CHAPTER 23

MEDICAL ASPECTS OF CHEMICAL, BIOLOGICAL, AND RADIOLOGICAL WARFARE

INTRODUCTION

This chapter will outline a brief history of chemical, biological, and radiological (CBR) warfare along with the recognition and treatment of conditions resulting from CBR agents. The chapter is divided into sections by agent type. Each section provides signs and symptoms of CBR conditions along with the treatment and decontamination procedures.

CHEMICAL WARFARE AGENTS

LEARNING OBJECTIVE:
Identify signs and symptoms of chemical agent exposure and provide appropriate medical treatment.

HISTORY

Throughout history, chemical weapons have been used in one form or another. The earliest form of chemical warfare was the use of spears and arrows dipped in poison. The Spartan mixed pitch and sulphur and ignited it to create toxic fumes during battle in order to incapacitate the enemy. Other armies dipped cloth in poison and lit it on fire to create a toxic cloud over opposing armies. These were simple forms of chemical warfare and it was not until recent history that it was used on a large-scale.

The first large-scale use of chemical agents came in World War I when, in 1915, the Germans released chlorine gas against the Allied positions at Ypres, Belgium. Over 5,000 casualties resulted. It is well documented that approximately one-third of all American casualties in this conflict were due to chemical agent attacks.

Chemical warfare during this time period was crude and often personnel were victims of their own chemical attacks, on both sides. During this time the development of gas masks began to protect forces against gas attacks.

During the interval between World Wars I and II, each of the major powers continued to develop its capability for chemical warfare, in spite of a ban by the Geneva Treaty. In isolated cases in the late 1930s, toxic chemicals were used. They were not used during World War II or authorized for use in Korea, Vietnam, or Desert Storm. Defoliants and riot-control agents were used with some degree of effectiveness in the jungles of Vietnam, as well as in tunnel and perimeter-clearing operations.

In recent history there has been documented use of chemical weapons used by other countries and terrorist groups. Iraq used mustard gas during the Iran-Iraq war in 1983. In 1984, Iraq used the nerve agent tabun during the same war. Iraq used chemical weapons in 1987 – 1988 against the Northern Kurds in their own county.

In 1995 a terrorist group in Japan, Aum Shinrikyo, produced and used sarin gas in a Tokyo subway. As a result a dozen people were killed and approximately 5,000 people were incapacitate or injured. The number of dead would have been higher if the agent was in a pure form.

Terrorist groups are adding a new twist to chemical warfare. There have been news reports and admissions by terrorist groups that they are actively developing chemical weapons. The production of chemical weapons on a small scale is not difficult. The space required to set-up a chemical agent lab is no larger than that of a narcotics drug lab (Fig. 23-1). The equipment necessary to produce chemical agents is available on the open market.
OVERVIEW

Chemical weapons are made with toxic chemicals and defined by the Chemical Warfare Convention as “any chemical which through its chemical action on life processes can cause death, temporary incapacitation or permanent harm to humans or animals.” It is also defined as toxic substances developed for the purpose to produce death, serious injury, or incapacitation through their toxicological effects on exposed humans or animals.

Chemical agents can be dispersed by several methods. Attacks can be accomplished with the use of aircraft, munitions, or dispersal devices. Aircraft can deliver a chemical attack by dropping bombs or lunching rockets. Munitions that deliver chemical agents are missiles, rockets, and mortars. Terrorist attacks are more likely to be accomplished using dispersal devices such as commercial sprayers or smoke generators. It is unlikely that an attack against a naval vessel in open water will occur. A naval vessel may more likely be involved in a chemical agent incident while in port.

Chemical agents may enter the body by several routes and the nature and onset of signs and symptoms may vary accordingly. The agents can be disseminated as a vapor or aerosol under ambient conditions. Vapor and aerosol chemical agents often enter the body through the respiratory tract (inhalation injury). The agent may be absorbed by any part of the respiratory tract from the mucosa of the nose and mouth to the alveoli of the lungs.

Vapors and droplets of liquids can be absorbed from the surface of the skin and mucous membranes. Toxic compounds that are harmful to the skin can produce their effects in liquid or solid state. Agents penetrating the skin may form temporary reservoirs under the skin; the vapors of some volatile liquids can penetrate the skin and cause adverse effects. See Table 23-1 for a list of common chemical weapons.

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Common Name</th>
<th>Class</th>
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<tbody>
<tr>
<td>AC</td>
<td>Hydrogen Cyanide</td>
<td>Blood Agent</td>
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<tr>
<td>CK</td>
<td>Cyanogen Chloride</td>
<td>Blood Agent</td>
</tr>
<tr>
<td>CG</td>
<td>Phosgene</td>
<td>Pulmonary Agent</td>
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<tr>
<td>CI</td>
<td>Chlorine</td>
<td>Pulmonary Agent</td>
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<td>CN</td>
<td>Mace</td>
<td>Riot Control</td>
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<tr>
<td>CR</td>
<td>dibenzoxazepine</td>
<td>Riot Control</td>
</tr>
<tr>
<td>CS</td>
<td>2-chlorobenzalmalonitrile</td>
<td>Riot Control</td>
</tr>
<tr>
<td>CX</td>
<td>Phosgene Oxime</td>
<td>Blister Agent</td>
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<td>DP</td>
<td>Diphosgene</td>
<td>Pulmonary Agent</td>
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<td>DM</td>
<td>Adamsite</td>
<td>Riot Control</td>
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<td>GA</td>
<td>Tabun</td>
<td>Nerve Agent</td>
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<td>GB</td>
<td>Sarin</td>
<td>Nerve Agent</td>
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<tr>
<td>GD</td>
<td>Soman</td>
<td>Nerve Agent</td>
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<tr>
<td>GF</td>
<td>cyclosarin</td>
<td>Nerve Agent</td>
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<tr>
<td>H</td>
<td>Mustard</td>
<td>Blister Agent</td>
</tr>
<tr>
<td>HD</td>
<td>Distilled Mustard</td>
<td>Blister Agent</td>
</tr>
<tr>
<td>HN</td>
<td>Nitrogen Mustard</td>
<td>Blister Agent</td>
</tr>
<tr>
<td>L</td>
<td>Lewisite</td>
<td>Blister Agent</td>
</tr>
<tr>
<td>OC</td>
<td>Oleoresin Capsicum</td>
<td>Riot Control</td>
</tr>
<tr>
<td>VX</td>
<td>S-2-(diisopropylamino)ethyl O-ethyl methylphosphonothioate</td>
<td>Nerve Agent</td>
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Table 23-1.—Names, Classes, and Symbols of Chemical Weapons
Chemical agents can be broken down into general classes of agents. The following are the classes of chemical agents that will be covered in this section: Blood Agents; Pulmonary Agents; Blister Agents (Vesicants); Nerve Agents; and Riot Control Agents. They may also be classified as either lethal or nonlethal.

- **Nonlethal** agents that are not designed to kill you

- **Lethal** agents are those that result in a 10 percent or greater death rate among casualties

Chemical agents are further classified as persistent or non-persistent, dependent upon the length of time they retain their effectiveness after dissemination.

- **Persistent** agents continue to present a hazard for considerable periods (days) after delivery by remaining as a contact hazard, or by slowly vaporizing to produce a hazard an inhalation hazard

- **Non-persistent** agents disperse rapidly after release and present an immediate, short duration (hours) hazard. They are released as airborne particles, aerosols, and vapors

Metrological conditions will influence the effectiveness and duration of chemical agents. Wind, temperature, and rain are major considerations when chemical agents are used as they will impact the length and intensity of exposure.

- Wind in an open area will disperse an agent quickly. Calm winds or protected areas (wooded areas, trenches, ditches, and urban areas) will allow an agent to stay in an area longer

- High temperatures decrease the persistency of agents and tend to cause higher vapor concentrations. This is especially true with the use of a Mustard agent

- Low temperatures increase the persistency of agents. Some agents may freeze, thus reducing the immediate contact hazard or vapor hazard. There is a danger of moving frozen agents, on clothing and equipment, into a warm building; when they warm-up there is a subsequent risk of toxic vapor being given off

- Rain washes away, dilutes, and promotes hydrolysis of agents. This reduces their effectiveness but does not make them harmless

**DETECTION EQUIPMENT**

There are several ways to detect the presence of chemical agents. Some detection methods are as simple as chemical reactive paper and as complex as electronic detection devices. Medical personnel should be familiar with three of the common detection methods.

The first method is M9 Chemical Agent Detector Paper. It is the most widely used method of detecting liquid chemical warfare agents. M9 paper indicates the presence of a nerve agent or a blister agent by turning a pink, red, reddish brown, purple color. It does not identify which agent gives the positive reading. The M-9 paper is self-adhesive and attaches to most surfaces.

The second method is the M8 Chemical Agent Detector Paper. It is used to test for the presence of liquid chemical agents. It can detect the presence of particular agents. When the paper touches a liquid agent, the paper will change color. The paper will turn Gold/Yellow for G class nerve agents and turns Olive or Verdana Green for VX. The paper turns red or purple when it comes in contact with blister agents.

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**NOTE:**
Neither M8 nor M9 paper can detect chemical warfare agent vapor.
The M256A1 chemical agent detector kit is a portable kit that detects nerve gas, mustard gas, and cyanide. The kit contains a package of M8 paper, detailed instructions, and a vapor sampler (12 enzymatic tickets that contain laboratory filter paper for detecting chemical agent vapors). The vapor sampler uses wet chemistry technology, in which ampules containing different substrates are crushed so that the liquids interact with strips of filter paper, chromatographic media, and glass fiber filter. These substrates are exposed to the vapor under suspicion. The reaction causes a color change, alerting the user to the presence of a chemical agent. The reactions typically take 15 minutes to occur.

PERSONAL PROTECTION

In a chemical attack, the first priority is to ensure the HM’s survival so that casualties can be treated. There are several items available to help HMs survive a chemical attack. Along with protective clothing, there is a protective mask, which should be put on at the first indication of a chemical attack. The mask will filter out all known chemical agents from the air and allow HMs to work in a chemically contaminated area.

If there is a known threat of possible chemical, biological, or radiological attack or personal need to enter known contaminated area, protective measures should be taken. Personal Protective Equipment (PPE) consists of the Joint Service Lightweight Integrated Suit Technology (JSLIST), Field M-40 Chemical/Biological Mask with hood, protective gloves, and protective boots.

Dependent upon the threat, forces may adopt a Mission-Oriented Protective Posture (MOPP) and there are five levels (Table 23-2). MOPP Gear consists of previously mentioned PPE to include an individual decontamination kit as well as antidotes. MOPP is a flexible system of protection against chemical, biological and radiological threats, which is used to facilitate mission accomplishment. MOPP does give the commander a range of choices regarding the level of chemical protection. Choices range from no protection at all to full protection.

