NWTHS/PANHANDLE
EMERGENCY MEDICAL SERVICES SYSTEM

RESPONSE TO WEAPONS OF MASS DESTRUCTION INCIDENTS
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Mission Statement

The NWTHS/PEMSS Weapons of Mass Destruction structured response protocol was developed to serve the citizens of The Texas Panhandle, and the City of Amarillo, by cooperating to achieve the comprehensive and efficient mitigation, preparedness, response, and recovery of a terrorist event involving a WMD that may occur within the PEMSS Region.

Goals

- Provide a focal point for weapons of Mass destruction issues within The City of Amarillo and the Texas Panhandle.
- Promote open communication regarding terrorism involving weapons of mass destruction incidents.
- Promote education regarding terrorism involving weapons of mass destruction incidents.
- Facilitate and recommend planning, training, equipment, and address interoperability issues regarding terrorism involving weapons of mass destruction incidents.
- Plan, coordinate, facilitate, and assess comprehensive state level training exercises regarding terrorism involving weapons of mass destruction incidents.
- Provide exercise support to jurisdiction’s terrorism involving weapons of mass destruction incident exercises.
MEDICAL TREATMENT PROTOCOLS

An act of terrorism in a metropolitan area may cause major health and medical consequences that could rapidly overwhelm virtually all local health facilities. In evaluating potential terrorist activity, consider the release of chemical or biological agents into crowded and contained areas, such as sports stadiums, office or public buildings, and transportation systems. Rapid identification of the chemical or biological agent is critical to proper disposition of patients and to management of affected areas.

All of these issues highlight the importance of proper education and advance planning. Effective response to a terrorist incident hinges on comprehensive planning and interagency cooperation. Local police, fire, EMS, and Disaster & Emergency Services (DES) agencies should form the first line of response.

First Responder Issues
Pre-incident planning for a terrorist event requires the identification of areas within each jurisdiction that may be vulnerable to terrorist attack. Although it is important to profile locations that are likely to be targeted by terrorists, it is important to remember that the proximity of a potential target is not the only threat. Chemical agents pose a threat during every phase of their existence: production, packaging, storage and delivery to the intended target. All of those locations and the places in between are at risk. Many common hazardous materials used in industry pose the same threat to emergency responders as the chemicals classified as nerve, blister, blood and choking agents. A terrorist can inflict equivalent damage just as effectively with a toxic industrial chemical, hazardous material or a military agent.

Signs and Symptoms of Attack
Chemical agents do not need to be lethal to be disruptive. Unlike an attack with explosives, the fact that a terrorist has attacked with a chemical agent may not always be obvious at first. Many of the early signs and symptoms produced by chemical warfare agents may resemble those of a variety of disorders, including stress. Other symptoms include psychological withdrawal, palpitations, gastrointestinal distress, headaches, dizziness, and inattentiveness. It may be the medical community that first recognizes that a chemical agent has been released. The patient's clinical presentation will offer clues about the type of toxic substance used. Victims exposed to a high concentration of nerve agents will be down with no obvious signs of trauma. On the other hand, victims exposed to blister agents may show no symptoms for up to 48 hours.

Responders should be alert for the following signs that a chemical agent may have been dispersed:

- Explosions that dispense liquids, mists or gas
- Explosions that seem only to destroy a package or bomb device
- Unscheduled and unusual spray being disseminated
- Abandoned spray devices
- Numerous dead animals, fish and birds
- Lack of insect life
- Mass casualties without obvious trauma
- Definite pattern of casualties and common symptoms
- Civilian panic in potential target areas (government buildings, public assemblies, etc.)

Responding To The Scene
If a chemical attack is suspected, first responding units must approach with caution. A responder who becomes a victim becomes more of a drain on available resources. They must be wary of contaminated terrain and contaminated objects. Hazmat response protocols must be initiated, as well as unified incident command. Victims and potential victims must be evacuated rapidly from the contaminated area and decontaminated as quickly as possible, if necessary. In certain situations, treatment may be initiated within the hot and/or warm zones of an incident by properly trained and equipped personnel. Control zones must be set up and the perimeter, as directed by incident command, secured. Responders must be on the alert for secondary devices, and must preserve the scene for evidence.
Select Agents of Terrorism

Chemical Agents
A chemical agent may be defined as a compound that, through its chemical properties, produces lethal or damaging effects in humans, animals, plants or materials. Unlike biological agents, chemical agents are usually man-made through the use of industrial chemical processes. Chemical agents are classified by their effects:

- **Lethal agents** are designed to kill, and are broken down into two subcategories: nerve agents and blood agents.
  - Nerve agents, the most deadly of all chemical agents, disrupt nerve transmission with organs and are quickly fatal in cases of severe exposure.
  - Blood agents (cyanides) interfere with the blood's ability to transport oxygen throughout the body.
- **Blister agents**, or vesicants, cause a blistering of the skin and mucous membranes, especially the lungs.
- **Choking agents**, or pulmonary agents, irritate the lungs, causing them to fill with fluid.
- **Incapacitating agents**, cause an intense (but temporary) irritation of eyes and respiratory tract.

The potential of the agent to do damage is measured by how readily it disperses. Chemical agents are either **persistent** or **non-persistent**. Wind and rain will increase the dispersion rate of a chemical agent. Heavy rains act to dilute both persistent and non-persistent agents and facilitate penetration into the ground.

**Persistent agents** have low volatility, evaporate slowly and are particularly hazardous in liquid form. They stay around for long periods of time (24 hours or longer) and contaminate not only the air but objects and terrain as well. Mustard and the nerve agent VX are examples of persistent agents.

**Non-persistent agents** are volatile and evaporate quickly, within several hours. Gases, aerosols, and highly volatile liquids tend to disperse rapidly after release. Phosgene, cyanide and the G series of nerve agents (with the exception of GD-Soman) are non-persistent agents. Because of their volatility, they pose an immediate respiratory hazard but are not particularly hazardous in liquid form.
Nerve Agents (GA, GB, GD, GF, VX)
The nerve agents Tabun (GA), Sarin (GB), Soman (GD), GF, and VX are considered the primary agents of threat because of their high toxicity and effectiveness through multiple routes of entry. They are absorbed through the eyes, respiratory tract, and skin. Nerve agents are chemically similar to organophosphate pesticides, but are up to a thousand times more potent. VX is the most potent of all nerve agents. It's approximately 50 times more toxic than cyanide gas. The dose that would be lethal to 50% of people exposed, or LD50, is 10 mg of liquid nerve agent VX on the skin of a 155-pound man. This is equivalent to a tiny drop about the size of Lincoln’s head on the back of a penny. The dosage lethal to 50% of the people exposed to vapors, or LC50, is 30 mg minutes per cubic meter. Nerve agent vapor in its pure form is colorless. Liquid GB is clear and resembles water. Liquid VX is amber and looks like light weight oil. VX is the least volatile of the nerve agents. That is, it is slowest to evaporate. GB is the most volatile; it evaporates more rapidly but even GB evaporates more slowly than water.

Nerve Agent Effects on the Human Body
When poisoned by a nerve agent, the action of the enzyme acetylcholinesterase is blocked. The normal function of acetylcholinesterase is to break down or hydrolyze the chemical acetylcholine. Acetylcholine is a neurotransmitter, or messenger chemical. There are two types of acetylcholine receptors, the nicotinic, that are found in the skeletal muscles, and the muscarinic, which are found in smooth muscles, glands and the central nervous system. Atropine blocks the muscarinic receptors. Nerve paths, which are divided into sections with gaps between the nerve endings and between the nerve ending and the target organ, are used to pass a command from the central nervous system to various organs. These gaps are crossed by acetylcholine, the messenger, which relays the command on to the next step and finally to the target. A main effect of nerve agents is to cause miosis, or pinpointing, of the pupils which diminishes visual acuity. Under normal conditions acetylcholine is broken down by the acetylcholinesterase, thus stopping the action. However, when a nerve agent inhibits the acetylcholinesterase, this enzyme cannot perform its normal function of hydrolyzing the acetylcholine. Acetylcholine then accumulates along the nerve path, and the target organ’s action continues uncontrolled. Muscles become hyperactive and twitch uncontrollably, and if the affected nerve is stimulating a gland, it will continue to secrete, resulting in excess drooling, tearing, urination, defecation, and emesis. Muscarinic effects may cause the most serious complications, including bronchoconstriction, laryngospasm, and respiratory depression or arrest.

