STEP
Simulation-Enhanced Training in Emergency Preparedness

A course for Health Care Providers

INTRODUCTION

A. Background

A medical disaster is one in which the destructive effects of natural or manmade forces overwhelm one's ability to properly allocate existing resources. Disasters cause human death and suffering, permanently transforming the character of the affected community. With the media providing continual live coverage of a disaster, the rest of society is brought into closer contact with the event. Natural disasters (floods, earthquakes, hurricanes, and tornadoes) still occur with striking regularity. More frightening, however, is the growing destructive character of manmade disasters, such as chemical explosions, nuclear meltdowns, and acts of terrorism. Terrorists in particular are now more willing and able to use weapons of mass destruction (WMD) against civilian targets. In all of these instances, healthcare workers are depended upon to alleviate suffering, allocate "limited" medical resources, and bring order to a chaotic environment.

B. Character of a Nuclear, Biological, or Chemical Terrorist Event

A terrorist attack using NBC weapons is very similar, in many respects, to a HAZMAT accident. Both are instantaneous events that have the potential for moving quickly from a localized situation to one involving a large part of the community. To prevent personal injury, first responders and medical personnel must wear appropriate personal protective equipment (PPE).

In many respects, an NBC terrorist attack is a HAZMAT incident with the potential for mass casualties (possibly thousands) and a crime scene all in one. There would be widespread psychological impact, and EMS and hospitals may become overwhelmed. Policies and procedures should be developed, exercised, and followed to ensure a safe and effective emergency response. The disaster plan must conform to the principles of the Incident Command System (ICS).

Realistically, the actions of medical personnel are not influenced by whether the incident is intentional (that is, terrorist or criminal) or accidental. In both cases, appropriate medical intervention will be delivered. It is essential that the facility be prepared to deliver this care in a manner that protects the medical worker. Nonetheless, an NBC terrorist attack on civilians presents a number of unique circumstances to consider.

1. Psychogenic casualties will predominate. These individuals will quickly overwhelm existing resources, and their presenting signs and symptoms may confuse the clinical picture. Once clinical injury has been ruled out, a crisis team of psychiatric assessment specialists should continue the evaluation in a more controlled setting.

2. Responding personnel are at risk for personal injury and secondary contamination. The strategically placed secondary explosive that detonated recently in Georgia (January 1997) reinforced the vulnerability of first responders. Hospital personnel are not immune from such an attack.

3. An intentional attack is a criminal event and "everything" becomes evidence. The emergency team must be aware of the importance of properly collecting and handling evidence ("chain of evidence").

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4. A terrorist incident crosses all jurisdictions. Medical personnel might be required to interact with a number of individuals from local law enforcement, the FBI, specialized response teams, the Center for Disease Control (CDC), the Public Health Department, elected officials, and the media. All of these individuals have special needs that will have to be addressed.

5. The level of violence associated with terrorist acts has produced more victims and higher mortality than is typically seen in natural disasters and hazardous materials accidents. In the Oklahoma City bombing, 168 people died. Contingency plans for the management of large numbers of dead victims should be developed.

C. Definition of Terrorism

Terrorism is the unlawful use of force or violence against persons or property to intimidate or coerce a government or civilian population in the furtherance of political or social objectives. According to the Central Intelligence Agency (CIA) and the FBI, "Terrorism based in the Middle East or elsewhere will someday almost certainly use chemical, biological, and possibly nuclear (weapons) against U.S. targets." This sentiment was recently echoed by Defense Secretary William Cohen, who warned at a press conference, "This scenario of nuclear, biological or chemical weapons in the hands of a terrorist cell or rogue nation is not only plausible, it's … quite real."

D. Current Trends in Terrorism

Terrorists appear to be more willing to use NBC weapons and explosive devices against civilian targets. The number of terrorist acts worldwide has actually decreased over the last few years, but the lethality of these acts has increased.

1. Nuclear. The end of the Cold War and the disintegration of the former Soviet Union have decreased the prospect for thermonuclear war. However, the region's dismal economic condition has created a new and dangerous situation regarding control of the Russian nuclear arsenal and its significant stockpile of nuclear materials. Those with financial and political means have attempted to acquire these weapon-grade materials on the black market. Over the past few years, there have been over 62 documented cases of nuclear smuggling worldwide. Countries such as Libya, Iraq, Iran, and North Korea are aggressively pursuing nuclear weapon capabilities.

2. Biological. In 1972, an American Fascist group, the "Order of the Rising Sun," used 30 to 40 kilograms (kg) of typhoid to contaminate the Chicago and St. Louis water supplies. In 1984, the "Red Army Faction" is alleged to have tried to use botulinum toxin in Paris. In 1986, 715 people became ill in Oregon when the Bhagwan Cult poisoned salad bars with salmonella. In 1995, a member of the Aryan Nation was arrested for ordering plague from a supply house in Maryland. Two members of the Minnesota Patriots Council were convicted for planning to use ricin in an assassination attempt.

3. Chemical. In 1985, the "Covenant Group" in Arkansas was found to have 33 gallons of cyanide in their possession. In 1992, the German police prevented the release of cyanide in a synagogue by Neo-Nazis. 1994 and 1995 heralded the use of sarin by the Aum Shinrikyo with devastating effects. The first attack in Matsumoto killed 7 people and sent 280 to the hospital. In March 1995, the release of sarin in the Tokyo subway left 12 dead and 5,500 injured. The Tokyo subway incident on March 20, 1995, is the most publicized of all the incidents and marks the beginning of a new era in terrorism. After the Tokyo attack, copycat attacks occurred in Japan with the use of cyanide, phosgene, and pepper spray. An FBI action forestalled a possible sarin attack in Disneyland shortly thereafter. The most recent terrorist use of a chemical was in Sydney, Australia. In 1997, two chlorine bombs were activated in shopping malls, injuring 14 and forcing the evacuation of 500 persons.

4. Explosive/incendiary. The World Trade Center Bombing in 1993 killed 6 and injured over 1,000 people. One month after the Tokyo subway attack, the Murrah Federal Building in Oklahoma City was bombed, killing 168 people and injuring more than 750. The U.S. government realized that terror could strike at America's heartland.

Americans are at risk at home and abroad. The rules of engagement have changed.

- In June 1996, the bombing of Khobar Towers in Dharhan killed 19 U.S. airmen.

- A bomb attack at the Olympic Centennial Park in Atlanta altered our sense of security.

First responders (fire, EMS and law enforcement) were targeted for the first time in the U.S. while responding to terrorist attacks. A secondary device was aimed at responders in the 1997 bombings of an Atlanta clinic and a nightclub.

E. Properties and Characteristics of Nuclear, Biological, and Chemical Weapons

Chemical and biological weapons are relatively easy to make. A terrorist needs to go no further than his nearest Internet connection to download the appropriate "recipe." Ingredients are cheap and easy to obtain. These weapons are difficult to detect (tasteless and odorless) and are disseminated via the air. A high index of suspicion should be present when large numbers of the population present with similar symptoms. Enclosed spaces provide ideal targets, particularly those that draw large crowds such as sporting events and underground modes of transportation.