<table>
<thead>
<tr>
<th>Mission-Oriented Protective Postures (MOPP)</th>
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<tbody>
<tr>
<td>MOPP Level</td>
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<tr>
<td>0</td>
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<tr>
<td>1</td>
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<td>2</td>
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<td>3</td>
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</table>

This chart provides a quick reference of what equipment needs to be worn

* MOPP Level 0 – Protective equipment should be within easy reach.

Table 23-2.—Mission-Oriented Protective Procedures (MOPP)
Chemical agents penetrate ordinary clothing rapidly. However, significant absorption through the skin requires a period of minutes. The effects of clothing penetration may be reduced by quickly removing the contaminated clothing and neutralizing the chemical agent on the skin by washing, blotting, or wiping it away. A chemical agent on the skin can be removed effectively by using the M291 skin decontamination kit (Fig. 23-2) or decontamination procedures associated with each agent.

![Image of M291 Skin Decontamination Kit](Image provided by: NAVSEA Damage Control, Fire Protection Engineering and CBR-D.)

Prompt decontamination of the skin is imperative. Decontamination of chemical agents on the skin within 1 minute after contamination is perhaps 10 times more effective than if decontamination is delayed 5 minutes. Quick decontamination procedures are associated with each agent. Detailed instructions on the use of skin decontamination kits can be found in the NAVMED P-5041, Treatment of Chemical Agent Casualties and Conventional Military Chemical Injuries, and in the kits themselves.

## CHEMICAL AGENTS

### NERVE AGENTS (VX, GA, GB, GD, GF)

Nerve agents are of greatest concern as compared to all chemical agents. They produce their effect by interfering with normal transmission of nerve impulses in the parasympathetic autonomic nervous system. Pharmacologically, the nerve agents are cholinesterase inhibitors (interfering with normal transmission of nerve impulses in the nervous system). Their reaction with cholinesterase tends to be irreversible, and reaction time varies with the agent.

### Characteristics

Physically, nerve agents are odorless, almost colorless liquids or vapors, varying greatly in viscosity and volatility. They are moderately soluble in water and fairly stable unless strong alkali or chlorinating compounds are added. They are very effective solvents, readily penetrating cloth either as a liquid or vapor. Other materials, including leather and wood, are fairly well penetrated. Butyl rubber and synthetics, such as polyesters, are much more resistant.

### Signs and Symptoms

Nerve agents can enter the body through the eyes, respiratory tract, and skin. Symptoms vary dependent upon the patient being exposed to either vapor of liquid forms of a nerve agent. The onset of symptoms range from within seconds to 18 hours, dependent upon the form and amount of agent.

- **Vapor**
  - Small Exposure Level – Miosis (constricted pupils), rhinorrhea (runny nose), and mild difficulty breathing
  - Large Exposure Level – Miosis; sudden loss of consciousness; convulsions; apnea (no breathing); flaccid paralysis; copious secretions from the nose, mouth, and lungs
- **Liquid**
  - Small to Moderate Exposure – Localized sweating, nausea, vomiting, and weakness
  - Large Exposure Level - Sudden loss of consciousness; convulsions; apnea (no breathing); flaccid paralysis; and copious secretions from the nose, mouth, and lungs

**Treatment**

Immediate care and administration of an antidote can mean the difference between life and death of a patient exposed to a nerve agent. Atropine, an acetylcholine blocker, is the drug of choice for treating nerve agent poisoning. The second drug used for nerve agent poisoning is Pralidoxime Chloride (2-PAM CL). 2-PAM CL removes the nerve agent from the enzyme acetylcholinesterase within the synaptic cleft (the space between the nerve cells) of the nervous system. Convulsive Antidote, Nerve Agent (CANA) or Diazepam 10mg autoinjector is used to control convulsions in patients (Fig. 23-3).

The MARK 1 antidote kit (Fig. 23-4) consists of an autoinjector of Atropine 2mg and an autoinjector of 2-PAM CL 600mg. A new autoinjector called Autoinjector Treatment; Nerve Agent Antidote (ATNAA) will replace the MARK 1 kit. The ATNAA is a single autoinjector that has two chambers that deliver 2.1 mg of Atropine and 600mg of 2-PAM CL in a single injection. ATNAA is used in the same manner as a MARK 1 kit.

**How to use an autoinjector**

Firm pressure automatically triggers the coiled mechanism and plunges the needle through the clothing into the muscle and at the same time injects the antidote into the muscle tissue. Using a jabbing motion may result in an improper injection or injury.

**Self-Aid/Buddy Aid**

1. At the first signs of nerve-agent poisoning, don protective mask.
2. Administer one MARK 1 kit intramuscularly, into the lateral thigh muscle or buttocks of the patient.
3. Position the needle end of the injector against the injection site.
4. Make sure not to hit any buttons or other objects.
5. Apply firm and even pressure to the autoinjector until the needle pushes into the injection site.
6. Hold the injector firmly in place for at least 10 seconds.

**NOTE:**
Do **NOT** use a jabbing motion. This may result in improper injection of antidote and/or injury to the patient.

7. Wait for 10 to 15 minutes to see if the symptoms subside.
8. If symptoms continue, administer another autoinjector. Note - a total of three MARK 1 kits can be administered at 10 to 15 minute intervals by non-medical personnel.
9. Patients with severe symptoms, more than one system involvement (i.e. gastrointestinal and respiration) should be given all three MARK 1 kits and CANA immediately.

**NOTE:**
The HM will use the casualty’s autoinjector(s) when providing aid to the casualty. The HM must never use their autoinjector(s) on the casualty as this will limit the antidote available if needed for self-aid.

**Medical Personnel**

If symptoms continue after three autoinjectors have been administered, medical personnel may administer repeated Atropine (2mg) injections. Atropine can be injected at five to ten minute intervals and until there is a reduction of both secretions and breathing difficulty.

If severe symptoms still persist after one hour of giving the three MARK 1 kits (Atropine and 2-PAM CL), three additional autoinjectors of 2 PAM CL 600mg should be given. **No more than six doses should be given of 2-PAM CL.** Discontinue 2 PAM CL use after respiratory distress has decreased.

If convulsions continue after 10 minutes of initial injection of CANA (Diazepam), a second dose may be administered. Observe patient for 5 to 10 minutes after injection, if the patient is still convulsing a third dose may be administered. Medical officers may choose to give more diazepam either IM or IV, if they deem it necessary.

**Decontamination**

Decontamination of the patient should be done as soon as possible. This will eliminate the potential of continued absorption of never agents into the patient. Conduct decontamination in the following order:

- Face
- Neck Area
- Chest Area
- Abdomen
- Arms and Hands
- Other exposed skin areas

Decontamination of liquid nerve agent exposure consists of removing all contaminated clothing. A M291 kit may be used or copiously irrigating the area with water to physically remove the nerve agent. The skin is then washed with an alkaline solution of soap and water or 0.5% hypochlorite solution (made by diluting household bleach 1:10) to chemically neutralize the nerve agent. Avoid hot water, strong detergents, and vigorous scrubbing, since they tend to enhance nerve agent absorption.

**BLISTER AGENTS (H, HD, HN, L)**

Blister agents, or vesicants, exert their primary action on the skin, producing large and painful blisters that are incapacitating. Although vesicants are classed as nonlethal, high doses can cause death. Mustards (H, HD, and HN) constituted both a liquid and vapor threat. Mustard agents are a major concern due to large stockpiles it and the ease of production.
Characteristics

Each agent is chemically different and will cause significant specific symptoms. They are all similar in their physical characteristics and toxicology. H, HD, and HN are oily, colorless or pale yellow liquids, sparingly soluble in water. HN (Nitrogen Mustard) is less volatile and more persistent than HD (Distilled Mustard) but has the same blistering qualities. Lewisite (L) is an arsenical (an arsenic-based compound). This blistering compound is a light to dark brown liquid that vaporizes slowly. All blister agents have a relatively high vapor density; it is more likely to flow to low spots such as valleys, ditches, holes, and the ground or deck.

Signs and Symptoms

Mustards (Fig. 23-5) are particularly insidious as they do not manifest their symptoms for several hours after exposure. Blister agents attack the eyes and respiratory tract as well as the skin. Patients exposed to mustard may remember seeing an oily substance and smelling an odor of garlic, mustard, or horseradish. Patients exposed to Lewisite (L) may remember observing puddles of a brown liquid or of smelling an odor similar to geranium.

The eyes are the most vulnerable part of the body to mustard gas. Contamination insufficient to cause injury elsewhere may produce eye inflammation. The eyes are the most sensitive part of the body. The first noticeable symptoms of mustard exposure will be pain and a gritting feeling in the eyes, accompanied by spastic blinking of the eyelids and photophobia. This may continue to develop with swelling of the eyelids, cornea damage, and moderate to severe pain.

The skin will develop erythema and blisters (Fig. 23-6). Typical blister agent cause blistering in about 12 hours but may be delayed for up to 48 hours, while Lewisite (L) causes intense pain upon contact. Areas affected the most will be in warm, sweaty areas of the body: the armpits, groin, and on the face and neck.
Inhalation of the gas is followed in a few hours by sore throat, sinus pain, and hoarseness. This may progress to a hacking cough and then to a productive cough and shortness of breath. Breath sounds may be crackles and rales.  Bronchopneumonia is a frequent complication. The primary cause of death is massive edema or mechanical pulmonary obstruction. Due to the pain associated with Lewisite (L) exposure, patients are more likely to don their protective mask early. Thus limiting the respiratory injuries that may normally occur as a result of exposure.

**Treatment**

There is no specific antidotal treatment for mustard (H, HD, and HN) poisoning. Physically removing as much of the mustard as possible as soon as possible, is the only effective method for mitigating symptoms before they appear. All other treatment is symptomatic and supportive; the relief of pain and itching, and control of infection.

In cases of systemic involvement, British

**Figure 23-5.—Mustard Agent**

*Image reprinted with permission from: U. S. Army Special Programs Division, Dugway Proving Grounds.*

Anti-Lewisite (BAL) was developed as an antidote for Lewisite (L). BAL is used as a chelating agent that combines with the heavy metal to form a water-soluble, nontoxic complex that is excreted. However, BAL is somewhat toxic and an injection of more than 3 mg/kg will cause severe symptoms. Do not use on patients allergic to peanuts.

**Decontamination**

Early decontamination will reduce the affect of blister agents. Decontamination within two minutes will reduce the toxic effects by more then 50%. Decontamination consists of removing all contaminated clothing. A M291 kit may be used or copiously irrigating the area with water to physically remove agents and then washing the skin with soap and water or 0.5% hypochlorite solution (made by diluting household bleach 1:10) to chemically neutralize the agent.