TREATMENT GUIDELINES
The treatment guidelines provided below assume that the medic/first responder is relatively certain that nerve agent poisoning has occurred. This may initially be determined by the pattern of prevailing symptoms outlined above.

DRUG THERAPY
The main principles of therapy for nerve agent poisoning are early treatment, assisted ventilation, bronchial suction, muscarinic cholinergic blockade (atropine), enzyme reactivation. (2 Pam Cl) and anticonvulsants (diazepam).

- **Atropine** intramuscular or intravenously is the drug of choice for treating nerve agent poisoning. It will dry secretions, reduce bronchoconstriction, and decrease gastrointestinal motility. Atropine administered with the auto injector will show some effectiveness in 3-5 five minutes.
- **Pralidoxime chloride** (2-PAM Cl, Protopam) in the auto injector (600 mg, 2 ml) is the second drug indicated for use in nerve agent poisoning cases. 2-PAM removes the nerve agent from the enzyme acetylcholinesterase and should be administered as early as possible after exposure.
- **Diazepam** in the 10-mg auto injector for use in controlling convulsions when intravenous access is unavailable or unsafe to attempt in the immediate care setting. The first responder is to administer one diazepam auto injector to the intended patient immediately after injecting the third MARK I Kit in severe poisoning cases. **Diazepam is not for self-administration.**
- **Albuterol** 2.5 mg (1 unit dose) via nebulization for any patient who presents with bronchospasm (wheezing) secondary to nerve agent exposure.
- **Atrovent** 0.5 mg (1 unit dose) via nebulization may also assist in drying secretions in any patient who presents with bronchospasm (wheezing) secondary to nerve agent exposure.
Mark I kits (MK-1 Kit)
A Mark I Chemical Agent Treatment kit is a rapid way to administer atropine and 2-PAM Chloride. Each treatment kit contains two auto-injectors, one injector contains 2 milligrams of atropine, and the other contains 600 milligrams of 2-PAM Chloride.

Decontamination
Patients suspected to be contaminated with liquid agent can be decontaminated with soap and water (preferred method) or a 0.5% hypochlorite solution (a 0.5% hypochlorite solution is prepared by mixing 1 part 5% hypochlorite [household bleach] with 9 parts water). Patients exposed only to vapor do not require decontamination.

Emergency Treatment - All Patients
Basic ABC management is critical in this patient population. In more severe cases early control of the patients airway and assisted ventilation may be required if resources are available.

**Mild Exposure: Pinpoint pupils only**
- No antidote administration recommended.

**Mild Exposure: Pinpoint pupils & Sudden Nasal discharge, No Shortness of Breath**

Atropine Sulfate (Basic Responder)
- **Age greater than 10 years** – 2 mg IM (1 atropen) x 1.
- **Age less than 10 years** – 2 mg IM (1 atropen) x 1.

Atropine use in patients < 10 years: *A single 2 mg IM auto-injector dose may be given only when IV route (ALS provider) is unavailable in order to provide a more exact IV dose.*

Atropine Sulfate (ALS Providers)
- **Age greater than 10 years** – 1 mg IV x 1.
- **Age less than 10 years** – 0.05 mg/kg IV/IO up to 1 mg x 1 or 0.5 mg IM if no IV access x 1.

Pralidoxime (2-PAM Cl)
- Not Indicated.

**Moderate Exposure: Moderate Shortness of Breath and/or Vomiting**

Atropine Sulfate (Basic Responder)
- **Age greater than 10 years** – 4 mg IM (2 Atropen-MK1 Kits). Repeat 2 mg (1 Atropen) every 5-10 minutes until SOB is relieved.
- **Age less than 10 years** – 2 mg IM (1 Atropen-MK1 Kit). Repeat 2 mg (1 Atropen) once in 10 minutes if still SOB is still present.

Atropine Sulfate (ALS Providers)
- **Age greater than 10 years** – 2 mg IV/IM repeated every 5-10 minutes until signs of atropinization occurs (secretions dry, pupils dilate, tachycardia etc.).
- **Age less than 10 years** – 0.05 mg/kg IV/IO/IM doubled every 5-10 minutes until signs of atropinization occurs (secretions dry, pupils dilate, tachycardia etc.).

Pralidoxime (All Providers)
- **Age greater than 10 years** – 1200 mg IM via 2 auto-injectors.
- **Age less than 10 years** – 600 mg IM via 1 auto injector.

Diazepam
- Not indicated unless seizure activity is present, if seizing follow severe exposure dosing.
Severe Exposure: Severe Shortness of Breath, Apnea, Unconsciousness, Seizures

Atropine Sulfate (Basic Responder)

- **Age greater than 10 years** - 6 mg IM (3 Atropens-MK1 Kits). Repeat 2 mg (1 Atropen) dose every 5-10 minutes until SOB is relieved / secretions dry or are drying.
- **Age less than 10 years** - 2 mg IM (1 Atropen-MK1 Kit). Repeat same dose every 5-10 minutes until SOB is relieved / secretions dry or are drying or ALS IV dosing is available.

Atropine Sulfate (ALS Providers)

- **Age greater than 10 years** - 2 mg IV/IM repeated every 5-10 minutes until signs of atropinization occurs (secretions dry, pupils dilate, tachycardia etc.).
- **Age less than 10 years** – 0.05 mg/kg IV/IO/IM doubled every 5-10 minutes until signs of atropinization occurs (secretions dry, pupils dilate, tachycardia etc.).

Pralidoxime (All Providers)

- **Age greater than 10 years** – 1800 mg IM (3 auto injectors) x 1.
- **Age less than 10 years** – 600 mg IM (1 auto injector) x 1

Diazepam (Basic Responder)

- **Age greater than 10 years** – 10 mg IM (1 CANA injector) when atropine and 1800 mg of 2-PAM is administered or anytime seizure activity occurs.
- **Age less than 10 years** – 10 mg IM (1 CANA injector) only if seizure activity is present and no IV access or advanced care is available.

Diazepam (ALS Providers)

- **Age greater than 10 years** – 5-10 mg IV at 2 mg/minute or 10 mg IM (1 CANA injector) when atropine and 1800 mg of 2-PAM is administered or anytime seizure activity occurs.
- **Age less than 10 years** – 10 mg IM (1 CANA injector) if no IV or IO access and only if seizure activity is present or 0.1-0.3 mg/kg IV or IO if vascular access available and seizure activity present.

**WATCH FOR RESPIRATORY DEPRESSION IN PATIENTS LESS THAN 10 YEARS**

CYANIDE COMPOUNDS (AC, CK)
Cyanide is one of the most rapidly acting poisons known to man. Although they have been used since World War I, cyanide compounds are highly volatile, rendering them less useful than chlorine, but because of the extensive use of cyanide in industry in the US, this agent still presents a credible threat for terrorist use. Hydrogen cyanide (AC) and cyanogen chloride (CK) as substances used in warfare.

**Cyanide Effects on the Human Body**

While liquid cyanide can be absorbed through the skin or eyes, the primary route of exposure is by inhalation or ingestion. Following absorption, cyanide is distributed rapidly to all organs and tissues in the body. Cyanide combines with ferric iron in cytochrome a3 (a component of the cytochrome oxidase complex in mitochondria) and inhibits this enzyme. Signs and symptoms include air hunger, hyperpnea, apnea, seizures, coma, and death.

**Emergency Treatment Guidelines**

Prompt recognition of cyanide poisoning is crucial; however, the signs and symptoms of acute cyanide poisoning are often nonspecific. Cyanide poisoning should be considered in the differential diagnosis of sudden death and sudden global neurologic deficit (coma, convulsions and encephalopathy, severe acidosis and shock), especially in multiple patients. The time to onset of symptoms and the severity of symptoms depend on the dose and route of exposure. Coma, tonic-clonic seizures, cyanosis, shock, and severe metabolic acidosis reflect a serious overdose requiring prompt institution of therapy. **Cyanosis is absent as long as the patient is breathing because of abnormal peripheral use of oxygen.** When present, cyanosis is a late sign. The odor of bitter almonds emanating from the patient may be present, although up to 40% of the population is unable to detect this odor.