1. Nuclear (radiation). These are an invisible threat whose adverse effects may be delayed for days to years depending on the dose. The risk to victims and responders varies by the route of exposure (that is, internal vs. external) and the type of ionizing radiation released (alpha, beta, gamma, or neutrons). In some respects, radiological weapons offer a unique advantage to healthcare providers. Instruments designed to check for their presence are readily available. This is not the case with chemical and biological weapons.

a. Ionizing (radiation). The hazards associated with radiation exposure will depend in part on the type of ionizing radiation involved. A shield can be used against some forms of radiation (such as alpha particles). Other types are so hazardous that they can even make materials they come in contact with radioactive (as is the case with neutron radiation).

2. Biological agents. These can be considerably more lethal than chemical agents. Because they are obtained from nature, they are far more accessible to the terrorist and relatively easy to produce. They are primarily an inhalation threat and are usually dispersed in their aerosolized form in the 1 to 5 micron size. They have effects that usually go undetected for days, until large numbers of the population begin to present with similar patterns of symptoms. They vary from incapacitating to lethal and fall into three categories:

•Bacteria: Plague, Anthrax, Tularemia

• Viruses: Smallpox, Viral Equine Encephalitis (VEE), Viral Hemorrhagic Fevers (VHF) (such as Ebola)

• Toxins: Botulinum, Ricin, Staphylococcal Enterotoxin B (SEB)

3. Chemical warfare agents. These are generally liquids that are disseminated as vapors or aerosols. They vary in persistence (ease of evaporation), have an onset time of a few seconds to hours, and are designed to irritate, incapacitate, injure, or kill. The chemical agents present an inhalation and dermal threat and are classified according to physiological effects.

Nerve Agents	Tabun, Sarin, Soman, VX
Cyanide	Hydrogen Cyanide, Cyanogen Chloride
Pulmonary Intoxicants	Phosgene, Chlorine
Miscellaneous	Ammonia
Vesicants	Mustard, Lewisite
Riot Control	Mace ®, Pepper Spray
Incapacitating Agents	BZ

F. Need for Preparedness

A 1995 NBC exercise in New York City determined that the first 100 emergency responders to arrive on the scene would be "killed" because they were not adequately prepared or trained to deal with this type of situation. In Los Angeles, a similar exercise left doctors admitting that "victims" would have seriously contaminated their hospitals. Similar results occurred in a 1997 exercise in Salt Lake City.

In 1996, the "Defense Against Weapons of Mass Destruction Act," more commonly known as the Nunn-Lugar-Domenici legislation, was passed. This bill recognizes the lack of preparedness of most first responders and provides the necessary funding and authority to train cities to respond safely to acts of terrorism.

II. NBC "DELTA" PLANNING CONSIDERATIONS

A. Background

Future terrorist attacks can hopefully be prevented by the work of law enforcement officials and various government agencies. Preparing to respond to this type of catastrophe is very similar to contingency planning undertaken for other types of manmade or natural disasters, as long as the unique characteristics of a terrorist attack are considered (as discussed earlier). Pro-active and integrated planning, coordination, training, and realistic drills will allow each community to respond to these events in an organized, efficient manner, using available Federal, State, and Local resources.

Successful disaster planning requires hospital personnel to be familiar with the ICS, standard operating procedures (SOPs), triage, and PPE. A disaster plan, however, is only as effective as the assumptions on which it is based. Unfortunately, most medical disaster planning initiatives are based on incorrect assumptions and misconceptions. As a result, many disaster plans prove to be ineffective in actual use.

In the hospital, disaster planning has traditionally been compartmentalized depending on the type of disaster. For example, one plan is created for external disasters, another for HAZMAT accidents, and a completely different plan for natural disasters. The end result is often a confusing, cumbersome, disaster-planning document that is unfamiliar to hospital personnel. Proficient disaster planning requires a unified, standardized, "all-hazards approach" to disasters that are likely to occur in the community.

B. Disaster Planning Misconceptions

Misconception #1: Disaster planning requires large mobilization of resources. Traditionally, medical disaster planning has focused on the need to rapidly mobilize large numbers of resources (physicians, nurses, support personnel, supplies, etc.) to care for multiple trauma victims, numerous displaced individuals, and the widespread devastation that seemingly accompanies a disaster. This occurs, in part, on the perception that disasters are emergencies that exceed or overwhelm the resources available in a given area or community. In fact, one of the biggest challenges in a disaster is managing these resources.

When a "real" disaster occurs (manmade or natural), large numbers of hospital personnel will arrive at the emergency department (ED) to offer assistance, often without being requested. Unfortunately, many of these "volunteers" are members of the medical staff who never participated in any previous disaster drills. These individuals typically are unfamiliar with routine ED policies and procedures, have never used or seen the disaster triage tagging system, and are uncomfortable receiving orders from the chain of command implemented during the disaster. The end result is a loss of control over the disaster event. The responding personnel function independently without any unified direction or control. Response to an NBC terrorism event would be further confused if these responders lacked NBC training.
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In reality, most problems in disaster response are not caused by a shortage of medical resources, but rather from failures to coordinate their distribution and utilization. This is most evident in the flow of casualties from the disaster area to the closest hospital. The closest hospital typically receives the greatest numbers of patients, while other hospitals in the area receive few, if any, disaster victims. In 29 recent mass casualty disasters, an average of 67 percent of the victims were treated at one hospital. Those hospitals not receiving victims had over 20 percent of their beds vacant. Using the lessons learned from this experience enabled the ICS to prevent overwhelming the closest hospitals after the 1996 bombing in Centennial Olympic Park.

Misconception #2: Most of the medical care for disaster victims is provided by pre-hospital personnel. During the planning process, many hospitals incorrectly assume that all of the disaster victims will arrive by ambulance after being triaged, decontaminated, and stabilized at the scene by pre-hospital personnel. Because of this assumption, hospitals are often caught unprepared for the number of patients that arrive by private vehicle who have never received any form of pre-hospital care. Most of these individuals will only have minor injuries, arrive unannounced to the closest ED, and will consume most of the bed space and personnel prior to the arrival of the most critical patients by EMS.

This lack of pre-hospital care is also the norm in HAZMAT accidents. During such events, only 18.5 percent of victims are treated at the scene of the incident, while 64 percent are transported to the hospital for definitive care. This general trend held true in the Tokyo sarin attack and should be expected if a major domestic NBC attack occurs. Hospitals, which often depend on fire personnel to perform patient decontamination while failing to train their own ED personnel in this procedure, place their hospital staff and facility at risk for secondary contamination. Although fire personnel are well versed in decontamination, they are often busy at the site of the release and unable to come to the hospital to perform this procedure. In addition, their training does not include antidote administration or airway management.

Misconception #3: A disaster plan equals disaster planning. All hospitals have disaster plans, but merely having this required document does not equate to preparedness. Most disaster plans fail for the following reasons:

- Very few disaster planners have actual experience since disasters are uncommon events.

Generic, off-the-shelf disaster plans lack input from local, responsible personnel. No training is provided, the plan is not realistically exercised, and therefore, the plan is not familiar to responsible personnel.

- Coordination with various responding agencies and departments and supporting tertiary medical centers is lacking or not adequately addressed. A disaster crosses all political, geographical, and functional jurisdictional borders.