**BLOOD AGENTS (AC, CK)**

Blood agents or cyanides basic physical actions disrupt oxygen utilization at the cellular level causing cellular suffocation. They are rapid acting lethal agents that have limited military use. Cyanide is a common chemical and found widespread in chemical synthesis and is easy for terrorist to obtain and to potentially be used in a terrorist attack. Characteristics
Cyanides are volatile and evaporate quickly to become vapors or gases. Hydrogen Cyanide (AC) has a bitter almonds smell. Although very deadly, they are non-persistent agents. Cyanides usually dissipate in less than 24 hours. Cyanide produces clinical effects by disrupting oxygen uptake by cells.

**Signs and Symptoms**

- **Moderate Exposure** (low concentrations)
  - Symptoms include transient increased rate and depth of breathing; dizziness; nausea and vomiting; headache; and eye irritation. Symptoms may progress to severe with continued exposure

- **High Exposure** (high concentrations)
  - The onset is rapid, often within minutes. Symptoms include transient increased rate and depth of breathing; convulsions (within 30 seconds); apnea; cardiac arrest (within a few minutes)

**Treatment**

Treatment begins with personnel protection by using a chemical protective mask. Remove the patient from the agent to fresh air. Treatment of cyanide poisoning is very effective if administered in a timely manner. Antidote treatment consists of a two step process:

1. Initial treatment of cyanides: two amyl nitrite ampules crushed and inhaled (every few minutes until eight ampules have been used) or intravenous sodium nitrate 300mg (a 300mg to 600mg dose given).
2. Administer intravenous sodium thiosulfate 12.5g (1 to 2 doses given).
3. Follow-up treatment consists of the two antidotes given at half the original dose if there is no response to the first dose.

The key to successful cyanide therapy is providing treatment early; cyanide acts rapidly on an essential enzyme system. The antidotes act rapidly to reverse this action.

If the specific antidote and artificial respiration are given early enough, the chance of survival is greatly enhanced.

**Decontamination**

Skin decontamination is usually not required as the agent evaporates quickly. Wet contaminate clothing should be removed and contained to prevent off gassing hazard. Skin should be cleaned by copiously irrigating the area with water to physically remove agents, and then washing the skin with soap and water.

**PULMONARY AGENTS (CG, CL, DP)**

Pulmonary agents damage the membranes in the lungs that separate the alveolar tissue resulting in fluid from the blood, known as plasma, to leak into the alveoli and fill them with fluid. This prevents necessary gas exchange within the alveoli causing hypoxia. This creates a condition known as pulmonary edema. This group includes phosgene (CG) and chlorine (Cl); HC Smoke and Ammonia should also be included as a pulmonary agent. A terrorist threat may come from the release of chlorine or ammonia.

**Characteristics**

These agents are usually in vapor form, typically heavier than air, and travel close to the ground. They tend to evaporate and disburse very quickly, dependent upon temperature and wind. Chlorine and ammonia have very distinct smells. Phosgene is a colorless gas with a distinctive odor similar to that of new-mown hay or freshly cut grass.

**Signs and Symptoms**

Early symptoms may be irritation of the eyes, nose, and airway. At this stage it may be difficult to distinguish a pulmonary agent from a riot agent. Symptoms may progress to coughing, difficulty breathing, hoarseness talking, sneezing, wheezing, and a feeling of tightness in the chest. More often, however, there will be no symptoms for 2 to 6 hours after exposure.
Latent symptoms are rapid, shallow, and labored breathing; painful cough; cyanosis; frothy sputum; clammy skin; rapid, feeble pulse; and low blood pressure. Shock may develop, followed by death. Auscultation of the lungs will reveal crackles and rales, in the lower lobes initially and progress to be heard throughout all fields.

Treatment

Initial treatment is to remove the patient from the source. There is no antidote for pulmonary agents. Keep the patient at complete rest. Even a little exertion can increase the effects of the agent and speed up the progression of pulmonary edema. Provide supportive care as necessary and treat symptomatically. Patients with shortness of breath may require assisted respirations and/or oxygen.

Decontamination

- **Vapors**
  - Exposure to fresh air or ventilate the area

- **Liquids**
  - Remove contaminated clothing and rinse the affected area with copious amounts of water

**RIOT-CONTROL/HARASSMENT AGENTS (CN, CR, CS, DM, OC)**

"Riot-control agents" is the collective term used to describe a collection of chemical compounds, all having similar characteristics which, though relatively nontoxic, produce an immediate but temporary effect in very low concentrations. These agents are used to harass enemy personnel or to discourage riot actions thus the weapon of choice for police when managing riots.
Characteristics

Unlike most agents, which are liquids under temperate conditions, riot control agents are crystallized solids that are dispersed as fine particles or in solution(s). Dispersal devices include small handheld aerosol cans, large tanks, grenades, and bombs. These agents irritate the skin, mucosa membranes, and airway, causing people to become unable to perform their job due to discomfort. There are two classes of riot-control/harassment agents: lacrimators and vomiting agents.

- **Lacrimators** (or tear gases) are essentially local irritants that act primarily on the eyes.
- **Vomiting agents** comprise the second class of agents in the riot-control category and cause nausea, vomiting, and general malaise in victims.

Signs and Symptoms

The main effect of riot control agents is pain, burning, and irritation to exposed skin and mucosa membranes. Other symptoms include salivation, increased nasal secretions, coughing, possible redness of skin, and possible shortness of breath. Lacrimators produce intense pain in the eyes with excessive tearing. Vomiting agents produce prolong periods of nausea and vomiting. The symptoms following the most severe exposure to vapors seldom last over 2 hours. After moderate exposure, they last only a few minutes.

Treatment

At the first signs of exposure, don protective mask. It is of the utmost importance that the mask be worn in spite of coughing, sneezing, salivation, and nausea. If the mask is put on following exposure, symptoms will increase for several minutes in spite of adequate protection. As a consequence, victims may believe the mask is ineffective and remove it, further exposing themselves.

While the mask must be worn, it may be lifted from the face briefly, if necessary, to permit vomiting or to drain saliva from the face piece. Carry on duties as vigorously as possible. This will help to lessen and shorten the symptoms.

Generally, patients require no therapy; removal from the environment is sufficient to affect recovery in a short time. Exposure to fresh air and letting wind blow into wide-open eyes, held open if necessary, is sufficient for recovery in a short time. Talking can relieve any chest discomfort after CS exposure. Less than 1% of people develop severe symptoms. There is no antidote for these agents. Patients need to be treated according to their symptoms.

- **Eyes:** The eyes should be flushed with water or saline and impacted particles should be removed. General care consists of a topical solution to relieve the irritation and topical antibiotics. An ophthalmologist should be consulted for further evaluation and care.
- **Pulmonary:** These agents may exacerbate chronic disease or unmask latent disease. Bronchospasm with wheezing and mild distress continuing hours after exposure may occur in latent asthmatic people. More severe effects and respiratory distress may occur in one with chronic bronchitis or emphysema. Management includes oxygen administration and bronchodilators if bronchospasms are present.
- **Skin:** The early erythema requires reassurance, but no specific therapy is indicated unless severe and prolonged more than an hour or two. Treat symptoms with soothing compounds such as calamine. Small vesicles should be left intact, but larger ones will ultimately break. Large, oozing areas have responded to compresses containing substances such as colloidal oatmeal, Burrow's solution, and other dermatologic preparations.
Decontamination

An important point to remember is that this material adheres to clothing, and a change of clothing may be necessary. Do not forget the hair (both head and facial) as a potential source of recontamination. The crystals can be released from the hair, skin, and clothes by a fan, wind, or the patient flapping their arms and rubbing hair. Heavily exposed patients can be decontaminated by washing with soap and water. Areas can be rinsed with a continuous flow of water. OC can be removed by washing with baby shampoo, milk, or vegetable oil to break up the resin and neutralize the agent.

DECONTAMINATION

The guiding principle in personnel decontamination is to avoid spreading contamination to clean areas and to manage casualties without aggravating other injuries (Fig. 23-7). It can be accomplished by either removing or neutralizing the agent. The process can be very extensive, dependent upon the agent or materials that need to be removed.

Figure 23-7.—Decontamination

Image reprinted with permission from: U. S. Army Special Programs Division, Dugway Proving Grounds.

Casualty Priorities

It may be necessary to decide whether to handle the surgical condition or the chemical hazard first. If the situation and the condition of the casualty permit, decontamination should be carried out first. The longer the chemical remains on the body, the greater the danger of spreading the chemical to other personnel and equipment.

The following order of priority for first aid and decontaminating casualties is recommended:

- Control of massive hemorrhage
- First aid for life-threatening shock and wounds
- Decontamination of exposed skin and eyes
- Removal of contaminated clothing and decontamination of body surfaces (if not in a toxic environment)
- Adjustment of the patient’s mask, if mask is necessary
- First aid for less severe shock and wounds

Decontamination Station Organization

In general, the decontamination station, or "dirty" area, receives casualties contaminated with a chemical agent. The arrangement of this area will vary with the site of the medical unit and the facilities available for decontamination. See Figure 23-8 for one decontamination site organization.
Each ship has a minimum of at least two decontamination stations, insofar as the hull design permits. The "dirty" areas should be topside or in some well-ventilated space. Personnel manning these areas should be provided with protective equipment.

In the "dirty" area, casualties will be decontaminated, undressed, showered, and passed along to clean areas. Both areas will be clearly marked as either "clean" or "contaminated," as appropriate. Decontamination kits, protective ointment, and an abundant supply of soap and water must be provided. In addition, standard first-aid items should be on hand. When possible, improvise supports (e.g., small boxes, blocks of wood, etc.) for stretchers to keep them raised off the deck.

**Handling of Contaminated Casualties**

The spread of contamination to uncontaminated personnel or to spaces not set aside to receive contamination must be avoided. Contaminated personnel, clothing, or equipment must be kept out of uncontaminated areas as the subsequent decontamination of such spaces is quite difficult. Contaminated clothing and gear must be placed in designated dump areas and, whenever practically possible, kept in metal cans with tightly fitting covers.

All casualties, after experiencing a chemical attack are to be considered contaminated unless there is certification of non-contamination. The initial management of a casualty contaminated by chemical agents will require removal of MOPP and decontamination with 0.5% hypochlorite before treatment.
LEARNING OBJECTIVE:
Identify signs and symptoms of a biological agent exposure and provide appropriate medical treatment.

HISTORY

The use of biological warfare has been recorded throughout history. Some of the early known uses of biological agents range from using a biological agent in drinking wells to moving driving infected animals into cities in hopes of spreading disease. In 1346, plague broke out in the tartar army during the siege of Kaffa. The attackers hurled the corpses of plague victims over the city walls. This caused an epidemic within the city and forced the defenders to surrender. It is believed that some of the infected people from the siege may have started the Black Death pandemic. The pandemic spread through Europe and is responsible for the death of one third (around 25 million people) of the population of Europe.

Biological Warfare has transformed over the years. It went from a crude form of spreading disease for a military advantage to a complex state sponsored program. In 1937, Japan started a sophisticated biological weapons program. The project was code-named “Unit 731” and was located 40 miles south of Harbin, Manchuria. The unit continued its work until it was destroyed in 1945. An investigation into the unit revealed that the Japanese researched numerous organisms and used war prisoners as research subjects.