**Decontamination**

Patients should be immediately removed from the area to a clean atmosphere. Because cyanide exposure is dose dependent, rapid evacuation may prevent moderate dose patients from progressing to severe dose patients. Cyanide is generally deployed in a gaseous state. If clothing is wet, it should be removed and sealed. Underlying skin can be decontaminated using soap and water.

**Emergency Treatment**

**Moderate Exposure: Conscious with Tachypnea, Dizziness, N/V or Headache**

- Immediate attention should be directed toward removal from the source of contamination, decontamination, if necessary, assisted ventilation, administration of 100 % oxygen, insertion of intravenous lines and institution of cardiac monitoring, if available.
- Frequent reassessment for symptom progression
- If unsure of severity, treat as for a severe exposure.

**Severe Exposure: Unconscious, Apnea**

*Management is Contingent Upon Immediate Availability of a Cyanide Antidote Kit*

**Amyl Nitrate Capsule (use only when IV access is not readily available) (ALS ONLY)**

- **All Ages** - Crush and allow patient to inhale for 30 seconds. If intubated or being ventilated with a BVM, place crushed capsule into BVM reservoir bag and ventilate for 30 seconds.

**Sodium Nitrite 3% (ALS ONLY)**

- **Age greater than 10 years** – 300 mg (10 mL) IV.
- **Age less than 10 years** – 0.3 mL/kg slow IV at 2-5 mL/minute, not to exceed 10 mL.

**Sodium Thiosulfate 25% (ALS ONLY)**

- **Age greater than 10 years** – 12.5 grams (50 mL) IV.
- **Age less than 10 years** – 1.6 mL/kg slow IV at 3-5 mL/minute, not to exceed 50 mL.

- May repeat adult IV medications once, at half the initial dose, when symptoms persist and medications are available. Do not repeat pediatric doses unless directed by a physician.
- The management of pulmonary edema is best achieved with positive pressure ventilation.
- Infuse IV medications more slowly when hypotension is present or occurs.

**Choking agents (Phosgene, Chlorine, Chloropicrin)**
Pulmonary ‘Choking’ agents generally produce mild to severe pulmonary symptoms, including the potential for profound pulmonary edema with delayed onset. Generally, patients are exposed and not contaminated with these agents, requiring no decontamination. Liquid phosgene has a high rate of evaporation and does not pose a threat other than as the source of vapor.

**Physical Findings**
Dyspnea and cough. Initial physical findings of exposure to high levels of a pulmonary agent are irritation of the mucous membranes, including eyes, nose, and airways. These symptoms will decrease with exposure to a clean atmosphere and oxygen therapy. Severe exposures may develop delayed onset pulmonary edema (2-24 hour onset). In severe cases of pulmonary edema, the patient may become hypotensive due to fluid shift.

**Decontamination**
*Patients should be immediately removed from the area to a clean atmosphere.* Because pulmonary agents are generally a gas in normal atmospheric conditions, patients will be exposed and not contaminated. In these situations, no decontamination is needed.

**Emergency Treatment**
- Immediate attention should be directed toward assisted ventilation, administration of 100 % oxygen, insertion of intravenous lines and institution of cardiac monitoring, if available.
- Symptomatic treatment per Standards of care (no specific antidote).

**Severe Exposure: Evidence of Non-Cardiogenic Pulmonary Edema**
- Consider early intubation and aggressive ventilatory support using PEEP (if available). Begin at 5 and increase to 10 CM H\textsubscript{2}O if indicated (ILS/ALS only).
- Albuterol 2.5 mg via nebulization if wheezing. May repeat 2.5 mg as needed for symptomatic bronchospasm (may be initiated at the EMT-Basic level).
- Methylprednisolone (Solu-Medrol) 125 mg IV (ALS only).
Vesicant Agents: Sulfur Mustard (HD), Nitrogen Mustard (HN), Lewisite, Phosgene Oxime (CX)

Vesicant agents are named for their tendency to cause blisters. Mustard gas is the best known vesicant, originally used in World War I. Lewisite, unlike mustard and mustard derivatives, causes immediate pain and skin irritation. Vesicant agents generally cause delayed effects (the exception being Lewisite) and symptoms. Within the first two hours, intervention is generally limited to decontamination. If a patient is encountered outside the two-hour window, care provided is supportive with the most pressing issues being airway management as well as pain control.

Physical Findings
Generally none within the first several hours post exposure. If the patient is seen outside this window: Lesions are primarily cutaneous, but respiratory, ocular, and GI manifestations may occur, as well as cough, bloody sputum, and dyspnea.

Decontamination
Patients suspected to be contaminated should be decontaminated with soap and water. Medical providers will require the proper protective equipment as determined by unified command, for patient management. Decontaminate by blotting and cleansing with soap and water. Avoid scrubbing and the use of hot water.

Note: Latex and rubber will absorb Mustard. Remember that time is critical for effective mustard decontamination because blister agents become “fixed” to tissue components within two minutes after deposition.

Management/Treatment
- Immediate attention should be directed toward assisted ventilation, administration of 100 % oxygen, insertion of intravenous lines and institution of cardiac monitoring, if available.
- Symptomatic treatment per Standards of care (no specific antidote).

Severe Exposure: Pulmonary Edema (non-cardiogenic)
- Consider early intubation and aggressive ventilatory support using PEEP (if available). Begin at 5 and increase to 10 CM H\text{2}O if indicated (ILS/ALS only).
- Albuterol 2.5 mg via nebulization if wheezing. May repeat 2.5 mg as needed for symptomatic bronchospasm (may be initiated at the EMT-Basic level).
Lacrimator Agents (Tear Gas)

Lacrimator (tearing) agents are widely used by law enforcement and the military. Types of lacrimator agents include bromobenzyl cyanide (CA), ortho-chlorobenzylidenemalonitrile (CS), dibenoxazepine (CR), 2-chloroacetophenone (CN), chloroacetophenone in chloroform (CNC), and chloroacetophenone and chloropicrin in chloroform (CNS).

Physical Findings
The most common effects are nasal and ocular discharges, photophobia, and burning sensations in the mucous membranes. Prolonged exposure may produce tightness in the chest, shortness of breath, and malaise and may cause vesiculations or bullae. Physical injuries may be observed from explosive discharge or kinetic effects of projectiles.

Decontamination
Patients suspected to be contaminated should be decontaminated with soap and water. Medical providers require protective masks and clothing for patient management since lacrimator agents are transmitted by physical contact. Decontaminate by blotting and cleansing with soap and water.

Emergency Treatment
- High flow oxygen for all symptomatic patients.
- Symptomatic treatment per Standards of Care (no specific antidote).

Emesis-Inducing Agents

Emesis agents, also termed nausea gases, are not used routinely in the US. Examples of such compounds include adamsite (DM), diphenylcyanoarsine (DC), and diphenylchloroarsinine (DA).

Physical Findings
They produce respiratory and skin irritation effects similar to those of lacrimator agents, as well as profound nausea.

Decontamination
No decontamination is required in the field. Ordinary clothing protects against these agents; chemical insert masks and standard gloves are adequate.

Emergency Treatment
- High flow oxygen for all symptomatic patients.
- Symptomatic treatment per Standards of Care (no specific antidote).

Psychochemical Agents (LSD, Mescaline, Psilocybin)

Psychochemical agents, often referred to as incapacitating agents used as a non-lethal incapacitating agent has long intrigued military commanders. Several agents have been tested, including lysergic acid diethylamide (LSD), mescaline, psilocybin, and psilocin. The only successful agent in production is benzilate (BZ).

Physical Findings
BZ is a delayed onset (1-4 h) agent causing tachycardia, dizziness, vomiting, blurred vision, stupor, confusion, and random activity. Affected persons may be docile, belligerent, stuporous, or confused. They may appear intoxicated.