Apathy is a major impediment to disaster preparedness. Any effective disaster preparedness program should devote considerable efforts towards motivational issues, or it will be doomed to failure. This lack of motivation occurs because disasters are low probability events, the disaster planning process typically lacks administrative support, and disaster drills are infrequently given appropriate emphasis.

C. Staff Protection

During a HAZMAT accident or incident involving NBC weapons, hospital personnel must be protected from the risk of personal injury. These individuals must be provided PPE and decontamination equipment and trained in its use.

1. Personal Protective Equipment. PPE refers to the use of materials that will protect the eyes, lungs, and skin of the responder. The use of appropriate levels of PPE are mandated by federal statutes from the Occupational Safety and Health Administration (OSHA), the National Institute of Safety and Health (NIOSH), and the EPA. In addition, the Joint Commission for the Accreditation of Health Organizations (JCAHO) has adopted these rules and will evaluate hospitals to make certain that they are followed.

All persons designated to be part of the emergency response to an NBC agent incident must have access to appropriate PPE and training for the use of these materials. This includes police, fire fighters, Hazardous Materials Response Teams (HMRTs), emergency medical technicians (EMTs), paramedics, nurses, physicians, and hospital employees.
Level A PPE is required for exposures in the area of chemical release (the hot zone), if air concentrations exceed those that are immediately dangerous to life and health (IDLH). This level of protection mandates a self-contained breathing apparatus (SCBA), is fully encapsulating, and is resistant to liquid and vapor penetration. This level of PPE is difficult to wear for long periods of time, and the rescuer is exposed to a claustrophobic environment with risk of heat stress.

Level B PPE is required for chemicals or substances that pose a potential inhalation hazard. This suit offers less protection than Level A, but when coupled with an SCBA or supplied air respirator (SAR), it provides adequate vapor and liquid protection. This is the level of protection necessary for hospital personnel, who are involved in the decontamination operation.

Level C PPE should be used when the atmospheric contaminants have been identified (that is, nerve or blister agents), concentrations measured, and an air purifying respirator is appropriate and available to remove the contaminants of interest. This clothing is protective for liquid and vapor. An air purifying respirator is one in which ambient air is passed through a filter element or canister that removes gaseous or particulate contaminants. There are two basic types of air-purifying respirators. The first is a negative-pressure respirator in which the air is pulled across the filter by inspiration. The second is a positive-pressure, powered air purifying respirator (PAPR) with a battery-operated blower that pushes ambient air through a filter and then into a mask.

OSHA mandates training in the use of protective equipment. At a minimum, all individuals who might be at risk for chemical contamination must receive Awareness Level training. Training to the Operations Level is required of personnel who will be donning PPE, decontaminating patients, and overseeing the decontamination operation.

2. Decontamination. Decontamination is the physical removal of harmful substances from victims, equipment, and supplies of a HAZMAT or NBC attack. It should be performed whenever there is a risk of secondary exposure from a hazardous substance. Failure to adequately "decon" NBC victims could not only increase the number and severity of casualties, but could also cripple medical response to a terrorist event.

Various methods for performing decontamination are available (such as mechanical removal, absorption, degradation, and dilution), with dilution being the most applicable to the hospital environment. Showering with large quantities of water dilutes the offending agent, thus reducing the patient's skin contamination load. The water is contained in a collection system for future disposal, if necessary.

Some chemicals and biological agents can be neutralized. Sodium Hypochlorite (household bleach), in proper dilution, is a common neutralizing agent for chemical and biological warfare agents. Surface nuclear contamination can be removed with soap and water.

Decontamination should be performed in a safe location adjacent to the scene of exposure. Victims should be decontaminated before transport to a receiving facility.

At the hospital, showers may be located either inside (permanent) or outside the ED (portable), in the ambulance bay or near the ED entrance. Permanent decontamination rooms inside the facility must be equipped with negative ventilation equipment to prevent facility contamination. Portable decontamination equipment allows for outside

decontamination and offers the advantage of mobility while being less expensive than equipping an indoor decontamination room.

Hospitals should have the capacity to safely assess and treat at least one patient exposed to a hazardous chemical. This requires Level B PPE with positive air pressure respirators, OSHA Operations Level training, policies and procedures for the use of decontamination equipment, and a disaster plan that utilizes hospital security to control access to the hospital.

III. MEDICAL NBC "DELTA" PLANNING CONSIDERATIONS

A. Historical Challenges

The goal of EMS and hospital personnel is to respond to a disaster in a rapid, efficient, and coordinated manner to save lives, preserve health, and minimize injuries. However, confusion over roles and responsibilities, poor communication, lack of planning, suboptimal training, and a lack of hospital integration into community disaster planning have been major contributors to problems seen in many previous disasters. These problems, if not addressed, would certainly frustrate response to an act of NBC terrorism.

B. Disaster Epidemiology

Most patients arrive at the hospital within 1.5 hours of the disaster, many with only minor injuries. Arrival of these patients most often is uncoordinated (that is, they may arrive on foot, or by car, bus, ambulance or personal vehicles, and usually not in any order of injury severity). Patients usually go to the closest hospital, regardless of the level of emergency care capability, and will overwhelm one hospital, leaving other hospitals with very few disaster casualties. The medical care component of the disaster usually is over in a few hours, well before the arrival of state or federal resources. As a result, local communities need to be self-sufficient in any type of disaster.

Disasters disrupt the "normal" delivery of healthcare to the population by adding a large burden of acute injuries and illnesses. While the disaster response occurs, the medical needs of the general population do not diminish. In fact, the stress of the disaster combined with the dangers posed to citizens involved in the rescue only serves to exacerbate the situation.

C. Psychogenic Casualties

Disasters have a tremendous emotional and psychological impact on victims and rescuers. Post-traumatic stress disorders have the potential to impact the functional capacity of these individuals for years. Disaster planning must include critical incident stress management programs and other psychological services for these affected individuals. These services need to be available and provided early in the course of the disaster. This will help healthcare providers to vent their feelings and provide a mechanism to identify individuals in need of further counseling.

D. Public Relations and the Media

The media is always present at every disaster, working to keep the public informed throughout the event. These individuals first will seek out an authoritative source for information. If this source is not available or the desired information is not forthcoming, the media then will talk with anyone to get a story. By doing so, the media are more likely to get unqualified and inaccurate information leading them to make, not surprisingly, erroneous conclusions that will only complicate the situation. The media, therefore, must be given frequent and reliable information from a credible source so they can accurately report the details of the disaster. Managed appropriately, the media can serve a useful role in allaying fears and rumors and informing the public about specific safety procedures to follow. The information provided to the media must be selective to balance the public "right to know" against the individual victim's right to privacy.

E. Handling of the Dead

Terrorist use of NBC weapons could result in large numbers of dead victims who require special handling procedures. These victims may be contaminated, and their clothing along with any embedded foreign material will be needed as evidence. In the case of a biological warfare attack (especially anthrax), disposition of the body will require specific decontamination procedures. Temporary morgues may be required.

IV. Disaster Planning

A. Readiness Phase

In preparing to respond effectively to an NBC disaster, hospitals must take an inventory of their current capabilities. Hospital personnel must be made aware of the overall disaster plan, which must be acted out realistically in disaster drills. The plan must address how increased numbers of patients will be triaged, decontaminated, and treated. If training, equipment, or supplies are lacking in specific areas (such as decontamination, PPE, antidotes, data tracking, etc.), these deficiencies must be rectified.