It is estimated that about 1,000 human autopsies were carried out at Unit 731 and most of the victims were exposed to anthrax. By 1945 the program had stockpiled 400 kilograms of anthrax to be used in a specially designed fragmentation bomb.

After reports of flights of Japanese planes suspected of dropping plague-infected fleas, plague epidemics broke-out in China and Manchuria. It was evident the Japanese used biological agents on civilian populations.

There has been shift in the use of Biological Weapons from a sophisticated biological program run by other countries to agents used by an individual or terrorist group (Fig. 23-9). The use of Anthrax in letters used in 2001 was believed to have been done by an individual. A domestic terrorist group, Rajnees, used Salmonella bacteria to contaminate salad bars, in Dalles, Oregon, in order to try and influence the local county election results in 1984.

Figure 23-9.—Small-Scale Biological Fermentor

Image reprinted with permission from: U. S. Army Special Programs Division, Dugway Proving Grounds.

Bioterrorism is a major concern for cities and states around the country. It is also a military concern today. One infected Sailor onboard a ship can infect the crew. The ship would have to be quarantined and would become combat ineffective.

The use of biological agents by terrorist groups is not outside the realm of reality. A terrorist plot was uncovered in England to use ricin in January 2003. Ricin was found in a package in a South Carolina postal facility in October 2003. It is not difficult to produce a biological agent.
The process can be done in a kitchen or bathroom within a house. A terrorist group can produce the agent and release it before anyone knows what happens.

**TYPES OF BIOLOGICAL AGENTS**

There are three different types of Biological Agents used as weapons:

**Bacteria**

Single celled organisms capable of causing a variety of diseases in animals, plants, and humans. Bacteria are living cells that carry many complex metabolic functions. Bacteria cause disease in human beings by one of two mechanisms: invading host tissues and producing toxins. Many bacteria utilize both mechanisms. Bacterial Agents are easy to produce in significant quantities to cause a threat to the community’s health (Fig. 23-10). Fortunately, the infections they produce often respond to a specific antibiotic therapy.

**Viruses**

Microorganisms are smaller than bacteria. Viruses are intracellular parasites that lack a system for their own metabolism, meaning, they require living cells in order to multiply. Viruses can infect host cells from humans, animals, plants, and bacteria. Virus-specific host cells can be cultivated in synthetic nutrient solutions and then infected with the virus to be grown. A virus typically causes changes in the host cell that eventually leads to cell death. The best way to control viral infections is to prevent them from occurring through the use of vaccines. Viral Agents can be more expensive and time consuming to produce when compared to bacterial and toxin agents.

**Toxins**

Harmful substances produced by a variety of living organisms, like bacteria, plants, and animals (Table 23-3). They are not man-made, they are non-volatile (i.e. no vapor hazard), and are usually not dermally active (with exception to the mycotoxins). Biological toxins are generally more toxic per weight than the chemical agents and represent some of the most toxic substances on Earth. Their lack of volatility is important to note because it makes them unlikely to produce either a secondary or person-to-person exposure. They will not present a persistent environmental hazard.

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Virus</th>
<th>Toxin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anthrax</td>
<td>Small Pox</td>
<td>Ricin</td>
</tr>
<tr>
<td>Plague</td>
<td>Hemorrhagic</td>
<td>Botulinum</td>
</tr>
<tr>
<td>Tularemia</td>
<td>Fevers</td>
<td>Toxin</td>
</tr>
</tbody>
</table>

Under special circumstances some types of bacteria can form spores (e.g. the genus *Bacillus*). Spores are a dormant form of the bacterium and can germinate when conditions are optimal. When compared to the vegetative bacterial cell, the spore is more resistant to cold, heat, drying, chemicals, and radiation.

![Figure 23-10.—Homemade Fermentor](Image reprinted with permission from: U. S. Army Special Programs Division, Dugway Proving Grounds.)

Table 23-3.—Type of Biological Agents and Examples of Each Type
NATURAL OR INTENTIONAL DISEASE OUTBREAK

It is difficult to determine if a disease outbreak is a result of a natural occurrence or an intentional biological attack. An epidemiological investigation of a disease outbreak will assist medical personnel in identifying the pathogen and use of proper medical intervention. This should be done whether the outbreak is an intentional release or a natural occurrence.

Some attacks may not be apparent and require a full epidemiological study. Small scale outbreaks or those occurring over a long period of time require further investigation. Outbreaks occurring in multiple geographical locations may be either a natural occurrence or an intentional release. An outbreak of a disease that is difficult to diagnose or that occurs naturally or with low mortality rates requires further studies to determine the cause of the outbreak.

There are incidents that do not require an epidemiological study to determine that an attack or intentional release has occurred. Diseases that have been eradicated, such as smallpox, is a good indication of intentional release. A disease with a low probability of occurrence, such as inhalational anthrax, is another good indicator. The final indicator would be a large scale outbreak occurring in a limited geographical area.

Some additional indicators of a possible biological agent release:
- Unusual disease for geographic area
- Absence of a competent natural vector
- Restricted geographic distribution, epidemiological grouping or clustering
- High morbidity and mortality compared with a normal occurrence of the disease
- Dead animals of multiple species are sentinels

With recent advances in diagnostic testing, biological agents can be detected in the field. A first-line presumptive test is the Hand-Held Assay Panel that can make an indication of the presence of several biological agents within 15 minutes. Portable laboratories have been developed that can provide field confirmatory diagnostics for several biological agents within an hour. These labs weigh less than 1,000 pounds and require three operators to run it. These labs utilize a rapid field detection method incorporating real-time Polymerase Chain Reaction (rt-PCR) based diagnostics for field confirmatory testing (Fig. 23-11). These confirmatory assays are based on the unique DNA sequences of potential biological threat agents. The system is excellent for detecting the presences of the DNA unique to pathogenic bacteria and viruses. These laboratory tests are extremely accurate.

PERSONAL PROTECTION

If a suspected biological agent is released in the field, personnel should don personnel protective equipment (PPE). This consists of the Joint Service Lightweight Integrated Suit Technology (JSLIST), Field M-40 Chemical - Biological Mask, protective gloves, and boots. Personnel should utilize this equipment in an
unknown biological environment and when conducting decontamination procedures

**BIOLOGICAL AGENTS**

**ANTHRAX (BACILLUS ANTHRACIS)**

Anthrax (*Bacillus anthracis*) is a disease caused by the bacterium *Bacillus anthracis*. It is a gram-positive, encapsulated, spore-forming, non-motile rod. The bacterium forms spores and these spores are usually the infective form of the bacteria. The *Bacillus* spores can survive in the environment for years or decades, awaiting uptake by the next host. It is primarily a disease of herbivorous mammals, although other mammals and some birds have been known to contract it. As a biological threat it can be produced with little cost from readily available equipment. An intentional release was implemented following the 9/11 attacks via the mail system; the precedent has been set for future terrorist attacks.

**Forms of Disease and Infectious Route**

Transmission of the disease is through contact with contaminated material. Humans generally acquire the disease directly or indirectly from infected animals or occupational exposure to infected or contaminated animal products. Person-to-person transmission does not occur. Articles and soil contaminated with spores may remain infective for several years. There are three types of anthrax in humans:

- Cutaneous anthrax; acquired when a spore enters the skin through a cut or an abrasion
- Pulmonary (inhalational) anthrax; from breathing in airborne anthrax spores
- Gastrointestinal anthrax; contracted from eating contaminated food, primarily meat from an animal that died of the disease

**Incubation Period**

The average is from 1 to 7 days, although incubation periods of up to 60 days can be possible.

**Signs and Symptoms**

- **Cutaneous Anthrax**: occurs within 2 weeks after exposure to spores
  - Skin infection begins as a raised itchy bump that resembles an insect bite, but within 1-2 days develops into a vesicle and then a painless ulcer, usually 1-3 cm in diameter, with a characteristic black necrotic (dying) area in the center
  - Regional adenopathy, fever, malaise, headache, nausea and vomiting may be present
  - About 20% of untreated cases of cutaneous anthrax will result in death
  - Deaths are rare with appropriate antimicrobial therapy
- **Inhalational Anthrax**: Onset, which occurs in two stages, usually begins within 10 days
  - Initial signs and symptoms may resemble nonspecific viral-like symptoms - fever, malaise, headache, sore throat, dyspnea (labored breathing), cough, and congestion of the nose, throat, and larynx are characteristic of the initial stage
  - The chest x-ray is the most sensitive test for inhalational anthrax
  - Mediastinal widening due to hemorrhagic lymphadenitis, a hallmark feature of the disease has been present in 70% of the bioterrorism related cases
  - Pleural effusions were present initially or occurred over the course of illness in all cases
  - These symptoms last generally 2-5 days and can be followed by a short period of improvement
  - The symptoms may progress to anterior chest pain, severe respiratory distress with dyspnea, diaphoresis (sweating), stridor, cyanosis, and shock
  - Inhalation anthrax is usually fatal
• **Gastrointestinal anthrax:** The intestinal disease form of anthrax may occur 2 - 5 days following the consumption of contaminated meat
  - Signs and symptoms include fever, diffuse abdominal pain, rebound abdominal tenderness, vomiting, constipation, and diarrhea
  - The primary lesion is ulcerative, emesis is blood-tinged or looks like coffee grounds, and stool may be blood-tinged or melenic (dark)
  - Bowel perforation can occur
  - The oropharyngeal form of the disease is characterized by local lymphadenopathy, cervical edema, dysphasic, and upper respiratory tract obstruction
  - Intestinal anthrax results in death in 25% to 60% of cases

**Treatment**

Antibiotics are the primary treatment required for Anthrax. Supportive care and adjunctive care as required for patients.

- **First Line**
  - Ciprofloxacin (500 mg twice daily orally or 400 mg every 12 hours intravenously)
  - **OR**
  - Doxycycline (100 mg every 12 hours orally or intravenously)
- **Second Line**
  - Amoxicillin (500 mg three times daily orally)
  - **OR**
  - Penicillin G (2 mU every 4 hours intravenously)
- **Third Line**
  - Rifampin, Clindamycin, Clarithromycin, Erythromycin, Vancomycin, and Imipenem

**Prophylaxis**

An FDA approved vaccine can be used for pre-exposure of high risk personnel. The vaccine is a series of six 0.5 ml subcutaneous doses at 0, 2, and 4 weeks; then 6, 12, and 18 months followed by a annual booster. A post exposure prophylaxis antibiotic regimen can be given following the same regimen as the treatment regimen.

**Isolation and Decontamination**

There is no isolation requirement for patients; use standard precautions when in contact with patients. Protective garments and mask are required when handling contaminated articles. Contaminated materials should be bagged and incinerated or autoclaved (121+1 degree C core temperature for 30 min). Spores are sensitive to 5-10% bleach solutions with a minimum of 10 minutes contact time. Large scale decontamination is difficult and costly.