Decontamination
Decontaminate by washing with soap and water (preferred method) or with dilute bleach solution. Protective masks with charcoal filters provide adequate protection. Gloves are not necessary, since the agent is not absorbed through the skin.

Emergency Treatment
- High flow oxygen for all symptomatic patients.
- Symptomatic treatment per Standards of Care (no specific antidote).
Administration of MARK 1 Auto-Injectors

Any large muscle, such as the buttocks, may be used, but the lateral thigh muscle is the muscle of choice. Clothing does not have to be removed—injections can be made directly through clothing. Avoid hitting any buttons or other objects.

- The injectors are in a plastic holder and numbered one and two. The atropine injector (injector # 1), is administered first. Then the 2-PAM Chloride (injector # 2), follows if indicated.
- To use the auto-injector, remove the safety clip, place the head of the injector against the side of the mid thigh and press down the base until the spring drives the needle through the seal and into the muscle, injecting the medication.
- **Hold the atropine injector firmly in place for at least 10 seconds.** The seconds can be estimated by counting "one thousand one, one thousand two," and so forth. Firm pressure automatically triggers the coiled mechanism. This plunges the needle through the clothing into the muscle and at the same time injects the atropine antidote into the muscle tissue.
- Next, inject 2 PAM Cl injector using the same procedure as you did for the atropine. This will now complete one set of nerve agent antidotes.
- Inject the CANA (diazepam), when indicated, using the same procedure as you did for the atropine and 2-PAM Cl. This will now complete one set of nerve agent antidotes.
- **Place all used injectors in clothing to keep track of doses.** As an alternative, use the enclosed red marker to make one vertical stripe on the victim’s or responder’s forehead or clothing.
- After administering the first set of injections, wait the appropriate time as previously outlined before administering a second set to assess for effectiveness. However, if you are able to walk and know who you are, you will not need a second set of antidote injections.

**WARNINGS**

- If symptoms of nerve agent poisoning are not relieved after administering one set of nerve agent antidote injections, seek someone else to check your symptoms. Another provider should administer the second and possibly a third set of injections, if needed.
- After administering one set of injections, you should decontaminate your skin if necessary, and put on any remaining protective clothing.
- Be careful handling used auto-injector units to prevent accidental needle sticks. The usual procedure to reduce the chance of accidental needle sticks is to bend the needle back against a hard surface at a 180-degree angle (when a sharps type container is unavailable).
### BASIC TREATMENT

**NERVE AGENTS**

**MILD EXPOSURE: PINPOINT PUPILS ONLY**

No antidote administration recommended.

<table>
<thead>
<tr>
<th>Age greater than 10</th>
<th>2 mgs IM</th>
<th>Age less than 10</th>
<th>2 mg IM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use IM doses in patients less than 10 only when IV route is unavailable.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**MILD EXPOSURE: PINPOINT PUPILS, SUDDEN NASAL DISCHARGE, NO SOB**

**ATROPEN AUTO INJECTOR**

<table>
<thead>
<tr>
<th>Age greater than 10</th>
<th>2 mgs IM</th>
<th>Age less than 10</th>
<th>2 mg IM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Use IM doses in patients less than 10 only when IV route is unavailable.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**MODERATE EXPOSURE: SOB AND/OR VOMITING**

**ATROPEN AUTO INJECTOR**

<table>
<thead>
<tr>
<th>Age greater than 10</th>
<th>4 mgs IM</th>
<th>Repeat 2 mg dose every 5-10 minutes until SOB is relieved.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age less than 10</td>
<td>2 mg IM</td>
<td>Repeat 2 mg dose once in 10 minutes if SOB is still present.</td>
</tr>
</tbody>
</table>

**2-PAM CI AUTO INJECTOR**

<table>
<thead>
<tr>
<th>Age greater than 10</th>
<th>1200 mg IM (2 auto injectors) x 1.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age less than 10</td>
<td>600 mg IM (1 auto injector) x 1.</td>
</tr>
</tbody>
</table>

**SEVERE EXPOSURE: SEVERE SOB, NOT BREATHING, UNCONSCIOUS, SEIZURES**

**ATROPEN AUTO INJECTOR**

<table>
<thead>
<tr>
<th>Age greater than 10</th>
<th>6 mg IM</th>
<th>Age less than 2</th>
<th>2 – 2 mg IM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Repeat 2 mg every 5-10 minutes until SOB is relieved / secretions are drying, ALS IV available.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**2-PAM CI AUTO INJECTOR**

<table>
<thead>
<tr>
<th>Age greater than 10</th>
<th>1800 mg IM x 1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age less than 10</td>
<td>600 mg x 1</td>
</tr>
</tbody>
</table>

**CANA AUTO INJECTOR (DIAZEPAM)**

<table>
<thead>
<tr>
<th>Age greater than 10</th>
<th>10 mg IM.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age less than 10</td>
<td>10 mg IM only if seizure activity is present, no IV, ALS available.</td>
</tr>
</tbody>
</table>

*Atropen use in patients < 10 years: A single 2 mg IM auto-injector dose may be given only when IV route (ALS provider) is unavailable in order to provide a more exact IV dose (0.05 mg/kg dose).*

### DECONTOamination PROCEDURES

**REMOVE PATIENT IMMEDIATELY TO A CLEAN ATMOSPHERE**

**NERVE AGENTS:** use soap and water or 0.5% hypochlorite solution for liquid exposures. If exposed is only to vapor, no decontamination required.

**CHOKING AGENTS:** none required, If gas exposure only.

**VESICANTS:** blot / cleanse with soap and water. Avoid scrubbing and hot water.

**CYANIDE:** If clothing is wet, remove and seal. Decon skin using soap and water.

**LACTRIMATORS / EMESIS INDUCING / PSYCHOCHEMICAL:** Use soap and water to clean. Providers require protective masks and clothing for patient management.

### AIRWAY / INHALATION INJURY

**Pulmonary edema, respiratory distress, wheezes (bronchospasm)**

**APNEA, SEVERE SOB:** BVM ventilation, if available and high flow oxygen in all cases.

**PULMONARY EDEMA:** BVM ventilation if available, high flow oxygen in all cases.

**BRONCHOSPASM:** Albuterol 2.5 mg via nebulization. May repeat same dose as needed until ALS intervention occurs.
# ADVANCED TREATMENT

## NERVE AGENTS

### MILD EXPOSURE: PINPOINT PUPILS ONLY
- No IM or IV antidote administration recommended.

### MILD EXPOSURE: PINPOINT PUPILS AND NASAL DISCHARGE

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ATROPINE</strong></td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>2 mg IV/IM x1</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>0.05 mg/kg IV up to 1 mg or 0.5 mg IM if no IV access x 1</td>
</tr>
</tbody>
</table>

### MODERATE EXPOSURE: SHORTNESS OF BREATH AND/OR VOMITING

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ATROPINE</strong></td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>4 mgs IV/IM. Repeat q 5-10 min. until atropinization occurs.</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>0.05 mg/kg IV/IO/IM, double q 5-10 min. until atropinization occurs.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PRLIDOXIME CI</strong></td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>1200 mg IM (2 auto injectors) x 1</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>600 mg IM (1 auto injector) x 1</td>
</tr>
</tbody>
</table>

### SEVERE EXPOSURE: SEVERE SOB, NOT BREATHING, UNCONSCIOUS, SEIZURES

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ATROPINE</strong></td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>6 mgs IV/IM. Repeat q 5-10 min. until atropinization occurs.</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>0.05 mg/kg IV/IO/IM, doubled q 5-10 min. until atropinization occurs.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PRLIDOXIME CI</strong></td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>1800 mg IM x 1</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>600 mg IM</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DIAZEPAM</strong></td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>10 mg IM or 5-10 mg at 2 mg/min if IV access available</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>10 mg IM only if seizure activity is present and no IV access available or 0.1-0.3 mg/kg if IV/IO access is available</td>
</tr>
</tbody>
</table>

Atropine use in patients < 10 years: A single 2 mg IM auto-injector dose may be given only when IV route (ALS provider) is unavailable in order to provide a more exact IV dose.