B. Planning Phase

1. Develop strategies to overcome resistance to preparedness. Incorporate all responsible individuals and agencies into the planning process. Keep the plan simple and cost effective and assign tasks to individuals that parallel their normal daily responsibilities. Plan for problems that are most likely to occur in any disaster, and develop policies and procedures to address them. Examples include:

- Communication problems between the triage area, the ED, and the command center

- Coordination and sharing of information

- Security, traffic control, hospital, and ED access
- Congestion in treatment areas by hospital personnel
- Proper identification of authorized personnel
- Triage, victim tracking, and decontamination
- Information needs of families, law enforcement, and the media
- Staff rotation to minimize fatigue and stress

Stress management.

2. Participate in joint planning. EMS, law enforcement, and hospital disaster planners need to work together in developing an organized approach to disasters and mass casualty events. To be successful, pre-hospital and hospital disaster plans need to be effectively integrated into the community disaster plan. This planning may be best accomplished through existing committees, such as the Local Emergency Planning Committee (LEPC), or in association with the local fire chief and local emergency management officials.

3. Develop mutual aid agreements. Hospitals and EMS providers should jointly develop mutual aid agreements concerning patient transfers during a mass casualty situation. Anticipating that the closest hospitals to the incident will see the majority of casualties, EMS should transport the less injured patients to outlying medical facilities to help ensure adequate medical care to all disaster victims.

4. Develop policies and procedures. Formulate policies and procedures that enable the safe identification, decontamination, and treatment of contaminated victims.

5. Acquire necessary equipment. Purchase PPE and decontamination equipment and train frequently utilizing this equipment to protect the safety of the hospital provider. All training and materials used must conform to OSHA standards and regulations. Routine protective gear (such as goggles, surgical facemask, gloves, and gown) may not offer sufficient protection in all circumstances (that is, nerve agent attack). Levels of PPE will be determined by the local jurisdiction.

6. Stockpile antidotes. Develop adequate caches of antidotes that are readily available to quickly and accurately treat victims of a chemical attack. EDs should maintain basic treatment guidelines for the types of NBC events likely to be seen. Summarizing this information on treatment cards or posters helps to regularly reinforce the information while streamlining the delivery of medical care.

7. Take measures to ensure your hospital remains viable during a disaster. Develop a plan to ensure security of the hospital during a terrorist or disaster event. Emergency

backup phone and communication systems need to be in place. Identifying alternative sites for providing medical care to patients with minor problems will help ensure continual access to healthcare.

C. Recovery Phase. During recovery, the focus of disaster response shifts away from the acute injuries and illnesses caused by the disaster to the everyday needs of the general population. The impacted population may have increased needs for medication, shelter, food, water, clothing, and emotional support that must be addressed. Hospitals must also contend with the emotional needs and fatigue of their own personnel.

V. KEY POINTS

Hospitals represent a vital disaster resource to local communities. After an NBC terrorist attack, all victims will be taken to EDs regardless of the institution's level of preparedness. It is essential that hospitals develop an understanding regarding the consequences of a terrorist attack and apply this information to their disaster preparedness activities.

Hospitals must be prepared to expand their scope of services in order to treat victims of an NBC weapon attack. This will require hospitals to stockpile certain antidotes, provide various levels of PPE, and commit to training appropriate staff. Special teams may need to be assembled that have unique knowledge and training in NBC weapons, decontamination, and antidote therapy. In some circumstances, these individuals may be called upon to interact with local response teams [such as HMRTs and metropolitan medical strike teams (MMSTs)] at the incident site. Together, the roles and responsibilities of these emergency care providers fall under the auspices of the ICS (Operations Branch).

Effective disaster planning requires coordinating existing resources in an efficient manner. This is best accomplished when disaster planners concentrate on the disaster planning process rather than solely on the development of a disaster plan. The approach to disaster planning for terrorist events is very similar to the planning process for other types of natural or man-made disasters. Coordination approaches to planning, training, and exercises will ensure an effective and integrated planning effort.

CHEMICAL AGENTS

I. INTRODUCTION

A. Background

Hospitals represent a vital disaster resource to local communities. After a terrorist attack, victims - either on their own or by emergency vehicles - will go to emergency departments regardless of the level of preparedness of the medical facility. It is essential that hospitals develop an awareness and operational level of understanding regarding the consequences of a terrorist attack.

B. Historical Perspective

Germany first utilized chemical warfare agents during World War I (WWI) at Ypres, Belgium, in the late afternoon of April 22, 1915. In that attack, the Germans released 168 tons of chlorine; and allies claimed that 5,000 troops were killed, but this was probably an inflated number for propaganda purposes.

In July 1917, the Germans first used sulfur mustard, again in Ypres, Belgium. This persistent agent (it does not evaporate readily and stays on terrain for a long time) caused many casualties but most survived as it damaged eyes, airways, and skin. The success of sulfur mustard as a weapon was because of its persistency in the battlefield, its delayed clinical effects, and its ability to cause casualties.

Overall, chemical agents caused large numbers of casualties in WW I, but killed fewer than 5 percent of these casualties, excluding those from Russia.

After World War II (WWII), Egypt allegedly used chemicals in Yemen, and Iraq used them against Iran and the Iraqi Kurds. On June 27, 1994, the Aum Shinrikyo, a well-funded Japanese religious cult, initiated the use of chemical warfare agent terrorism in Japan. The nerve agent GB, or sarin, was manufactured in a secret facility in Japan and was first released in Matsumoto, Japan with about 280 casualties and 7 deaths. Nine months later, on March 20, 1995, sarin was released in five separate subway cars in downtown Tokyo. There were 12 deaths, hundreds injured (a few dozen seriously), and thousands who sought medical care. Some of the first responders were contaminated, a few of the hospital staff suffered exposure to the chemical, possibly due to vapor off-gassing from clothing. Some of the responders required admission to hospitals.

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C. Terrorist Threat

Former Senator Sam Nunn has stated that "…there is no greater threat to this nation's, and indeed the world's, national security than the illicit spread of these devices."

(October 31, 1995) Chemicals make an excellent weapon for the terrorist. The effects can be immediate or delayed, the chemicals can be delivered by a variety of routes, the cost is low, the chemicals are available, and they are easily transported. Most countries are poorly prepared to deal with a terrorist chemical attack. Many terrorist groups receive financial support for training and military weapons from wealthy foreign governments. Iran has an extensive chemical warfare agent capability and has supported Hezbollah, Hamas, and the Palestinian Islamic Jihad. Local community planning should encompass the possibility of a military-style attack. Transport of phosgene, cyanide, anhydrous ammonia, and chlorine is a daily event in most large cities. Rail cars, which can contain up to 30,000 gallons of chemical, are susceptible to a terrorist pipe bomb. Tear gas, which is available in many stores, can be used by a terrorist to cause panic and fear. So dual use of industrial chemicals as agents for a terrorist attack is a real concern.

D. Current Preparedness

The U.S. government has determined that it is not prepared to deal with the consequences of a chemical attack. As a result, plans have been made to train first responders, EMS personnel, fire fighters, law enforcement, and hospital personnel regarding the proper approach to the management of the chemically contaminated patient.