**PLAGUE (YERSinia Pestis)**

Plague (*Yersinia Pestis*) is an infectious disease that affects animals and humans. It is caused by the bacterium *Yersinia pestis*. This bacterium is found in rodents and their fleas and occurs in many areas of the world, including the United States. It is a gram-negative rod that is non-motile, and non-sporulating. It is easily destroyed by sunlight and drying. When released into air, the bacterium will only survive for up to one hour, depending on conditions. Plague is endemic in many countries in Africa, in the former Soviet Union, the Americas and Asia. As a biological threat, plague was produced and weaponized in large quantities by the former Soviet Union. It would not be difficult for a terrorist group to obtain and produce plague.
Forms of Disease and Infectious Route

- **Pneumonic plague**: Occurs when *Y. pestis* infects the lungs. This type of plague can spread from person to person through the air. Transmission can take place if someone breathes in aerosolized bacteria, which could happen in a bioterrorist attack. Pneumonic plague is also spread by breathing in *Y. pestis* suspended in respiratory droplets from a person or animal with pneumonic plague.

- **Bubonic plague**: This is the most common form of plague. This occurs when an infected flea bites a person or when materials contaminated with *Y. pestis* enter through a break in a person's skin. This occurs in nature with infected fleas and rodents.

- **Septicemic plague**: Occurs when plague bacteria multiply in the blood. It can be a complication of pneumonic or bubonic plague, or it can occur by itself.

Incubation Period

The average is 1 to 7 days and may be a few days longer for those immunized who develop the illness. Primary plague pneumonia incubation is usually short 1 to 4 days.

Signs and Symptoms

- **Pneumonic**: The onset is sudden, with high fever, chills, malaise, tachycardia, intense headache, and severe myalgias (muscle pain). The patient appears profoundly ill. Rapidly developing pneumonia followed by cough with hemoptyis, dyspnea, stridor, cyanosis, and death. Death results from respiratory failure, circulatory collapse, and shock. If treatment does not start within 24 hours of onset of symptoms, the disease is 100% fatal.

- **Bubonic**: It is characterized by swollen painful lymph nodes called buboes, high fever, chills, headache, and malaise. The resulting bubo is usually 1 to 10 centimeters in diameter, swollen, painful and warm to the touch. It can cause so much pain that the affected body part cannot be moved. The bubo usually develops in the groin (Fig. 23-12), but may also appear in the armpit or neck, depending on where the flea bite occurred. Bubonic may progress to either Septicemic or Pneumonic.

![Figure 23-12. Swollen Lymph Glands "Buboes" Caused by Plague Bacteria (Bubonic Plague)](image)

*Image reprinted with permission by: National Museum of Health and Medicine, Armed Forces Institute of Pathology (MIS 219900-7B). Washington, DC.*

- **Septicemic**: This phase of plague usually follows bubonic in most cases. In addition to the preceding signs the patient develops prostration, circulatory collapse, septic shock, organ failure, hemorrhage, disseminated intravascular coagulation, and necrosis of the extremities can be seen.

Laboratory testing can confirm plague. Small gram-negative and/or bipolar-staining coccobacilli can be seen on a smear taken from affected tissues of a sputum sample, cerebral spinal fluid, or aspiration from a bubo. Plague is confirmed by a positive culture.
Treatment

Treatment should be started immediately once plague is suspected. Immediate administration of antibiotics is required to reduce mortality. This is especially true with primary pneumonic plague in which the mortality rate approaches 100% if not initiated within 18 – 24 hours. Supportive care should include hemodynamic monitoring.

- Primary Antibiotics
  - Streptomycin (1g every 12 hours intravenously)
  - Gentamicin (5mg/kg intravenously or intramuscular every day or 2mg/kg loading dose, then 1.7 mg/kg every 8 hours intravenously)
- Alternative Antibiotics
  - Doxycycline, Ciprofloxacin, or Chloramphenicol

Prophylaxis

Post exposure prophylaxis for face-to-face contact of patients with pneumonic plague or personnel exposed to an intentional release aerosol plague should be given antibiotics for the duration of the exposure plus 7 days. There is currently no approved vaccination (in the United States) for plague.

- Primary Prophylaxis
  - Doxycycline (100 mg orally twice a day)
- Alternative Prophylaxis
  - Ciprofloxacin or Chloramphenicol

Isolation and Decontamination

Standard precautions should be used for bubonic and septicemic plague. Respiratory isolation and precautions against airborne spread is required for pneumonic plague. Rid patients and their clothing of fleas. Decontamination can be done with a 1% bleach solution. Patients can be cleaned with a soap and water solution.

The *Y. pestis* bacterium can be inactivated by heat (greater than 15 minutes at 55°-72°C) or direct sunlight (more than 2 hours).

TULAREMIA (FRANCISELLA TULARENSIS)

*Tularemia*, also known as “rabbit fever,” is a disease caused by the bacterium *Francisella tularensis*. It is typically found in animals, especially rodents, rabbits, hares, and ticks. It is usually a rural disease and has been reported in all U.S. states except Hawaii. It is a gram-negative, non-motile coccobacillus. The bacterium has several subspecies with varying degrees of virulence. As a biological threat, it is rarely fatal (approx. 1-2%), but can be seriously incapacitating. *F. tularensis* is one of the most infectious pathogenic bacteria, requiring less than 10 organisms for infection. It was used as a biological weapon during WWII, and engineered to be antibiotic/vaccine resistant by the United Soviet Socialist Republic (USSR); now Russia.

Forms of Disease and Infectious Route

There are two subspecies and several types of the disease that are determined by the route of infection. Of the subspecies, Jellison type A and B, Type A is the most virulent. Humans become infected through bites from infective arthropods (ticks and deer flies), handling infectious animal tissues or fluids, direct contact with, or ingestion of, contaminated matter, or inhalation of infective aerosols. Tularemia is not spread from person to person.

- **Ulceroglandular**: is the most common form of tularemia and usually occurs following a tick or deerfly bite, or after handing of an infected animal.
- **Pneumonic and typhoidal**: forms of the disease would likely be the predominate forms following an intentional aerosol release of the agent. *Tularemia*, in aerosol form, is considered a possible bioterrorist agent.
Incubation Period

Usually 3 to 5 days with a range of 1 to 14 days.

Signs and Symptoms

Tularemia presents generally with abrupt onset fever, headache, chills, generalized body aches, and nausea begins suddenly. Diagnosis of tularemia is confirmed by serological testing.

- **Ulceroglandular**: A skin ulcer appears at the site where the organism entered the body. The ulcer is accompanied by regional lymphadenopathy usually in the armpit or groin.
- **Pneumonic and typhoidal**: Symptoms include cough, chest pain, and difficulty breathing. This form results from breathing dusts or aerosols containing the organism. Develops when other forms of tularemia are left untreated and the bacteria spread through the blood stream to the lungs.

Treatment

The use of antibiotics is necessary for the treatment of Tularemia.

- **Primary Antibiotics**
  - Streptomycin (1g every 12 hours intramuscular) or
  - Gentamicin (5mg/kg intravenously or intramuscular every day)
- **Alternative Antibiotics**
  - Doxycycline, Ciprofloxacin, or Chloramphenicol

Prophylaxis

Post exposure antibiotics should begin within 24 hours of exposure and continue for 14 days.

- **Preferred Prophylaxis**
  - Doxycycline (100 mg orally twice a day)
  - Ciprofloxacin (500 mg orally twice a day)

Isolation and Decontamination

Isolation is not recommended for tularemia patients, given the lack of person-to-person transmission. Standard precautions are recommended when treating patients. Clothing or linens contaminated with body fluids of patients with tularemia should be disinfected. The agent is rendered harmless when exposed to heat (55°C for 10 minutes) and susceptible to 1% bleach solution followed by 70% alcohol solution and standard water chlorination.

**BOTULINUM TOXIN (CLOSTRIDIUM BOTULINUM)**

Botulinum Toxin is produced by *Clostridium botulinum*, an encapsulated, anaerobic, gram-positive, spore-forming, rod-shaped bacterium. It is a neuroparalytic (muscle-paralyzing) disease blocking acetylcholine release from peripheral nerves. The toxin is highly lethal and easy to produce and release. The toxin is the most toxic substance known and is 10-15,000 times more toxic than VX nerve agent by weight. As a biological threat botulinum can be easily produced and disseminated. It has been used in former state sponsored bioweapons programs in Japan, Germany, US, USSR, Iran, Iraq, North Korea, and Syria.

**Forms of Disease and Infectious Route**

Seven antigen types (A-G) of the toxin exist; only types A, B, E and F are known to cause illness in humans. There are no reported cases of person-to-person transmission. There are four kinds of botulism:

- **Food-borne botulism**: Occurs when a person ingests pre-formed toxin that leads to illness within a few hours to days
- **Infant botulism**: Occurs in a small number of susceptible infants each year who harbor *C. botulinum* in their intestinal tract
- **Wound botulism**: Occurs when wounds are infected with *C. botulinum* that secretes the toxin
• **Inhalational botulism:** Occurs when the *C. botulinum* in aerosol form is inhaled. There is no natural occurrence of this type and is feasible to be used as a weapon by a terrorist group or another country.

**Incubation Period**

Neurological symptoms usually appear within 12 to 36 hours. It may take several days to develop if exposed to a low dose. Time to onset is does dependent.

**Signs and Symptoms**

The classic symptoms of botulism include cranial nerve palsies (double vision, blurred vision, drooping eyelids, slurred speech), dysphagia, dry mouth and throat, and muscle weakness. These are all symptoms of the muscle paralysis caused by the bacterial toxin. If untreated, these symptoms may progress to cause paralysis of the arms, legs, trunk and respiratory muscles. Laboratory confirmation can be obtained by a bioassay of the patient’s blood serum.

**Treatment**

Early administration of Trivalent antitoxin (which neutralizes against toxin types A, B, and E) or Heptavalent antitoxin (for types ABCDEFG is held by the US Army). Respiratory failure is managed with intubation and mechanical ventilation. Parenteral fluids and supportive care may be required.

**Prophylaxis**

A Pentavalent (ABCDE) toxoid is distributed by the CDC to immunize laboratory workers and protect troops against attack.

**Isolation and Decontamination**

Isolation is not required for botulism patients. Use standard precautions when treating patients and good hand washing after handling soiled material.

The toxin is readily inactivated by sunlight (1 to 3 hours) or heat (80°C for 30 minutes, 100°C for several minutes) and surfaces may be decontaminated with a 1% bleach solution. Clothing and skin should be washed thoroughly with soap and water.