## CYANIDE EXPOSURE

### MODERATE EXPOSURE: CONSCIOUS WITH TACHYPNEA, DIZZINESS, N/V or HA
- High flow oxygen therapy and frequent reassessment for symptom progression

### SEVERE EXPOSURE: UNCONSCIOUS, APNEIC

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amyl Nitrate Capsule (only if no IV access is readily available)</td>
<td>Inhalation for 30 seconds. If intubated, place into reservoir bag.</td>
</tr>
<tr>
<td>Sodium Nitrite 3%</td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>300 mg (10 mL) IV.</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>0.3 mL/kg slow IV/IO.</td>
</tr>
<tr>
<td>Sodium Thiosulfate 25%</td>
<td></td>
</tr>
<tr>
<td>Age greater than 10</td>
<td>12.5 grams (50 mL).</td>
</tr>
<tr>
<td>Age less than 10</td>
<td>1.6 mL/kg slow IV/IO.</td>
</tr>
</tbody>
</table>

## AIRWAY / INHALATION INJURY

Pulmonary edema, respiratory distress, wheezes (bronchospasm)

### NON-CARDIOGENIC PULMONARY EDEMA:
- Intubate and PEEP at 5-10 CMH₂O

### BRONCHOSPASM:
- Albuterol 2.5 mg, Ipratropium 0.5 mg neb PRN. Solu-Medrol 125 mg IV.
BIOLOGICAL AGENTS OF TERRORISM

Introduction
Biological warfare is the intentional use of micro-organisms, and toxins, generally, of microbial, plant or animal origin to produce disease and/or death in humans, livestock and crops. The attraction for bio-weapons in war, and for use in terrorist attacks is attributed to their low production costs, The easy access to a wide range of disease-producing biological agents, their non-detection by routine security systems, and their easy transportation from one location to another are other attractive features. Their properties of invisibility and virtual weightlessness render detection and verification procedures ineffectual and make non-proliferation of such weapons impossibility.

Biological/Chemical warfare characteristics
Biological, chemical and nuclear weapons possess the common property of wreaking mass destruction. Though biological warfare is different from chemical warfare, there has always been the tendency to discuss one in terms of the other, or both together. One of the main goals of biological warfare is the undermining and destruction of economic progress and stability. The emergence of bio-economic warfare as a weapon of mass destruction can be traced to the development and use of biological agents against economic targets such as crops, livestock and ecosystems.

Popular scenarios of bioterrorism include the use of psychotic substances to contaminate food; the use of toxins and poisons in political assassinations; raids with crude biological cloud bombs; use of dried viral preparations in spray powders; and low-flying cruise missiles adding destruction and havoc with genetically-engineered micro-organisms.

Preparedness and Response at the Local Level
The covert release of a biological agent will almost certainly go initially undetected in most areas of the country. Infected persons will begin to present at doctors' offices, managed care clinics, and hospital emergency department days, and perhaps weeks, after the release of the biological agent. In an overt release, officials will have advanced notice of the outbreak, but most local public health systems will be overwhelmed by community requests for information, prophylaxis, and treatment as soon as the threat was made public. In a biological release, there will most likely be no explosion, fire, or visible crime scene. Therefore, the first responders to a biological event will be alert EMS personnel, physicians, well-trained and prepared public health practitioners who detect the unusual event, and report it to their response partners.

When data about an outbreak are gathered and analyzed, there is a need to rapidly disseminate this information to other city departments, hospitals, clinics, independent physicians, and other providers. In a bioterrorist event, this information also needs to be shared with law enforcement agencies and the broad array of groups responsible for protecting the public. Effective education and communication, consisting of clear and concise information, will help assure the public that the situation is being addressed competently and quickly. This requires the effective use of a public information officer in conjunction with designated terrorism officers or other public health agency spokesperson. The CDC's "Interim Recommended Notification Procedures for Local and State Public Health Department Leaders in the Event of a Bioterrorist Incident" is a guidance resource which may be used. This protocol moves from initial recognition of an event through the state and federal response partners that may be involved in the incident.
Decontamination of Chemical Agents

Introduction
In protection against chemical warfare agents the decontamination is an important unavoidable part. The aim of decontamination is to rapidly and effectively render harmless or remove poisonous substances both on personnel and equipment. High decontamination capacity is one of the factors which may reduce the effect of an attack with CW agents. In this way, it may act as a deterrent.

The need for decontamination should be minimized to the extent possible by contamination avoidance and early warning. Equipment can be covered, for example, or easily decontaminated equipment can be chosen by means of suitable design and resistant surface cover.

Decontamination is time consuming and requires resources. Nerve agents and substances causing injury to the skin and tissue are easily soluble in, and penetrate many different types of material, such as paint, plastics and rubber, all of which renders decontamination more difficult. If CW agents have penetrated sufficiently deep, then toxic gases can be released from the material for long periods. By adding substances which increase the viscosity of a CW agent, its persistence time and adhesive ability can be increased.

These thickened agents will thus be more difficult to decontaminate with liquid decontaminants since they adhere to the material and are difficult to dissolve. Good body and respiration protection is essential for the maintenance of operational capacity for a limited period after a CW attack. If the aggressor has used persistent substances, the unit generally must be decontaminated when regrouping or reorganizing.

The need for decontamination can only be established by means of detection. If detection is not possible, then decontamination must be done solely on suspicion of contamination, e.g., if the unit has passed on the fringe of a contaminated area.

Decontaminants
All decontamination is based on one or more of the following principles:
- to destroy CW agents by chemically modifying them (destruction),
- to physically remove CW agents by absorption, washing or evaporation,
- to physically screen-off the CW agent so that it causes no damage.

Most CW agents can be destroyed by means of suitable chemicals. Some chemicals are effective against practically all types of substances. However, such chemicals may be unsuitable for use in certain conditions since they corrode, etch or erode the surface. Sodium hydroxide dissolved in organic solvent breaks down most substances but should not be used in decontaminating skin other than in extreme emergencies when alternative means are not available.

Decontaminants that have effect only against a certain group of substances can be an alternative in favor of a substance with general effect. The condition is that they will have a faster and better effect against the substance in question and/or a milder effect. Examples of such substances are chloramine solutions which are often used to decontaminate personnel. These have good effect against mustard agent and V-agents but are ineffective against nerve agents of G-type (Sarin, Soman, Tabun). A water solution of soda rapidly renders nerve agents of G-type harmless but when used in connection with V-agents, it produces a final product which is almost as toxic as the original substance. This does not prevent V-agents being washed-off with a soda solution, provided a sufficient amount is used. However, the final product will always be poisonous.

The disadvantage of specifically-acting decontaminants is partly that it is necessary to know which CW agent has been used and partly that access to several different types of decontaminating substances is required.
Decontamination methods
CW agents can be washed and rinsed away, dried up, sucked up by absorbent substances, or removed by heat treatment. Water, with or without additives of detergents, soda, soap, etc., can be used, as well as organic solvents such as fuel, paraffin and carburetor spirit. Emulsified solvents in water can be used to dissolve and wash-off CW agents from equipment.

When decontaminating by washing, consideration must be taken to the poisonous substance remaining in the decontaminant unless the CW agent has first been destroyed. The penetration ability of a CW agent can be enhanced when mixed with solvent. Today, there is an international development towards chemically resistant paints and materials, which implies that water-based methods will become more effective. However, the need for penetrating decontamination methods will remain for many years.

When washing with water - particularly with hot water and detergent - the CW agent will often be decomposed to some extent through hydrolysis. Detergents containing perborates are particularly effective in destroying nerve agents. Without an addition of perborates in the detergent, the hydrolysis products of V-agents may still remain toxic unless the pH is sufficiently high. Mustard agent is encapsulated by the detergent and, consequently, the hydrolysis rate decreases in comparison with clean water. However, the low solubility of mustard agent makes it difficult to remove without the addition of detergent, but the water used will still contain undestroyed mustard agent.

Small areas of terrain, e.g., first-aid stations or gun sites, may be decontaminated by removal of the top-soil. Another alternative is to cover the soil with chlorinated lime powder (sludge), which is a decontaminant with general effect and which releases active chlorine. CW agents which have penetrated into the soil, from where they release toxic vapor, are screened-off since the gas and liquid is destroyed by the chlorinated lime.