Although many communities in the U.S. have Hazardous Materials Response Teams (HMRTs), these teams have not been universally trained and their equipment has not been standardized. Training has not yet extended to an awareness of the effects of chemical warfare agents. Many teams may not be readily available to perform search and rescue after a terrorist attack. Also, it may be difficult initially for first responders to recognize that injuries are due to a chemical release. Thus, first responders (police, fire, EMS) and hospital providers may become secondary victims.

Chemical cross-contamination of ambulances and hospitals due to a lack of preparedness, PPE, and decontamination capability could cripple the capacity of the local pre-hospital and hospital system. One patient exposed to a hazardous chemical can contaminate a transport vehicle and temporarily close a hospital emergency department (ED).

Few hospital providers or EMS personnel have access to or have been trained to use PPE. For communities to be prepared, law enforcement, fire, EMS, and hospital personnel must develop policies and procedures to address:

The safe identification, decontamination, treatment, and transport of the chemically contaminated victim.

The procurement of adequate caches of antidotes that are readily available to quickly and accurately treat the victims of a chemical agent attack.

The purchase of appropriate PPE and decontamination equipment, with frequent training utilizing this gear to protect the safety of the first responder and hospital caregiver.

E. Chemical Warfare Agents

Chemical warfare agents are hazardous chemicals that have been designed for use by the military to irritate, incapacitate, injure, or kill. Some have local effects on the eyes, skin, or airways (riot control agents, chlorine), some have only systemic effects (hydrogen cyanide), and some have both (nerve agents and vesicants).
The types of chemical warfare agents listed on this slide are the ones we will focus on during this module. Incapacitating agents, chemical agents that might cause psycological effects, might also be used, but these will not be discussed.
Some of the chemical warfare agents are said to have characteristic odors, such as those listed here. However, these are not adequate warning properties for the purpose of protecting yourself against adverse health effects associated with exposure, as we shall see in a moment.
Suppose you are work one day, when you receive a call that a chemical has been released in a single subway station at 10 AM after morning rush hour. EMS is dispatched to the scene. One patient is dead, two patients are unconscious but still breathing, and five patients are weak and complaining of eye pain, but are still ambulatory. Other subway travelers are frightened and begin running from the scene. Two emergency medical technicians (EMTs) from another ambulance arrive first, and while bag-mask ventilating two of the seriously injured victims, become weak, short of breath, and complain of dim vision and eye pain.

- What do you think is going on?
- What actions should be taken immediately?
- Who should be contacted?
- What options are available for:
-
  - Protecting the first responders?
  - Treatment for the victim?
  - Stabilization and transport?

II. NERVE AGENTS

You probably recognized that in Scenario #1, the EMTs were exposed to nerve agent vapor. The nerve agents are tabun (GA), sarin (GB), soman (GD), and VX. Nerve agents are the most toxic of all the weaponized military agents. These agents can cause sudden loss of consciousness, seizures, apnea, and death. GB, or sarin, is one of the more commonly stockpiled nerve agents, and it may be inhaled as a vapor, or cause toxic effects by contact with the skin in the liquid form. VX is mainly a liquid skin hazard at normal ambient temperatures. These chemicals are easily absorbed through the skin, eyes, or lungs. The diagnosis of a nerve agent poisoned casualty must be made clinically. There usually is not time for laboratory confirmation. Nerve agents (and similar substances) inhibit cholinesterase, an enzyme present in tissues and blood; there is a laboratory test to determine its activity in blood. The nerve agents belong to a class of chemicals called organophosphates and have a physiological effect similar to that of many insecticides commonly found in the community, such as malathion, diazinon, and chlorpyrifos.

A. Characteristics

Nerve agents are stored and transported in the liquid state. The initial diagnosis of nerve agent exposure is made by the presenting clinical signs and symptoms, with confirmation by laboratory tests or onsite detection. The G-agents such as sarin (GB), soman (GD), and tabun (GA) are volatile liquids at normal temperatures; although, the most volatile, sarin, evaporates at about the same rate as water. In liquid form, the G-agents can be absorbed through the skin and eyes; vapor is absorbed by inhalation and through the eye, but not through the skin unless the concentration of vapors is extremely high. The G-agents liquids are more effective in penetrating skin when the chemical is trapped between the skin and clothes. GB rapidly evaporates and is considered to be a non-persistent agent; that is, it does not remain on terrain or objects very long, whereas VX is persistent due to its low volatility. Though liquid at normal temperatures, VX has the consistency of motor oil, seldom presents a vapor hazard. VX is much more toxic (100 to 150 times) than sarin when on the skin because sarin evaporates from the skin surface.

B. Toxicity and Effects

Nerves communicate with muscles, organs, and other nerves by releasing chemicals or neurotransmitters at their connection site (synapse). One of the most common neurotransmitters is acetylcholine (ACh), which is released and collects at the receptor site stimulating the end organ to respond and produce a variety of effects: muscle contractions, gland secretion, and nerve-to-nerve conduction. When a nerve impulse reaches the synapse, ACh is released from the nerve ending and diffuses across the synaptic cleft to combine with receptor sites on the next nerve, and the electrical message continues.

To stop further stimulation of the nerve, ACh is rapidly broken down by acetylcholinesterase (AChE), producing choline, acetic acid, and the regenerated enzyme. Thus, a "check and balance" system prevents the accumulation of ACh and the resultant over-stimulation of nerves, muscles, and glands. The term "nerve agents" refers to chemicals that produce biological effects by inhibiting the enzyme AChE, thus allowing the neurotransmitter ACh to accumulate. Included among the "nerve agents" are some drugs (such as physostigmine and pyridostigmine) and some insecticides (Sevin®, malathion, and related insecticides). These compounds cause the same biological effects as the nerve agents developed for military use, but the latter are more than a hundred-fold more potent. As a result of inhibition of AChE, the neurotransmitter ACh accumulates to over-stimulate the organs it normally stimulates in the portion of the nervous system. This causes hyperactivity in these organs. These are all innervated by the cholinergic portion of the nervous system and have muscarinic receptors, nicotinic receptors, or a combination (central nervous system and cardiovascular system). The clinical effects of nerve agents are in organs that have cholinergic receptors. These are divided into muscarinic sites and nicotininc sites. Organs with muscarinic receptors include smooth muscles and exocrine glands; those with nicotinic sites are skeletal muscles and pre-ganglionic fibers.

Over-stimulation at muscarinic sites will increase secretions. The victim may experience increased saliva, tearing, runny nose, phlegm in the airways, and sweating.

Accumulated acetylcholine. The accumulated ACh also causes pinpoint pupils (miosis), bronchoconstriction (shortness of breath), and hyperactivity of the gastrointestinal tract (nausea, vomiting, and diarrhea).

Nicotinic receptors. There are also nicotinic receptors which are stimulated by ACh. Over-stimulation causes skeletal muscle fasciculations, twitching, cramping, weakness, and finally paralysis. There is also stimulation of the pre-ganglionic fibers which may contribute to hypertension and tachycardia.
The combination of pinpoint pupils and muscle fasciculations is the most reliable clinical evidence of organophospate (nerve agent) poisoning.

Cardiovascular. Bradyarrhythmias, heart block, tachyarrhythmias (sinus tachycardia), and ventricular arrhythmias (ventricular tachycardia and ventricular fibrillation) may occur, but most disappear once the antidote is given.