**RICIN**

Ricin is a potent toxin that has potential to be used as an agent of biological warfare and as a weapon of mass destruction (WMD). It is derived from the beans of the castor plant (*Ricinus communis*) and can be made from the waste material left over from processing castor beans. The toxin blocks protein synthesis at the cellular level. The loss of protein synthesis leads to irreversible cell death and tissue damage. As a biological threat, it is considered relatively easy to produce and can be delivered by aerosolization, injection, or ingestion.

**Form of disease and Infectious Route**

Route of exposure is inhaled aerosol, ingestion, and parenteral (injected). Clinical manifestations depend on the route of exposure and amount of absorption. It can be in the form of powder, mist, pellet, or it can be dissolved in water or weak acid. There is no person to person transmission.

**Incubation Period**

Symptoms will onset within a few hours by ingestion and within 18-24 hours by inhalation. If a lethal dose is experienced, time to death is a matter of a few days.

**Signs and Symptoms**

- **Ingestion:** Mild exposure can lead to nausea, vomiting, diarrhea, and abdominal pain. Severe poisoning can result in GI tract symptoms progressing (4-36 hours) to renal and liver failure, and finally death.
• **Inhalational:** Illness within 8 hours including cough, shortness of breath, fever, respiratory distress. This will progress to pulmonary edema, airway necrosis, and death

• **Injection:** Initial (<6 hours) weakness and myalgias (muscle pain), progressing (24-36 hours) to vomiting, hypotension, multiorgan failure, and death

**Treatment**

There is no known antidote or other specific treatment. Provide supportive care for fluid loss due to gastroenteritis, cardiac and respiratory support as needed.

**Prophylaxis**

None.

**Isolation and Decontamination**

Isolation in not required for Ricin patients. Use standard precautions when treating patients. Ricin can be heat inactivated at 85°C for 10 minutes or 50°C for 1 hour. Decontamination can be accomplished with soap and water. The agent is inactivated with 1% bleach solution for 10 minutes contact time.

**SMALLPOX (VARIOLA MAJOR AND VARIOLA MINOR)**

Smallpox is a serious, contagious, and sometimes fatal infectious disease. It is caused by the *variola* virus that emerged in human populations thousands of years ago. Except for two known laboratory stockpiles, the *variola* virus has been eliminated. However, in the aftermath of the events of September and October, 2001, there is heightened concern that the *variola* virus might be used as an agent of bioterrorism.

As a Biological threat, smallpox presents a great risk. The last reported case was in 1977 and routine smallpox vaccination ceased in 1980, thus the current population is highly susceptible.

If smallpox was intentionally released it would spread rapidly through aerosols or mucous membrane transmission. The virus has been successfully weaponized in long range missiles by the former Soviet Union which is now Russia and various other eastern-block countries. There is some question about the control of Russian virus stocks.

**Forms and Infectious Route**

Generally, direct and fairly prolonged face-to-face contact is required to spread smallpox from one person to another. Smallpox can also be spread through direct contact with infected bodily fluids or contaminated material such as bedding or clothing. Humans are the only natural hosts of *variola*. Smallpox is not known to be transmitted by insects or animals. There are two clinical forms of smallpox:

- **Variola major** is the severe and most common form of smallpox, with a more extensive rash and higher fever. There are four types of *variola major* smallpox: Ordinary, modified, flat, and hemorrhagic. Historically, *variola major* has an overall fatality rate of about 30%; however, flat and hemorrhagic smallpox usually are fatal
- **Variola minor** is a less common presentation of smallpox, and a much less severe disease, with death rates historically of 1% or less

**Incubation Period**

From 7 to 19 days, usually 10 to 14 days to the onset of illness and 2 to 4 additional days before the onset of a rash.

**Signs and Symptoms**

Symptoms of smallpox infection usually appear within 10 to 12 days after exposure to the virus. The first symptoms of smallpox may be difficult to distinguish from other flu-like illnesses and include: High fever, fatigue, malaise, headache, backache, and rash.
A characteristic rash, most prominent on the face, arms, and legs, follows 2 to 3 days after the first symptoms. The rash starts with macules and quickly progress to papules (Fig. 23-13). After a few days, the lesions become pustular vesicles and begin to crust early in the second week. Scabs develop and then separate and fall off after about 3 weeks.

**Figure 23-13.—Typical Centrifugal Rash Distribution**

*Image reprinted with permission by: National Museum of Health and Medicine, Armed Forces Institute of Pathology (Reeve 48135). Washington, DC.*

**Treatment**

There is no proven treatment for smallpox. People with the disease can benefit from intravenous fluids and medicine to control fever or pain as well as antibiotics for any secondary bacterial infections that may occur. If an infected person gets the smallpox vaccine within 4 days after exposure to the virus, it may lessen the severity of illness or even prevent illness.

**Prophylaxis**

Immediate vaccination or revaccination should be administered to all exposed personnel and those living in the immediate vicinity (ring vaccination).

**Isolation and Decontamination**

Patients should be considered contagious from the onset of the rash until all scabs separate. They should be isolated using airborne and droplet precautions during this period. The aerosolized virus may persist for 24 hours under optimal conditions. Extra care must be taken with infected clothing and bedding as well as infected scabs and bodily fluids. Contaminated materials should be bagged and incinerated or autoclaved. Decontamination can be accomplished by using standard hospital-grade disinfectants such as quaternary-ammonia compounds as these are effective in killing the virus. They should be used on surfaces to disinfect hospitalized patients’ rooms or other contaminated surfaces. A 5-10% bleach solution with at 10 minute contact time is also recommended.

**HEMORRHAGIC FEVERS**

Viral hemorrhagic fevers (VHFs) refer to a group of illnesses that are caused by four distinct families of viruses. The viruses are characterized by fever and bleeding. The overall vascular system is damaged and the body's ability to regulate itself is impaired. While some types of hemorrhagic fever viruses can cause relatively mild illnesses, many of these viruses cause severe life-threatening disease.

As a biological threat, Marburg and Ebola viruses exhibit potential for development as biological weapons. They are highly infective and provide a person-to-person aerosol transmission. These particular viruses have an extremely high mortality and there is no treatment available.
Forms of Disease and Infectious Route

There are four families of VHF:

**Arenaviruses:** Family of viruses whose members are generally associated with rodent-transmitted disease in humans via rodent urine and excrement. There has also been documented person-to-person transmission with close contact in a health care setting. Each virus usually is associated with a particular rodent host species in which it is maintained. Arenavirus infections are relatively common in humans in some areas of the world and can cause severe illnesses.

**Filoviruses:** Family of viruses called Filoviridae can cause severe hemorrhagic fever in humans and nonhuman primates. So far, only two members of this virus family have been identified: Marburg virus and Ebola virus. Four species of Ebola virus have been identified: Ivory Coast, Sudan, Zaire, and Reston. Ebola-Reston is the only known filovirus that does not cause severe disease in humans. This group of viruses is transmitted by persons-to-person contact.

**Bunyaviruses:** This group of viruses is vector-borne and transmission occurs via an arthropod vector (mosquito, tick, or sandfly), with the exception of Hantaviruses. Hantaviruses are transmitted through contact with deer mice feces. This group includes Crimean Congo Hemorrhagic Fever, Rift Valley fever, and Hantaan.

**Flaviviruses:** This is a group of several viruses that includes Dengue virus and Yellow Fever Virus. These viruses are transmitted by the bite from an infected arthropod (mosquito or tick).

Incubation Period

This varies with each virus.

Signs and Symptoms

VHF are illnesses that are characterized by fever and bleeding. Specific signs and symptoms vary by the type of VHF, but initial signs and symptoms often include marked fever, fatigue, dizziness, muscle aches, loss of strength, and exhaustion. Patients with severe cases of VHF often show signs of bleeding under the skin, in internal organs, or from body orifices like the mouth, eyes, or ears. However, they may bleed from many sites around the body; patients rarely die because of blood loss. Severely ill patient cases may also show shock, nervous system malfunction, coma, delirium, and seizures. Some types of VHF are associated with renal (kidney) failure. Laboratory diagnosis is required.

Treatment

Patients receive supportive therapy, but generally speaking, there is no other treatment or established cure for VHF. Ribavirin, an antiviral drug, has been effective in treating some individuals with Lassa fever or hemorrhagic fever with renal syndrome. Treatment with convalescent-phase plasma has been used with success in some patients with Argentine hemorrhagic fever.

Prophylaxis

The only approved VHF vaccination is for Yellow Fever.

Isolation and Decontamination

All VHF patients should be placed in strict isolation. Strict contact precautions should be instituted along with airborne precautions to the maximum extent possible. Providers who must be in contact should be in protective clothing and wear a HEPA respirator. All contaminated materials should be autoclaved or incinerated. These agents are susceptible to 1% bleach solution and phenolic disinfectants.
RADIOLOGICAL WARFARE

LEARNING OBJECTIVE:

Identify medical conditions that occur after due to radiation exposure and provide appropriate medical treatment.

The principles for medical treatment of casualties, as developed from previous experiences in conventional warfare, are applicable in the treatment of casualties produced by radiological warfare. With the exception of ionizing radiation effects, the type of injuries produced in nuclear warfare are similar to those of conventional warfare. Standardized techniques of treatment must be adopted for all types of casualties so the greatest number of patients can receive maximum medical care in the shortest period of time with the greatest economy of medical personnel and equipment.

HISTORY

The first use of an atomic weapon during war took place during WWII. The death and devastation evidenced by the nuclear weapons in wartime (in Hiroshima and Nagasaki, Japan, at the end of World War II) has, to date, kept it from being used again. Although a nuclear non-proliferation treaty has been signed by most of the major powers, nuclear weapons are still a part of the arsenal of many countries of the world. Other countries are continuing to develop nuclear weapons such as North Korea and Iran.

Recent history has shown a disturbing change in the potential use of nuclear weapons. Terrorist organizations have added a new dimension to the use of radiological material. These groups have been seeking a nuclear bomb or the materials necessary to develop a nuclear device. Terrorist organizations may not be able to develop a fully functional nuclear bomb; they may develop and utilize a radiological disbursal device (RDD) or dirty bomb.

A RDD is any device that causes the purposeful dissemination of radioactive material across an area without a nuclear detonation. Such a weapon can be easily developed and used by a person with conventional explosives and access to radioactive material (radionuclides; radioactive isotopes). A radioactive source is blown up using conventional explosives and radioactive material is scattered across the targeted area as debris. The material dispersed can originate from any location that uses radioactive sources, such as a nuclear waste processor, a nuclear power plant, a university research facility, a medical radiotherapy clinic, or an industrial complex.

The use of a RDD type of weapon is likely to cause more of a psychological impact than that of radiation injuries. The resulting blast would cause conventional casualties to become contaminated with radionuclides and would complicate medical evacuation within a contaminated area.