The physical screening-off of CW agents by covering them can be done in the terrain by spreading a layer of soil or gravel over the contaminated area. The effect will be improved if bleaching powder is mixed into the covering material. Another example of covering is to use special plastic foil to cover contaminated areas inside vehicles. In this way, the personnel will be protected against transfer of liquid.

Individual Decontamination
The most important decontamination measure naturally concerns the individual. If it is suspected that skin has been exposed to liquid CW agents, then it must be decontaminated immediately (within a minute). All experience confirms that the most important factor is time; the means used in decontamination are of minor importance. Good results can be obtained with such widely differing means as talcum powder, flour, soap and water, or special decontaminants.

In complete decontamination, clothes and personal equipment must also be decontaminated. If clothes have been exposed to liquid contamination, then extreme care must be taken when undressing to avoid transferring CW agents to the skin. There may be particular problems when caring for injured since it may be necessary to remove their clothes by cutting them off. This must be done in such a way that the patient is not further injured through skin contact with CW agents. During subsequent treatment it is essential to ensure that the entire patient is decontaminated to avoid the risk of exposing the medical staff to the CW agents.

A factor common to all individual decontaminants is that they can effectively remove CW agents on the surface of the skin. However, they have only limited ability to remove CW agents which have become absorbed by the skin, even though very superficially. CW agents that have penetrated into the skin therefore function as a reservoir which may further contribute to the poisoning also after completed decontamination.

In some cases, a wet method may give a better result in decontaminating deeply penetrated agents than a dry method. Reports from France indicate that a solution of potassium permanganate gives effective destruction of CW agents on the surface of the skin and also a certain penetrating effect. There are also individual decontaminants which can simultaneously function as a protective cream for use as a prophylactic.
Decontamination of Equipment
Immediate decontamination of personal equipment and certain other kinds of smaller equipment is generally done with individual decontaminants. However, these substances are only capable of decontaminating liquid CW agents covering the surface. The decontamination is mainly done to prevent further penetration into the material and to decrease the risk when handling the equipment.

CW agents easily penetrate different materials and into crevasses and will thus be difficultly reached by methods only designed for superficial decontamination. When a CW agent has penetrated into the surface, it is necessary to use some kind of deep-penetrating method. If such a method cannot be used, then it must be realized that the equipment cannot be used for a long period. Depending on the type of CW agent used and prevailing weather, i.e., temperature, wind velocity and precipitation (water solubility), the "self-decontamination" may take many days or even weeks. The absorption into the surface and natural chemical degradation are important factors influencing the self-decontamination period.

Decontamination by boiling is also an effective method. The advantage in comparison with heat is that hot water hydrolyzes and renders harmless many types of CW agents. The method may be of some interest in small-scale decontamination of rubber material, e.g., protective masks.

Decontamination of CW agents which have penetrated deeply into the surface can also be done with decontaminants which are capable of penetrating the contaminated material. There are different substances with varying properties. A modern decontaminant is the German Münster emulsion which consists of calcium hypochlorite, tetrachlorethylene, emulsifier ("phase transfer" catalyst) and water. Instead of tetrachlorethylene, the more environmentally harmless xylene is sometimes used.
BACTERIAL AGENTS

Bacteria are unicellular organisms. They vary in shape and size from spherical cells - cocci - with a diameter of 0.5-1.0 (m (micrometer), to long rod-shaped organisms - bacilli - which may be from 1-5 (m in size. Chains of bacilli may exceed 50 (m. The shape of the bacterial cell is determined by the rigid cell wall. The interior of the cell contains the nuclear material (DNA), cytoplasm, and cell membrane, that are necessary for the life of the bacterium. Many bacteria also have glycoproteins on their outer surfaces which aid in bacterial attachment to surface receptors on cells and are of special importance in their ability to cause disease. Under special circumstances some types of bacteria can transform into spores. The spore of the bacterial cell is more resistant to cold, heat, drying, chemicals and radiation than the bacterium itself. Spores are a dormant form of the bacterium and, like the seeds of plants, they can germinate when conditions are favorable.

Bacteria can cause diseases in human beings and animals by means of two mechanisms which differ in principle: in one case by invading the tissues, in the other by producing poisons (toxins). In many cases pathogenic bacteria possess both properties. The diseases they produce often respond to specific therapy with antibiotics. This manual will cover several of the bacteria or rickettsia considered to be potential BW threat agents: Bacillus anthracis (Anthrax), Vibrio cholerae (Cholera), Yersinia pestis (Plague), Francisella tularensis (Tularemia), and Coxiella burnetii (Q Fever).

ANTHRAX

Bacillus anthracis is a rod-shaped, gram-positive, sporulating organism, the spores constituting the usual infective form. Anthrax is a zoonotic disease with cattle, sheep and horses being the chief domesticated animal hosts, but other animals may be infected. The disease may be contracted by the handling of contaminated hair, wool, hides, flesh, blood and excreta of infected animals and from manufactured products such as bone meal, as well as by purposeful dissemination of spores. Transmission is made through scratches or abrasions of the skin, wounds, inhalation of spores, eating insufficiently cooked infected meat, or by flies. All human populations are susceptible. Recovery from an attack of the disease may be followed by immunity. The spores are very stable and may remain viable for many years in soil and water. They will resist sunlight for varying periods.

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

Diagnosis: Physical findings are non-specific. Possible widened mediastinum. Detectable by Gram stain of the blood and by blood culture late in the course of illness.

Treatment: Although usually not effective after symptoms are present, high dose antibiotic treatment with penicillin, ciprofloxacin, or doxycycline should be undertaken. Supportive therapy may be necessary.

Prophylaxis: A licensed vaccine for use in those considered to be at risk of exposure. Vaccine schedule is 0, 2, and 4 weeks for the initial series, followed by boosts at 6, 12, and 18 months and then a yearly booster. Oral ciprofloxacin for known or imminent exposure.

Decontamination: Secretion and lesion precautions should be practiced. After an invasive procedure or autopsy is performed, the instruments and area used should be thoroughly disinfected with a sporicidal agent (iodine or chlorine).
CHOLERA

Vibrio cholerae is a short, curved, motile, gram-negative, non-sporulating rod. There are two serogroups, O1 and O139, that have been associated with cholera in humans. The O1 serotype exists as 2 biotypes, classical and El Tor. The organisms are facultative anaerobes, growing best at a pH of 7.0, but able to tolerate an alkaline environment. They do not invade the intestinal mucosa, but rather “adhere” to it. Cholera is the prototype toxigenic diarrhea, which is secretory in nature. All strains elaborate the same enterotoxin, a protein molecule with a molecular weight of 84,000 Daltons. 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 can withstand freezing for 3 to 4 days. It is readily killed by dry heat at 117°C, by steam and boiling, by short exposure to ordinary disinfectants, and by chlorination of water.

Signs and Symptoms: Incubation period is 12-72 hours. Asymptomatic to severe with sudden onset. Vomiting, headache, intestinal cramping with little or no fever followed rapidly by painless, voluminous diarrhea. Fluid losses may exceed 5 to 10 liters per day. Without treatment, death may result from severe dehydration, hypovolemia and shock.

Diagnosis: Clinical diagnosis. ‘Rice water’ diarrhea and dehydration. Microscopic exam of stool samples reveals few or no red or white cells. Can be identified by darkfield or phase contrast microscopy, and by direct visualization of darting motile Vibrio.

Treatment: Fluid and electrolyte replacement. Antibiotics (tetracycline, ciprofloxacin or erythromycin) will shorten the duration of diarrhea and shedding of the organism.

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

Decontamination: Personal contact rarely causes infection; however, enteric precautions and careful hand-washing should be employed. Bactericidal solutions (hypochlorite) would provide adequate decontamination.
PLAGUE
Yersinia pestis is a rod-shaped, non-motile, non-sporulating, gram-negative, bipolar staining, facultative anaerobic bacterium. Plague is a zoonotic disease. Rodents (rats, mice, ground squirrels) in areas where plague is present can be infected with the bacteria. Fleas which 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. At near freezing temperatures, it will remain alive from months to years but is killed by 15 minutes exposure to 72°C. It also remains viable for some time in dry sputum, flea feces, and buried bodies but is killed with several hours of exposure to sunlight.