Central nervous system. Acute severe effects include loss of consciousness, seizures, and apnea. Effects from a mild exposure include nervousness, fatigue, minor memory disturbances, irritability, and other minor psychological symptoms. The latter, whether caused by a severe or mild exposure, might linger for 4 to 6 weeks after exposure before resolving.

C. Forms of Nerve Agent Exposure

1. Vapor. After exposure to a small amount of vapor to a volatile nerve agent like GB, the most common effects are miosis (often with pain in the eye or head, complaints of dim or blurred vision, or possibly nausea and vomiting), conjunctival injection, rhinorrhea, and some degree of bronchoconstriction and bronchosecretions (with associated complaints of "a tight chest" or "shortness of breath"). After exposure to a large amount of vapor, the casualty will almost immediately lose consciousness, and seizures will begin within 1 to 2 minutes. After several minutes of seizing, apnea and flaccid paralysis will occur.
If the exposure has been small and a victim is removed from the area of the exposure, shortness of breath may improve. In this situation, the removal of clothing is often adequate decontamination. Effects begin within a minute or so after vapor exposure and generally do not worsen significantly once the casualty is out of the contamination. Peak effects usually occur within the first 5 minutes following exposure.

2. Liquid. Persistent agents like VX present more of a liquid contact hazard. The onset of effects following exposure can be delayed from 10 minutes to 18 hours after contact with the agent, depending on the dose. Initially, small fasciculations and diaphoresis might be noted on the skin at the site of the droplet. If the droplet is larger, the effects might be gastrointestinal(GI), including nausea, vomiting, and diarrhea. If a droplet the size of a Lethal Dose 50 percent (LD_{50}) (10 mg for VX as shown on this penny) is on the skin, sudden loss of consciousness, seizures, flaccid paralysis, and apnea will occur within minutes.

D. Treatment

1. Self-protection. The process of treating nerve agent casualties may be divided into several components. The first and most important concept is to protect yourself. Although liquid contaminated casualties are unlikely to present directly to the hospital ED prior to decontamination by emergency responders, medical personnel should always protect themselves by assuming the presence of liquid contamination, unless a clear "vapor-only" exposure history is obtained. Whenever possible, areas of liquid contamination should be decontaminated prior to patient handling to minimize spread of contamination and cross-contamination or other providers.

2. Decontamination. In the immediate aftermath of the sarin nerve agent attack in Tokyo, over 650 patients presented to St. Luke's Hospital within several hours after the release of sarin. With high numbers of vapor-exposed patients presenting to a medical facility under these conditions, minimum decontamination should include removal of patient's clothing and jewelry. This will hopefully prevent secondary chemical exposures to hospital personnel due to vapor off-gassing. If the patient has been exposed to liquid nerve agent (such as after spraying or an explosion), survivors will require complete decontamination with soap and water, or possibly a decontaminant such as sodium hypochlorite (household bleach) at the scene prior to evacuation. This household bleach should be diluted 1:10 (nine parts water and one part bleach) before being applied to the skin, then rinsed off with water. Patients arriving at the ED with an unclear exposure history who are symptomatic from nerve agent exposure should be fully decontaminated with soap and water or sodium hypochlorite before entering treatment areas.

3. Airway and ventilation. Establishment of a patent airway is essential for the survival of the severely exposed patient. Severely intoxicated patients will die if aggressive airway management is not quickly available. With large numbers of victims, rapid scene and resource assessment will influence triage decisions regarding interventional therapy. Because of the intense bronchoconstriction and secretions associated with nerve agent exposure, effective ventilation may not be initially possible due to high airway resistance (50 to 70 cm H_2O). Adequate atrophinization will reverse these muscarininc effects; **therefore, atropine should be administered before other measures are attempted.** Endotracheal intubation, followed by positive pressure ventilation with a bag-valve mask, should be performed as quickly as possible. Periodic suctioning of secretions will help to improve ventilation and air exchange. Patients with seizures and respiratory failure can be saved with immediate and adequate intervention.

4. Antidote administration. Three medications are used to treat the signs and symptoms of nerve agent intoxication: atropine sulfate, pralidoxime chloride, and diazepam. The general indications for use of these antidotes will be presented first, followed by a discussion of their use in the treatment of mild, moderate, or severe nerve agent intoxication.

a) Atropine. Atropine works to block the effect of the accumulated neurotransmitter, ACh, at muscarinic sites. The more ACh at the sites, the more atropine is required to

counteract its effects. Atropine can be administered intravenously (IV), intramuscularly (IM), or endotracheally (ET). Parenteral atropine will reverse the muscarininc effects such as rhinorrhea, salivation, sweating, bronchoconstriction, bronchorrhea, nausea, vomiting, and diarrhea. Atropine will not reverse nicotinic effects such as fasciculations, twitching, or muscle weakness. Nor are miosis or ciliary body spasm reversed by parenteral atropine; relief of intractable pain in or around the eye requires the installation of 1 percent homatropine or atropine topically. Although the IV route of atropine administration is preferred when treating system effects, this should be avoided in hypoxic nerve agent casualties. Because studies have documented the occurrence of ventricular fibrillation when atropine is administered IV to hypoxic animals, atropine should be administered IM in these patients.

The initial parenteral dose of atropine is 2 to 6 mg in the adult, with subsequent doses titrated to the severity of the nerve agent signs and symptoms. Treatment for chemical nerve agent exposure might require up to 10 to 20 mg of atropine. (In patients poisoned with insecticides, over 2,000 to 3,000 mg of atropine might be necessary.) When atropine therapy exceeds the amount necessary to reverse the effect of the cholinergic hyperstimulation, it may cause toxicity manifested by dry mouth, flushing, and diminished sweating, but this would be extremely unlikely in a patient poisoned by an organophosphate (OP) compound. Side effects in unexposed people (not poisoned by OP compounds) include mydriasis, blurred vision, tachycardia, and diminished secretions. The latter (i.e., loss of sweating) may be of concern in a hot environment.

Atropine dosing is guided by the patient's clinical presentation and should be given until secretions are dry or drying and ventilation becomes less labored. When shortness of breath, increased airway resistance, and secretions have abated and the patient is breathing easier, he or she has received enough atropine. Heart rate and pupillary size, ordinarily accurate reflections of atropine dosing, are not useful for clinical monitoring after nerve agent exposure.

b) Pralidoxime chloride (2-PAMCl). This is an antidote that can specifically break the bond between the nerve agent and the enzyme AChE and remove the agent. This will free the enzyme, making it once again available to break down ACh. Clinically, this will decrease muscle twitching, improve muscle strength, and allow the patient to breathe better; however, it has little effect on the muscarinic effects described previously. The bond between the enzyme and the nerve agent can **age,** is a process by which the enzyme and agent become irreversibly bound. This means that if the antidote is not administered within 4 to 6 hours after sarin exposure (the aging time for the sarin-enzyme complex) or within 60 hours after VX exposure (the aging time for the VX-enzyme complex), the bond becomes permanent. Usually, there is plenty of time to treat patients with 2-PAMCl after exposure to nerve agents with the exception of GD. The soman-enzyme complex ages in about 2 minutes.