The detonation of a nuclear weapon or RDD is not the only form of potential radiological contamination of military forces. Other sources of radiological contamination are:

- The destruction of a nuclear reactor
- A nuclear accident
- Improper nuclear waste disposal
- Many materials used in military ordnance, equipment, and supplies

U.S. forces may be operating in a theater that has nuclear reactors that are not designed to U.S. specifications and are without containment vessels. These reactors may be lucrative enemy artillery or bombing targets. Military operations in these areas, if contaminated, could result in military personnel receiving sufficient radiation exposure or particulate contamination to warrant medical evaluation and remediation.
TYPES OF RADIATION

Radioactivity may be defined as the spontaneous and instantaneous decomposition of the nucleus of an unstable atom with the accompanying emission of a particle, a gamma ray, or both. The actual particles and rays involved in the production of radiation injuries are the alpha and beta particles, the neutron, and the gamma ray.

When radiation interacts with atoms, energy is deposited, resulting in ionization. It is this ionization that becomes a health concern because it may damage certain critical molecules or structures in a cell. Damage to a sufficient number of molecules within the cell, the cell will not be able to carry on its normal functions and will die. There are two modes of radiation action within a cell:

- **Direct action** is when radiation may directly hit a particularly sensitive atom or molecule in the cell. The resulting damage is irreparable; the cell either dies or performs improperly.
- **Indirect action** is when radiation interacts with water molecules in the body. The energy deposited in the water leads to the creation of unstable, toxic hyperoxide molecules. These molecules damage sensitive molecules and affect sub-cellular structures.

**Alpha**

Alpha particles are emitted from the nucleus of some radioactive elements. They are heavy, very short-range particles that are not able to penetrate clothing or human skin. Alpha-emitting materials can be harmful to humans if the materials are inhaled, swallowed, or absorbed through open wounds. If absorbed in the body, particles will cause significant cellular damage in the immediate area adjacent to the particle's physical location. Care must be taken around possible contaminated sites because instruments cannot detect alpha radiation through even a thin layer of water, dust, paper, or other material, because alpha radiation is non-penetrating.

**Beta**

Beta radiation is a light, short-range particle and is actually an ejected electron. This form of radiation may travel several feet in the air and is moderately penetrating, more than Alpha particles. Beta radiation can penetrate human skin to the "germinal layer," where new skin cells are produced. If high levels of beta-emitting contaminants are allowed to remain on the skin for a prolonged period of time, they may cause skin injury. They cause serious internal damage if absorbed into the body.

**Gamma and X – Rays**

Gamma rays are electromagnetic waves. Gamma rays and x-rays are different types of radiation, as a biological impact they have the same effect. Gamma radiation and x-rays are highly penetrating electromagnetic radiation. Gamma radiation or x-rays are able to travel many feet in air and many inches through human tissue. They readily penetrate most materials and are sometimes called "penetrating" radiation and constitute mainly an external hazard to humans. Dense materials are needed for shielding from gamma radiation. Clothing provides little shielding, but will prevent contamination of the skin by gamma-emitting radioactive materials.

**Neutrons**

Neutrons are emitted only during a nuclear fusion or detonation and are a form of high penetrating radiation that presents no fallout hazard. They have significant mass and interact with the nuclei of atoms, severely disrupting atomic structures. Compared to gamma rays, they can cause 20 times greater damage to tissue. Neutrons and gamma rays are an important medical consideration in a nuclear explosion since their range is great enough to produce biologic damage, either alone or in conjunction with blast and thermal injuries.
UNITS OF RADIATION

The radiation absorbed dose (rad) is used to measure a quantity of absorbed dose of radiation. It relates to the amount of energy absorbed in material. This does not describe the biological effects of different radiation. The International System of Units (SI) is replacing common American terminology; the new absorbed dose unit for radiation is the gray (Gy). The unit for dose, gray, is used for all forms of ionizing radiation (Table 23-4). Dose is the total amount of energy absorbed per gram of tissue. The exposure could be single or multiple and either short or long in duration.

Equal doses of different types of radiation cause different amounts of damage to living tissue. For example, 1 Gy of alpha radiation causes about 20 times as much damage as 1 Gy of x-rays. The energy from x-rays are dispersed through the human body, whereas the energy from an alpha particle is highly concentrated and will kill cells in the immediate vicinity of the particle. Therefore the equivalent dose was defined to give an approximate measure of the biological effect of radiation. For comparison, the 'background' dose of natural radiation received by a US citizen is around 3 mSv (300 mrem) per year. A lethal full-body dose of radiation for a human is around 4 - 5 Sv (400 - 500 rem) instantaneously.

MEASUREMENT DEVICES

There are special devices used to measure field radiation levels. Some devices are currently available to conduct radiological surveys and measure radiation levels on equipment and personnel. They are the AN/VDR-2 and the AN/UDR-13 for field use and the AN/PDQ-1 for shipboard use. There are other radiation detection systems available; these are a few examples of what is in current use.

<table>
<thead>
<tr>
<th>SI Unit</th>
<th>Old Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Gy</td>
<td>1 joule/kg</td>
</tr>
<tr>
<td>1 Gy</td>
<td>100 Rad</td>
</tr>
<tr>
<td>10 milliGray (mGy)</td>
<td>1 Rad</td>
</tr>
<tr>
<td>1 Sv</td>
<td>100 rem</td>
</tr>
<tr>
<td>10 milliSievert (mSv)</td>
<td>1 rem</td>
</tr>
</tbody>
</table>

Table 23-4.—SI Conversion Chart

Adapted from Medical Management of Radiological Casualties 2nd ed.

Two other units that may be used to express radiation units are Roentgen equivalent man (Rem) and Sievert (Sv):

- **Rem** is the special unit used to derive a quantity of exposed dose
  - It relates to the absorbed dose in human tissue as related to biological damage from radiation
  - The dose equivalent in rems is equal to the absorbed dose in rads multiplied by the quality factor (1 rem = 0.01 Sv)

- **Sievert** is the SI unit of any of the quantities expressed as dose equivalent
  - The dose equivalent in sieverts is equal to the absorbed dose in grays multiplied by the quality factor (1 Sv=100 rems)
Radiac Set AN/VDR-2

The AN/VDR 2 (Fig. 23-14) is used to perform ground radiological surveys in vehicles or in dismounted mode by individual personnel as a handheld instrument. The device provides a quantitative measure of radiation to assist with the decontamination of personnel, equipment, and supplies. The device can be used with vehicle power or internal batteries.

![Radiac Set AN/VDR-2](image1.png)

Figure 23-14.—Radiac Set AN/VDR-2


Radiac Set AN/UDR 13

The AN/UDR 13 (Fig. 23-15) is a compact, handheld, or pocket carried, tactical device that can measure prompt gamma/neutron doses from a nuclear event. It can measure the total gamma dose and the dose rate from nuclear fallout. The LCD provides data readout and warning and mode messages. The unit is worn by an individual and allows the monitoring of radiation dose. It provides a warning to inform a person they have received a set amount of radiation; they can depart the contaminated site.

![Radiac Set AN/UDR-13](image2.png)

Figure 23-15.—Radiac Set AN/UDR-13


Radiac Set AN/PDQ 1

The AN/PDQ-1 (Fig. 23-16) is a multi range Radiac device that detects beta and gamma radiation. The AN/PDQ-1 uses a Geiger-Mueller ionization chamber and has two ranges for radiation levels, mR/hr and R/hr. The probe measures gamma radiation and can also be used to detect beta radiation when the probe shield is open. It is powered by two D cell batteries and is used for personnel monitoring and surveys for hot spots onboard a ship. The device can be used to detect radiation levels on a patient during decontamination. The AN/PDQ-1 uses a Geiger-Mueller ionization chamber and has an operating range of 0-1000 R/HR. The device has two ranges for radiation levels, mR/hr and R/hr. The probe measures gamma radiation and can be used to detect beta radiation when the probe shield is open.

![Radiac Set AN/PDQ-1](image3.png)

Figure 23-16.—Radiac Set AN/PDQ 1

*Image reprinted with permission from: NAVSEA Damage Control, Fire Protection Engineering and CBR-D.*
EXPOSURE FACTORS

Personnel entering contaminated areas to either remove casualties or work in decontamination stations have two major concerns. The first concern is the prevention of their own contamination, and the second is the prevention or reduction of radioactive exposure. Contamination can be avoided by decontaminating patients and equipment before handling, wearing appropriate protective clothing and equipment, avoiding highly contaminated areas, and strictly observing personal decontamination procedures.

Radioactive material presents a direct risk to the health and safety of all personnel. This risk can be avoided (or at least minimized) by following some simple guidelines and using common sense. The three major factors that guide actions to avoid exposure are time, distance, and shielding.

Time

Radioactive decay and the decomposition of fallout products progress rapidly in the early hours after a nuclear blast, and the hazards to rescue workers can be reduced considerably if operations can be delayed until natural decay has reduced the level of radioactivity. Some radioactive materials may take a number of years to decay before it reaches a safe level of radioactivity. In either case, a rescue worker may have to enter a contaminated area before safe levels can be achieved. If so, the workers exposure time to radioactive material must be limited.

The more time spent in a contaminated environment, the higher Gy dose. Limiting the time of exposure is essential if total avoidance is not possible. Rotating personnel entering an exposure risk area, planning actions to minimize time in the area, and prompt decontamination reduce the total time the individual is exposed, thereby reducing the dose of radiation absorbed by the body.

Distance

Both radioactive particles and electromagnetic waves (gamma rays) lose energy and consequently lose their ability to harm tissue as they travel away from their source. The farther a person is away from the source, the reduction of the exposure level. The closer a person is to a radioactive source, the higher Gy dose will be absorbed by that person.

Shielding

Shielding is an essential component in preventing/reducing radiation exposure. Alpha and beta particles have very little penetrating power, and intact skin forms an adequate barrier in most cases. Gamma radiation has much greater penetrating power and presents the greatest risk of exposure and damage to tissue.

Lead is the most effective shielding material. Wood, concrete, other metals, and heavy clothing will somewhat reduce the amount of gamma radiation that reaches the body. Most particle exposure is the result of inhalation or ingestion, although radiation particles may enter the body through burned, abraded or lacerated skin. Breaks in skin integrity (i.e. rash, laceration, or psoriasis) will increase the penetration of gamma rays, x-rays, and beta rays resulting in a more contaminated patient.

PERSONNEL PROTECTION EQUIPMENT (PPE)

Protective Masks

Standard issue chemical protective masks afford excellent protection from inhalation and ingestion of radioactive material. Radon and tritium gas will pass through the filters, but short exposures are not medically significant. Increasing oral fluids and maintaining sufficient urine output will adequately treat tritium exposures. Vehicle fires produce dangerous chemical fumes from burning metals and plastics and deplete closed-space oxygen; a self-contained breathing apparatus may be necessary in such cases.
Protective Clothing

Commercial anti-contamination suits (Tyvek® Anti-C Suits) are ideal but offer little advantage over standard MOPP–4. Chemical-protective over-garments provide excellent contamination protection as well as protection from chemical-biological agents in the combat environment.