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

**Diagnosis:** Clinical diagnosis. After an incubation period varying from 2-3 days for primary pneumonic plague, presumably dependent upon the dose of inhaled organisms, onset is acute and often fulminant. The presentation is one of malaise, high fever, chills, headache, myalgia, cough with production of a bloody sputum, and toxemia. A presumptive diagnosis can be made by Gram or Wayson stain of lymph node aspirates, sputum, or CSF. Plague can also be cultured.

**Treatment:** Early administration of antibiotics is very effective. Supportive therapy for pneumonic and septicemic forms is required.

**Prophylaxis:** A licensed, killed vaccine is available. Initial dose followed by a second smaller dose 1-3 months later, and a third 3-6 months later. A booster dose is given at 6, 12 and 18 months and then every 1-2 years. This vaccine may not protect against aerosol exposure.

**Decontamination and Isolation:** Secretion and lesion precautions with bubonic plague. Strict isolation of patients with pneumonic plague. Heat, disinfectants and exposure to sunlight renders bacteria harmless.
Francisella tularensis is a small, aerobic, gram-negative cocco-bacillus, often varying in size and shape. It is non-motile and non-sporulating. Tularemia (also known as rabbit fever and deer fly fever) is a zoonotic disease and humans acquire the disease under natural conditions through inoculation of skin or mucous membranes with blood or tissue fluids of infected animals, or 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 tularemia often having a pneumonic component. The organism 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.

**Signs and Symptoms:** Ulceroglandular tularemia presents with a local ulcer and regional lymphadenopathy, fever, chills, headache and malaise. Typhoidal or septicemic tularemia presents with fever, headache, malaise, substernal discomfort, prostration, weight loss and a non-productive cough.

**Diagnosis:** Clinical diagnosis. Physical findings are usually non-specific. Chest x-ray may reveal a pneumonic process, mediastinal lymphadenopathy or pleural effusion. Routine culture is possible but difficult. The diagnosis can be established retrospectively by serology. After an incubation period varying from 2 to 10 days, presumably dependent upon the dose of organisms, onset is usually acute. Ulceroglandular disease usually manifests as regional lymphadenopathy, fever, chills, headache, and malaise, with or without a cutaneous ulcer. In those 5 to 10 percent of cases with no visible ulcer, the syndrome is known as glandular tularemia. Primary ulceroglandular disease confined to the throat is referred to as pharyngeal tularemia. Oculoglandular tularemia occurs after inoculation of the conjunctivae with infectious material. Typhoidal or septicemic tularemia manifests as fever, prostration, and weight loss, but without adenopathy. Diagnosis of primary typhoidal tularemia is difficult, as signs and symptoms are non-specific and there frequently is no suggestive exposure history. Respiratory symptoms of substernal discomfort, and a non-productive cough may also be present. Radiologic evidence of pneumonia or mediastinal lymphadenopathy may or may not be present in all forms of tularemia but is most common with typhoidal disease. Identification of organisms by staining ulcer fluids or sputum is generally not helpful. Routine culture is difficult, due to unusual growth requirements and/or overgrowth of commensal bacteria. The diagnosis can be established retrospectively by serology.

**Treatment:** Administration of antibiotics (streptomycin or gentamicin) with early treatment is very effective. Prophylaxis: A live, attenuated vaccine is available as an investigational new drug. It is administered once by scarification. A two week course of tetracycline is effective as prophylaxis when given after exposure.

**Decontamination:** Secretion and lesion precautions should be practiced. Strict isolation of patients is not required. Organisms are relatively easy to render harmless by heat and disinfectants.
Q-FEVER
The endemic form of Q fever is a zoonotic disease caused by a rickettsia, Coxiella burnetii. Its natural reservoir is sheep, cattle and goats, in which it grows to especially high concentrations in placental tissues. Exposure to infected animals at parturition is an important risk factor for the endemic disease. It is excreted also 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.

**Signs and Symptoms:** Fever, cough, and pleuritic chest pain 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.

**Diagnosis:** Q fever is not a clinically distinct illness and may resemble a viral illness or other types of atypical pneumonia. The diagnosis is confirmed serologically. Following the usual incubation period of from 10 to 20 days, Q fever generally occurs as a self-limiting febrile illness lasting 2 days to 2 weeks. The incubation period varies according to the numbers of organisms inhaled, with longer periods between exposure and illness with lower numbers of inhaled organisms (up to forty days in some cases). The disease generally presents as an acute non-differentiated febrile illness, with headaches, fatigue, and myalgias as prominent symptoms.

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

**Prophylaxis:** Treatment with tetracycline during the incubation period may delay but not prevent the onset of symptoms. An inactivated whole cell vaccine is effective in eliciting protection against exposure, but severe local reactions to this vaccine may be seen in those who already possess immunity.

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

Viruses are the simplest type of microorganism and consist of a nucleocapsid protein coat containing genetic material, either RNA or DNA. In some cases the virus particle is also surrounded by an outer layer of lipids. Viruses are much smaller than bacteria and vary in size from 0.02 (m to 0.2 (m (1 (m = 1/1000 mm). Viruses lack a system for their own metabolism and are therefore dependent on the synthetic machinery of their host cells: viruses are thus intracellular parasites. This also means that the virus, unlike the bacterium, cannot be cultivated in synthetic nutritive solutions but requires living cells in order to multiply. The host cells can be from human beings, animals, plants, or bacteria. Every virus needs its own special type of host cell because a complicated interaction is required between the cell and virus if the virus is to be able to multiply. Many virus-specific host cells can be cultivated in synthetic nutrient solutions and afterwards can be infected with the virus in question. Another usual way of cultivating viruses is to let them grow on chorioallantoic membranes (from fertilized eggs). The cultivation of viruses is a costly, demanding, and time-consuming process. A virus normally brings about changes in the host cell such that the cell dies. This handbook will cover a virus considered by some to be the most likely viral agent that would be used in a BW attack, the alpha-virus that causes Venezuelan equine encephalitis, known as VEE. The handbook also covers the smallpox viruses and some viruses that cause hemorrhagic manifestations; both could potentially be employed as BW agents.

SMALLPOX

Variola virus is the cause of smallpox. It is an Orthopox virus and occurs in at least two strains, one of which causes variola major, and the other causes a milder disease, variola minor. Despite widespread 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-naive populace. Although the fully-developed cutaneous eruption of smallpox is unique, earlier stages of the rash could be mistaken for varicella. 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.

Signs and Symptoms: The incubation period of smallpox averages 12 days, and contacts are quarantined for a minimum of 16-17 days following exposure. Clinical manifestations begin acutely with malaise, fever, rigors, vomiting, headache, and backache. 2-3 days later lesions appear which quickly progress from macules to papules, and eventually to pustular vesicles. They are more abundant on the extremities and face, and develop synchronously.

Diagnosis: Electron and light microscopy are not capable of discriminating variola from vaccinia, monkeypox or cowpox. The new PCR diagnostic techniques may be more accurate in discriminating between variola and other Orthopoxviruses.

Treatment: At present there is no effective chemotherapy, and treatment of a clinical case remains supportive.

Prophylaxis: Immediate vaccination or revaccination should be undertaken for all personnel exposed. Vaccinia-immune globulin (VIG) is of value in post-exposure prophylaxis of smallpox when given within the first week following exposure.

Isolation: Strict quarantine with respiratory isolation for a minimum of 16-17 days following exposure for all contacts. Patients should be considered infectious until all scabs separate.
VENEZUELAN EQUINE ENCEPHALITIS

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.

**Signs and Symptoms:** VEE is characterized by inflammation of the meninges of the brain and of the brain itself, thus accounting for the predominance of CNS symptoms in the small percentage of infections that develop encephalitis. The disease is usually acute, prostrating and of short duration. Sudden onset of illness with generalized malaise, spiking fevers, rigors, severe headache, photophobia, and myalgias. Nausea, vomiting, cough, sore throat, and diarrhea may follow. Full recovery takes 1-2 weeks.