MARK I kit. Atropine and pralidoxime chloride (2-PAMCl), are used by the military in autoinjectors which together are called the MARK I kit. The atropine autoinjector contains 2 milligrams (mg) of atropine and is administered IM by pressing the end of the device onto the thigh. A spring pushes the needle into the muscle and causes the atropine to be injected. This device causes atropine to be absorbed more rapidly than when

administered by a conventional needle and syringe. The other autoinjector contains 600 mg of 2-PAMCl. The Food and Drug Administration (FDA) has approved the autoinjectors, but local protocols will determine their use in the field.

c) Diazepam. Seizures are treated with benzodiazepines such as diazepam. These medications can be used IV or via an autoinjector which contains 10 mg of diazepam. Some authorities recommend treating all severely exposed patients with diazepam whether they are convulsing or not. If three atropine MARK I kits are required initially because of the victim's clinical presentation, diazepam should be administered immediately thereafter.

5. Treatment regimen.

a) Latent effects: Asymptomatic victims who present to the ED alleging exposure to nerve agents should be considered potentially exposed, triaged for other injuries, and observed for up to 1 hour if a vapor exposure is alleged, or up to 18 hours if a liquid exposure is possible (or if the exposure history is uncertain).

b) Mild effects. The presence of miosis and rhinorrhea requires observation only. If the victim is suffering from airway effects (shortness of breath, chest tightness, and profuse airway secretions) that are not improving, then treat with 2 mg of atropine IM or IV, or with the MARK I kit. Supplemental oxygenation will be needed only in those patients with pulmonary or cardiac disease. IM atropine dosing can be repeated at 5 to 10 minute intervals as needed. [Note: Patients with pinpoint pupils may have severe light sensitivity and pain, but only require reassurance since these symptoms will resolve. At the hospital, these patients should be given a topical eye medication (homatropine) only for relief of severe pain in the eye(s) or head because the drug causes blurred vision. This may be done if miosis occurs as part of moderate or severe systemic effects as well.]
If there are mild effects from liquid exposure (localized sweating and fasciculations at the site of liquid contact), give 600 mg 2-PAMCl IM (MARK I kit) or 1 gram (gm) 2-PAMCl IV slowly over 20 to 30 minutes.

c) Moderate vapor exposure. Be more aggressive with moderate vapor exposures. Symptoms will include those for mild exposures with more severe respiratory distress and may be accompanied by muscular weakness and possibly GI effects (vomiting and diarrhea). Initial dose for these patients is 1 or 2 MARK I kits containing a total of 2 mg atropine and 600 mg 2-PAMCl. Treatment may also be given IV, with 2 to 4 mg atropine given IV push, and 1 gram of 2-PAMCl given by IV infusion slowly.
This dosing can be followed by repeat doses of 2 mg of atropine at 5 to 10 minute intervals as needed, and 600 mg of 2-PAMCl for a total of 1,800 mg 2-PAMCl with the MARK I kit IM (or 1 gm 2-PAMCl IV for a total of three doses at hourly intervals). Antidotes can also be given IV, with atropine given in 2 mg increments at 5 to 10 minute intervals, and 2-PAMCl given by infusion, 1 gm over 20 to 30 minutes, for a total of 3 doses at hourly intervals.
For moderate toxicity several hours after liquid exposure, 2 mg of atropine and 600 mg 2-PAMCl should be given initially. Repeated doses of atropine and 2-PAMCl may also

be necessary. Oxygen may be needed in those with cardiac or pulmonary disease who have severe breathing difficulty, but generally, it is not necessary.

The treatment of nerve agent exposures can be summarized in the table

Treatment Protocol for Nerve Agent

Exposure	Clinical	Treatment
Latent	None	None, observe for 1 hour with vapor, for 18 hours if liquid or unknown exposure
Mild	Miosis with dim and/or blurred vision, rhinorrhea, shortness of breath.	One MARK I kit or Atropine 2 mg IM/IV and 2-PAMCl 600 mg IM or 1 gm IV.
Moderate	Above, but more severe; or vomiting and diarrhea	One MARK I kit or Atropine 2 mg IM/IV and 2-PAMCl 600 mg IM or 1 gm IV. Repeat 2 mg Atropine at 5-10 minute intervals until agent effects diminish.
Severe	Above plus Flaccid paralysis, respiratory distress, cyanosis, seizures; severe effects in two or more organ systems.	Oxygen, bag mask, intubate after three MARK I kits or Atropine 6 mg IM and 2-PAMCl 1800 mg IM or 1 gm 2-PAMCl IV repeated twice at hourly intervals. Repeat 2 mg Atropine at 3-5 minute intervals until atropinized. Diazepam for seizures.

6. Special Age-Related Antidote Dosing Considerations

a) Atropine. Certain members of the population may be more sensitive to atropine. These include infants, young children, and the elderly. Pediatric experts have divided the age groups for IM administration of atropine. These doses may be repeated as clinically indicated.

Category	Age	Dose
Infant	0 to 2 years	0.5 mg single dose
Child	2 to 10 years	1.0 mg single dose
Adolescent	Young adult	2.0 mg single dose

If atropine is to be given IV, then the dose is 0.02 mg/kilogram (kg) for infants up through young adults.
If only standard MARK I kits are available, the use of a 2 mg atropine autoinjector can be used, but infants and small children are at risk of being injured by the autoinjector needle. The most significant adverse effect of high dose atropine in the younger patient is the inhibition of sweating.
Elderly: In the frail or medically compromised adult, use a 1 mg dose and repeat as necessary.

b) Pralidoxime chloride. No data are available for 2-PAMCl use in nerve agent exposed children. The standard IV dose for a patient from an infant to a 70-kg person is 15 mg/kg, with the dose repeated twice at hourly intervals. Above 70 kg, the dose should be a total of 1 gm, repeated twice at hourly intervals as necessary. For IM use, the doses should be :

Weight	Dose
< 20 kg	15 mg/kg
> 20 kg	600 mg autoinjector

These may be adjusted according to subsequent clinical presentation.
Elderly: If frail, hypertensive, or with renal disease, use one-half the usual adult dose of 2-PAMCl (7.5 mg/kg IV).
If hypertension becomes significant during the administration of the 2-PAMCl, treat with IV phentolamine as follows:

Adult: 5 mg IV
Child: 1 mg IV

c) Diazepam. Recommended pediatric doses:

Infants > 30 days to age 5 0.2 to 0.5 mg/kg IV slowly every 2 to 5 minutes to maximum dose of 5 mg
Children > 5 years 1 mg IV every 2 to 5 minutes to maximum dose of 10 mg

III. BLISTER AGENTS OR VESICANTS

Vesicants cause blistering. They may be plant, animal, chemical, or sunlight. Those to be discussed are the chemical warfare vesicants, namely sulfur mustard and Lewisite. A close relative of sulfur mustard, nitrogen mustard, was the first cancer chemotherapeutic agent.

