 - 40 psi: Lethality threshold.

Clinical presentation:

- Blast injury has traditionally been characterized as Primary, Secondary, Tertiary, and Quaternary.
  - Primary Blast Injury – this is the injury that results from exposure to the blast overpressure and *only results from high order explosives*. The incidence of primary blast injury ranges from 2% to 75% depending upon the report and the circumstances of the incident. Injuries include:
    - Ruptured tympanic membrane
    - "Blast lung" which includes pulmonary contusions, pneumothorax, pneumomediastinum, pulmonary laceration, bronchopleural fistula which may lead to air embolus, alveolar damage with interstitial and alveolar hemorrhages and edema.
      - The classic presentation of blast lung includes dyspnea, bradycardia, and hypotension. Chest x-ray reveals a characteristic "butterfly" appearance.
    - Air embolism can present as stroke, MI, acute abdomen, blindness, deafness, or spinal cord injury.
    - Gastrointestinal injury including bowel and mesenteric contusions, bowel rupture, and solid organ damage.
    - Brain injury such as concussion.
  - Secondary Blast Injury – these injuries are generally more common that primary blast injury and result from high velocity shrapnel and debris from the explosion striking the victim typically resulting in penetrating trauma, although large debris fragments may cause blunt injury.
  - Tertiary Blast Injury – tertiary blast injuries result when the victim is thrown or propelled by the blast wind into another object. Typical injuries include:
    - Fractures (often multiple) and dislocations
    - Closed head and traumatic brain injury
    - Extremity amputations
    - Multiple abrasions and contusions as the patient tumbles about
  - Quaternary Blast Injury – injuries or complications not included in the other categories including burns, toxic products such as smoke, fumes or carbon

monoxide, angina, asthma or COPD exacerbations due to dust inhalation, embedded body parts from a suicide bomber, and crush injuries.

Mortality: Varies depending upon the circumstances of the explosions (i.e., open space [lower mortality] vs enclosed space [higher mortality]), the type of explosive used (higher mortality for high order explosives), and the injuries sustained.

Diagnosis:

- History of presence at an explosion should prompt a careful physical exam and diagnostic studies.
- Complaints of hearing loss, tinnitus, ear pain, nausea, amnesia indicate proximity to a blast.
- Findings of bleeding from the ear or nose, cyanosis, cough, hemoptysis, rales, rhonchi, abdominal tenderness, guarding or rigidity require further evaluation.
- The presence of any open wounds, no matter how small or insignificant appearing, requires evaluation for possible foreign body or shrapnel.

NOTE: Communication difficulties may exist with victims from an explosion because of hearing loss. Written communication and instructions may be necessary.

Management:

- Initial care, as for any trauma victim, is focused upon assuring an adequate airway, ventilation and circulation.
- Diagnostic studies include chest x-ray, pulse oximetry, serial hemoglobin determinations, and computed tomography of the chest, abdomen, and head as necessary.
- Supplemental oxygen is provided for hypoxemia.
- If positive pressure ventilation is being performed, careful observation for barotrauma and tension pneumothorax as well as air embolus must be undertaken.
    - Tube thoracostomy should be performed as necessary.
    - Prophylactic tube thoracostomy may be considered, particularly prior to general anesthesia for surgery and air transport.
- Signs of arterial gas embolism are treated with 100% oxygen administration, left lateral decubitus position with the head down, and treatment in a hyperbaric chamber, if available.
- Gastrointestinal injuries are treated as for any trauma patient; however, it is important to remember that signs of injury may be delayed.
- Penetrating trauma is managed as for any trauma patient with penetrating injury with careful evaluation for the presence of shrapnel or other foreign bodies.
    - Up to 10% of victims with secondary blast injury will have penetrating eye trauma requiring careful evaluation and ophthalmologic referral.
    - Open wounds may be contaminated. Delayed primary closure should be considered.
    - Assure adequate tetanus immunization status.

- Orthopedic injuries are managed as for any trauma patient.
    - Patients with amputations from blast injury often have a grim prognosis due to uncontrolled hemorrhage from the amputation as well as exposure to the overpressure. Tourniquet application is indicated to control hemorrhage from an amputated extremity.

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