**Diagnosis:** Clinical diagnosis. Physical findings are usually non-specific. The white blood cell count often shows a striking leukopenia and lymphopenia. Virus isolation may be made from serum, and in some cases throat swab specimens.

**Treatment:** Supportive only.

**Prophylaxis:** A live, attenuated vaccine is available as an investigational new drug. A second, formalin-inactivated, killed vaccine is available for boosting antibody titers in those initially receiving the live vaccine.

**Decontamination:** Blood and body fluid precautions should be practiced. Human cases are infectious for mosquitoes for at least 72 hours. The virus can be destroyed by heat (80 degrees centigrade for 30 minutes) and ordinary disinfectants.
VIRAL HEMORRHAGIC FEVERS

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.

Signs and Symptoms: VHF's 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.

Diagnosis: Definitive diagnosis rests on specific virologic techniques. Significant numbers of military personnel with a hemorrhagic fever syndrome should suggest the diagnosis of a viral hemorrhagic fever.

Treatment: Intensive supportive care may be required. Antiviral therapy with ribavirin may be useful in several of these infections. Convalescent plasma may be effective in Argentine hemorrhagic fever.

Prophylaxis: The only licensed VHF vaccine is yellow fever vaccine. Prophylactic ribavirin may be effective for Lassa fever, Rift Valley fever, CCHF, and possibly HFRS.

Decontamination and Isolation: Decontamination with hypochlorite or phenolic disinfectants. Isolation measures and barrier nursing procedures are indicated.
Toxins are defined as any toxic substance of natural origin produced by an animal, plant, or microbe. They are different from chemical agents such as VX, cyanide, or mustard in that they are not man-made. They are non-volatile, are usually not dermally active (mycotoxins are an exception), and tend to be more toxic per weight than many chemical agents. Their lack of volatility also distinguishes them from many of the chemical threat agents, and is very important in that they would not be either a persistent battlefield threat or be likely to produce secondary or person to person exposures. Many of the toxins, such as low molecular weight toxins and some peptides, are quite stable, where as the stability of the larger protein bacterial toxins is more variable. The bacterial toxins, such as botulinum toxins or shiga toxin, tend to be the most toxic in terms of dose required for lethality, whereas the mycotoxins tend to be among the least toxic compounds, thousands of times less toxic than the botulinum toxins. Some toxins are more toxic by the aerosol route than when delivered orally or parenterally (ricin, saxitoxin, and T2 mycotoxins are examples), whereas botulinum toxins have lower toxicity when delivered by the aerosol route than when ingested. Botulinum is so toxic inherently, however, that this characteristic does not limit its potential as a biological warfare agent. The utility of many toxins as military weapons is potentially limited by their inherent low toxicity (too much toxin would be required), or by the fact that some which are very toxic, such as saxitoxin, can only feasibly be produced in minute quantities. The lower the lethal dose for fifty percent of those exposed (LD50), in micrograms per kilogram, the less agent would be required to cover a large sized area. The converse is also true, and means that for some agents such as ricin, very large quantities (tons) would be needed for an effective open-air attack.

**BOTULINUM TOXINS**

The 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 people. When inhaled, these toxins produce a clinical picture very similar to food borne intoxication, although the time to onset of paralytic symptoms may actually be longer than for food borne 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".

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

**Diagnosis:** The onset of symptoms of inhalation botulism may vary from 24 to 36 hours, to several days following exposure. Botulinum toxins act by binding to the presynaptic nerve terminal at the neuromuscular junction and at cholinergic autonomic sites. These toxins then act to prevent the release of acetylcholine presynaptically, and thus block neurotransmission. This interruption of neurotransmission causes both bulbar palsies and the skeletal muscle weakness seen in clinical botulism.

Unlike nerve agents, where there is in effect too much acetylcholine due to inhibition of acetylcholinesterase, the problem in botulism is lack of the neurotransmitter in the synapse. Thus, pharmacologic measures such as atropine are not helpful in botulism and could even exacerbate symptoms.

**Treatment:** Intubation and ventilatory assistance for respiratory failure. General supportive care. Administration of botulinum antitoxin may decrease progression to respiratory failure and hasten recovery.

**Prophylaxis:** Pentavalent toxoid vaccine (types A, B, C, D, and E) is available as an IND product for those at high risk of exposure.

**Decontamination:** Hypochlorite (0.5% for 10-15 minutes) and/or soap and water. Toxin is not dermally active and secondary aerosols are not a hazard from patients.
STAPHYLOCOCCAL ENTEROTOXIN B

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.

Signs and Symptoms: Relevant exposures to SEB are projected to cause primarily clinical illness and incapacitation. However, higher exposure levels can lead to septic shock and death. Intoxication with SEB begins 3 to 12 hours after inhalation of the toxin. Victims may experience the sudden onset of fever, headache, chills, myalgias, and a nonproductive cough. More severe cases may develop dyspnea and retrosternal chest pain. Nausea, vomiting, and diarrhea will also occur in many patients due to inadvertently swallowed toxin, and fluid losses can be marked. The fever may last up to five days and range from 103 to 106 F, with variable degrees of chills and prostration. The cough may persist up to four weeks, and patients may not be able to return to duty for two weeks.

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

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

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

Decontamination: Hypochlorite (0.5% for 10-15 minutes) and/or soap and water. Destroy any food that may have been contaminated.
RICIN
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 from them. 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 as well by vascular collapse and death. This toxin may also cause disseminated intravascular coagulation, microcirculatory failure and multiple organ failure if given intravenously in laboratory animals.

Signs and Symptoms: 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.

Diagnosis: Signs and symptoms noted above in large numbers of geographically clustered patients could suggest an exposure to aerosolized ricin. The rapid time course to severe symptoms and death would be unusual for infectious agents. Lab findings are nonspecific but similar to other pulmonary irritants which cause PE. Specific serum ELISA is available. Acute and convalescent sera should be collected.

Treatment: Management is supportive and should include treatment for pulmonary edema. Gastric decontamination measures should be used if ingested.

Prophylaxis: There is currently no vaccine or prophylactic antitoxin available for human use, although immunization appears promising in animal models. Use of the protective mask is currently the best protection against inhalation.

Decontamination: Weak hypochlorite solutions and/or soap and water can decontaminate skin surfaces. Ricin is not volatile, so secondary aerosols are generally not a danger to health care providers.

TRICHOTHECENE MYCOTOXINS (T2)
The trichothecene mycotoxins are low molecular weight nonvolatile compounds produced by filamentous fungi (molds) of the genera Fusarium, Myroctecium, Trichoderma, Stachybotrys and others. The structures of approximately 150 trichothecene derivatives have been described in the literature. Heating to 500 F for 30 minutes is required for inactivation, while brief exposure to NaOH destroys toxic activity. The potential for use as a BW toxin was demonstrated to the Russian military shortly after World War II when flour contaminated with species of Fusarium was baked into bread that was ingested by civilians. Some developed a protracted lethal illness called alimentary toxic aleukia (ATA) characterized by initial symptoms of abdominal pain, diarrhea, vomiting, prostration, and within days fever, chills, myalgias and bone marrow depression with granulocytopenia and secondary sepsis. Survival beyond this point allowed the development of painful pharyngeal/laryngeal ulceration and diffuse bleeding into the skin (petechiae and ecchymosis), melena, bloody diarrhea, hematuria, hematemesis, epistaxis and vaginal bleeding. Pancytopenia, and gastrointestinal ulceration and erosion were secondary to the ability of these toxins to profoundly arrest bone marrow and mucosal protein synthesis and cell cycle progression through DNA replication.

Signs and Symptoms: 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.

Diagnosis: Should be suspected if an aerosol attack occurs in the form of "yellow rain" with droplets of yellow fluid contaminating the clothes and the environment.

Treatment: There is no specific antidote. Super activated charcoal should be given orally if swallowed.

Prophylaxis: The only defense is to wear a protective mask and clothing during an attack. No specific immunotherapy or chemotherapy is available for use in the field.

Decontamination: The outer uniform should be removed and exposed skin should be decontaminated with soap and water. Eye exposure should be treated with copious saline irrigation. Once decontamination is complete, isolation is not required.