Standard hospital barrier clothing as used in Universal Precautions is adequate for emergency treatment of limited numbers of contaminated casualties. Medical personnel should be decontaminated following patients’ emergency treatment and decontamination.

In avoiding particle exposure, full personnel-protective clothing and a protective mask with hood provides the best protection. A protective mask and foul-weather gear will provide lesser, but adequate protection. In cases where no protective breathing devices are available, some protection is afforded by breathing through a folded towel, handkerchief, or several surgical masks. Avoid hand-to-mouth contact, eating, or smoking in contaminated areas.

EFFECTS ON PERSONNEL

Direct exposure to radiation will damage body tissue. The extent of the damage is dependent upon the quantity of radiation delivered to the body, the type of radiation, the dose rate, duration of exposure, and the organs exposed (Table 23-5). Experience has shown that the faster the onset of early symptoms (nausea and vomiting); the higher the dose of radiation received by the patient.

<table>
<thead>
<tr>
<th>Time Onset</th>
<th>Estimated Dose</th>
<th>Degree of ARS</th>
<th>Fatality Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 hr</td>
<td>&lt; 30 Gy</td>
<td>Lethal</td>
<td>100</td>
</tr>
<tr>
<td>&lt; 1 hr</td>
<td>6 – 30 Gy</td>
<td>Lethal</td>
<td>90 - 100</td>
</tr>
<tr>
<td>1 – 2 hr</td>
<td>6 – 8 Gy</td>
<td>Very Severe</td>
<td>90 – 100</td>
</tr>
<tr>
<td>2 – 4 hr</td>
<td>2 – 6 Gy</td>
<td>Moderate</td>
<td>0 - 80</td>
</tr>
<tr>
<td>3 – 6 hr</td>
<td>1 – 2 Gy</td>
<td>Mild</td>
<td>0</td>
</tr>
<tr>
<td>None</td>
<td>0 – 1 Gy</td>
<td></td>
<td>0</td>
</tr>
</tbody>
</table>

Table 23-5.—Estimated (Whole Body) Radiation Dose by Time of Symptom Onset

Adapted from Medical Management of Radiological Casualties 2nd ed.

ACUTE RADIATION SYNDROME (ARS)

Acute Radiation Syndrome or ARS is an acute illness caused by irradiation of the body by a high dose of penetrating radiation in a very short period of time, usually a matter of minutes. It is also known as radiation toxicity or radiation sickness. The major cause of this syndrome, in its simplest form, is cell death in specific tissues. Examples of people who suffered from ARS are the survivors of the Hiroshima and Nagasaki atomic bombs or the firefighters that first responded after the Chernobyl Nuclear Power Plant event in 1986.
Signs and Symptoms

The first symptoms (Prodromal Phase) of ARS are typically nausea, vomiting, diarrhea, and malaise. These symptoms will start within minutes to days after the exposure, will last for minutes up to several days, and may come and go.

The second phase or (Latent Phase) results in the person usually looking and feeling healthy for a short time, after which will become sick again (Latent Period). Time of onset will vary between 0 – 15 days dependent upon the degree of ARS.

Patients may experience loss of appetite, fatigue, fever, nausea, vomiting, diarrhea, and possibly even seizures and coma. This seriously ill stage may last from a few hours up to several months.

The third phase (Manifested Illness) time of onset ranges from day one (Lethal) to over 2 weeks (Mild). Symptoms are convulsions, ataxia, tumor, lethargy, severe diarrhea, fever, and electrolyte disturbance for lethal ARS. Symptoms for moderate to very severe ARS are severe leucopenia (decreased white blood cells), purpura (purple colored spots or patches), hemorrhage, pneumonia, and hair loss after 3 Gy. Mild ARS is demonstrated by moderate leucopenia (decreased total number of white blood cells in the circulating blood).

People with ARS typically have some skin damage. This damage can start to show within a few hours after exposure and include swelling, itching, and redness of the skin (resembling a severe sunburn). Hair loss is common with these patients. The skin may heal for a short time, followed by the return of swelling, itching, and redness days or weeks later. Complete healing of the skin may take from several weeks up to a few years depending on the radiation dose received.

The chance of survival for people with ARS decreases as the radiation dose increases, an inverse relationship. Most people who do not recover from ARS will die within several months of exposure. The cause of death in most cases is the destruction of the person’s bone marrow, which results in infections and internal bleeding. For the survivors, the recovery process may last from several weeks up to 2 years.

Treatment

Evaluate ABCs, stabilize any life threatening injuries, and decontaminate. Provide supportive care: Antiemetic, fluids, antibiotics, pain management, and a clean environment. Treat other wounds as necessary. Transfer patient to a medical treatment facility for additional care when he/she is decontaminated.

CHRONIC RADIATION SYNDROME (CRS)

Chronic Radiation Syndrome (CRS) is a medical condition caused by long term exposure to low dose radiation. It is highly unlikely to affect military personnel in an operational setting. It would require prolonged deployments to heavily contaminated areas or long term ingestion of highly contaminated food or water. A person may develop this condition after prolonged exposed to a near-ground weapon detonation, RDD, or major reactor accident that creates contamination with high dose rates.

Signs and Symptoms

Clinical symptoms are diffuse and may include sleep and/or appetite disturbances, generalized weakness and easy fatigability, increased excitability, loss of concentration, impaired memory, mood changes, vertigo, ataxia, paresthesias, headaches, epistaxis, chills, syncope episodes, bone pain, and hot flashes.

Clinical findings may include localized bone or muscle tenderness, mild hypotension, tachycardia, intention tremor, ataxia, weakness, hyperreflexia (occasionally hyporeflexia), delayed menstrual cycles, and underdeveloped secondary sexual characteristics.
Laboratory findings include mild to marked shortage of all types of blood cells and abnormal bone growth.

**Treatment**

After the patient is removed from the radiation environment, clinical symptoms and findings slowly resolve, and complete recovery has occurred from the lower doses. The patient should receive follow-up care at a treatment facility when possible.

**RADIATION DERMATITIS**\(^{36,37}\)

There are two types of Radiodermatitis, Acute and Chronic. Acute Radiodermatitis usually occurs after heavy contamination of bare skin with beta emitting material. This can result from the use of a RDD. It rarely develops in situations where personnel are fully clothed.

This condition is best prevented by washing off the contaminated material. Chronic Radiodermatitis is a result of long term exposure to low levels of radiation (Table 23-6).

<table>
<thead>
<tr>
<th>Radiation</th>
<th>Dose</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>6 – 20 sv</td>
<td>Erythema only</td>
</tr>
<tr>
<td></td>
<td>20 – 40 sv</td>
<td>Skin breakdown in 2 weeks</td>
</tr>
<tr>
<td></td>
<td>&gt;3000 sv</td>
<td>Immediate skin blistering</td>
</tr>
<tr>
<td>Chronic</td>
<td>&gt;20 sv</td>
<td>Dermatitis with possible cancer risk</td>
</tr>
</tbody>
</table>

*Table 23-6.*—Radiation Dermatitis

**Signs and Symptoms**

In either Acute or Chronic Radiodermatitis, the symptoms are the same and the severity is dependent upon the dose received. The area becomes red and irritated. Other symptoms include hair loss in the affected area, edema, decreased sweating, wet or dry shedding of the outer layers of skin, ulcerations, bleeding, and skin cell death. Chronic exposure may result in permanent and irreversible symptoms. Additionally, Squamous Cell Carcinoma may develop within a few years.

**Treatment**

The primary treatment is removal of the radiation source. Removal of contaminated clothing and the bare skin, to include the hair, should be thoroughly washed with soap and water. Cool compresses and pain medication may be used to control the pain. A topical moisturizing cream may provide some relief. Transfer patient to a treatment facility when possible.

**PSYCHOLOGICAL EFFECTS**

Radiation illness symptoms in just a few personnel can produce devastating psychological effects on an entire unit that is uninformed about the physical hazards of radiation. This acute anxiety has the potential to become the dominant source of stress within a unit.

Personnel are more likely to focus on radiation detection and thus increase the potential of injury from conventional battlefield hazards.

**OTHER INJURIES**

Apart from the ionizing radiation effects, most of the injuries suffered in a nuclear weapon explosion will not differ greatly from those caused by ordinary high explosives and incendiary bombs. The types of injuries usually seen are blast, shock wave, burns, and eye burns (flash blindness).

**Treatment**

Most injuries resulting from the detonation of a nuclear device are likely to be mechanical wounds resulting from collapsing buildings and flying debris, and burns caused by heat and light released by the detonation.
A burn is a burn, regardless of whether it is caused by a nuclear explosion or by napalm, and its management remains the same. This is also true of fractures, lacerations, mechanical injuries, and shock. In none of these is the treatment dictated by the cause. For most of the conventional injuries, standard first-aid procedures should be followed.

NOTE:
The following words of caution should be considered when treating wounds and burns:

Dressings for wounds and burns should follow a closed-dressed principle, with application of an adequate sterile dressing using aseptic techniques.

Make no attempt to close the wound, regardless of its size, unless authorized by a physician.

If signs of infection and fever develop, give antibiotics.

DECONTAMINATION

The presence of radiological contamination can be confirmed by passing a radiation detector (radiac) over the entire body. Open wounds should be covered prior to decontamination. Wound debridement is completed by irrigation with normal saline and a clean dressing placed over the wound. Wounds can be verified clean with the use of a radiac. Contaminated clothing should be carefully removed, placed in marked plastic bags, and removed to a secure location within a contaminated area. Bare skin and hair should be thoroughly washed, and if practical, the waste water should be sequestered and disposed of appropriately.

Radiological decontamination should never interfere with medical care. Unlike chemical agents, radioactive particles will not cause acute injury to medical personnel. Decontamination sufficient to remove chemical agents is more than sufficient to remove any radiological contamination.

Radiological decontamination is performed in an identical manner to chemical decontamination. The main difference is in timing. Chemical decontamination is an emergency. Radiological decontamination is not.

Care must be taken to not irritate the skin. If the skin becomes erythematous, some radionuclides can be absorbed directly through the skin. Surgical irrigation solutions, normal saline, should be used in liberal amounts to clean wounds, the abdomen, and the chest. All such solutions should be removed by suction instead of sponging and wiping. Only copious amounts of water, normal saline, or eye solutions are recommended for the eye.

CERTIFICATION OF DECONTAMINATION

Careful examination of the body with a certified radiac, such as the AN/VDR–2, will confirm adequate decontamination. Particular attention must be paid to the hands, fingers, face, hair, and feet. Once safe levels are achieved, the patient can be certified clean.

SUMMARY

There have been changes in recent years with regard to the threat of chemical, biological, and radiological warfare. Terrorism has added a new aspect to this threat. This chapter has outlined a brief history of CBR warfare, agents (chemical and biological), detection devices for CBR weapons, and decontamination methods.