A. Sulfur Mustard

Sulfur mustard is a vesicant or blister agent, which is a vapor inhalation and liquid contact hazard. Mustard causes injury to the eyes, skin, airways, and some internal organs. This chemical warfare agent has a delayed action, and exposure to it may result in blisters on the skin, temporary blindness, and respiratory distress. More extensive injury can result in death because of respiratory failure from airways injury, or sepsis as a result of bone marrow damage, decrease in white blood cells, and an impaired immune system. There is no specific therapy. Mustard is absorbed and causes chemical cellular damage within 1 to 2 minutes, but clinical effects do not begin for hours. There is no immediate pain, there is no immediate skin discoloration, and there is no immediate eye irritation. Hours later, the casualty realizes that he or she has been exposed and presents to the ED for evaluation and treatment. The onset time for clinical effects ranges from 2 to 48 hours; most commonly is between 4 and 8 hours. Despite years of research, the exact mechanism by which mustard damages cells is unknown. It alkylates DNA and clings to proteins and other cellular components. The end result is DNA damage and cellular death. The injury is very similar to that produced by radiation, and mustard is a radiomimetic agent. As mentioned earlier, the three organ systems directly affected topically by mustard are the eyes, skin, and respiratory tract. There is a spectrum of eye involvement. The eye lesion, after a small exposure to mustard, may consist only of mild conjunctivitis. A larger exposure will produce a more severe conjunctivitis, lid inflammation and edema, blepharospasm, and corneal roughening. These casualties will be unable to open their eyes and will be temporarily without sight. A larger exposure, particularly if by liquid, may produce corneal opacification, corneal ulceration, or corneal perforation. Miosis is sometimes observed after mustard exposure and is thought to be due to cholinergic effects.

Skin effects begin hours after exposure with erythema as shown in this Iranian casualty (accompanied by burning and itching), followed later by the development of small vesicles; later, these small vesicles coalesce to form blisters. The size and depth of the lesion depends on the amount of exposure and whether exposure was by vapor or liquid. Coagulation necrosis extending into the dermis may develop under blisters caused by liquid, such as those blisters shown on this photograph of a worker's hand several days after he was accidentally exposed to liquid mustard.

Mustard damages the mucosa or lining of the airways. This damage begins in the upper airways and descends in a dose-dependent manner to the smallest bonchiole. After a small exposure or initially after a large exposure, there may be epistaxis, sinus discomfort, and a mild to moderate pharyngitis with a hacking cough. After a moderate exposure, or later after a large exposure, there may be laryngitis with voice loss and a

productive cough. If the exposure is large, the agent reaches the smallest airways to cause dyspnea and productive cough, as the mustard will damage not only the mucosa, but the underlying musculature as well. At this stage, there may be hemorrhagic pulmonary edema around the bronchiloes, as depicted above, but otherwise, pulmonary edema is rare.
Gastrointestinal effects within the first 24 hours following exposure include nausea and vomiting. These effects are thought to be in part due to cholinergic stimulation. There may be some added effects of mustard on the GI tract from the swallowed tracheal secretions.
Gastrointestinal effects seen after 3 to 5 days are thought to be due to tissue destruction in the abdomen.
Absorption of significant amounts of mustard produces damage to and death of the stem or precursor cells of the bone marrow. If this occurs, the white blood cell count, after an initial increase because of the toxic exposure, starts decreasing on about the third or fourth day after exposure and continues downward until recovery begins. If the amount of mustard absorbed is quite large, there is no recovery and the cell count will reach zero. Survival usually does not occur when this happens. The red blood cells and platelets also decline following the white blood cells. The absence of these cells increases susceptibility to infection and contributes to death.
Treatment is largely supportive since there is no antidote for the effects of sulfur mustard. Decontamination should consist of physical removal of any residual agent by whatever means available. The casualties should remove all clothing, rings, and jewelry. Skin decontamination may be performed with soap and water, or one-tenth strength household bleach (0.5 percent sodium hypochlorite). Contact time with the bleach should be brief, and the area should be flushed with water afterwards. Decontamination must be done as quickly as possible since cellular damage occurs in as little as two minutes.
Decontamination of the casualty at the ED 30 minutes or more after contact with mustard will not change the clinical course of the patient's illness, but is effective in preventing cross-contamination of providers.

1. Skin. Soothing creams or lotions might be effective for irritation and itching. Large blisters should be unroofed and denuded areas irrigated several times a day followed by a topical antibiotic (Silvadene, etc.) will prevent skin bacterial superinfection. Oral pain medications will likely be necessary. Fluid requirements should be assessed, but fluid replacement is **less** than is usually required with thermal burns. Care must be taken not to overhydrate ("burn formula resuscitation"). Rarely will burns be full thickness requiring skin grafting.

2. Eyes. A discussion of the therapy for ocular exposures is best begun by repeating the observation that mustard fixes to tissues within the first several minutes after exposure. Gentle irrigation with saline or water during this time period will be helpful. Aggressive attempts to pry apart severely painful, blepharospastic eyelids to accomplish an irrigation 30 minutes or more after exposure is of dubious value, since the damage has been done and the agent has evaporated or has been absorbed. With severe eye injuries, homatropine or other mydriatics should be applied topically to prevent synechiae formation. Topical analgesics may be used for initial examination. However, oral pain

medication is preferred to topical analgesics, which may allow damage to the cornea and delay healing. Topical antibiotics should be applied several times a day and vaseline should be applied to lid edges to prevent them from adhering. Many ophthalmologists feel that the application of topical steroids within the first 24 hours, but not after, might be of benefit. Early involvement of an ophthalmologist is advised, and a visual acuity should be obtained before treatment measures are instituted.

3. Lungs. Upper or minor airway symptoms (sore throat, non-productive cough, hoarseness) may be relieved by steam inhalation and cough suppressants. The initial chemical pneumonitis should be treated in the usual manner; however, antibiotics should not be used until an organism is demonstrated, which usually occurs between the third and fifth day post-exposure. A patient with severe airway effects will benefit from oxygen and assisted ventilation, particularly positive end expiratory pressure (PEEP) or continuous positive airway pressure (CPAP). Intubation should be performed if there are signs of severe upper airway involvement, and it should be done early before laryngeal spasm or edema makes it difficult. Bronchodilators may be needed; if they fail to relieve bronchospasm, steroids may be tried. Steroids, however, are of questionable benefit otherwise.

B. Lewisite

Lewisite is a vesicant that has been stockpiled militarily, but there have been few human exposures to the chemical. Lewisite is rapidly absorbed by the eyes, skin, and lungs and produces blisters similar to sulfur mustard. In contrast to sulfur mustard, however, **Lewisite is highly irritating on initial exposure.** It also produces visible lesions more quickly. Unlike mustard, it does not damage the bone marrow.

1. Skin. Lewisite causes greater skin damage than sulfur mustard. A gray area of dead skin can progress to blisters and severe tissue necrosis and sloughing.

2. Lungs. Since lewisite causes immediate irritation to the nose and sinuses, and effort by the victim to evacuate the area of contamination may prevent more severe lung damage. Pseudomembrane formation is common.

3. Cardiovascular system. Lewisite causes increased capillary permeability, leading to volume depletion and hepatic and renal injury.

4. Treatment. Decontamination with hypochlorite (bleach) or soap and water will remove most of the chemical if it is performed quickly after contamination. An antidote is available, called British anti-Lewisite (dimercaprol or BAL), which can be used IM to reduce the systemic effects of the vesicant. As it is administered parenterally, BAL has no effect on Lewisite damage to the skin and eyes.

IV. CYANIDE

Scenario #2: After the victims have been cleared from the subway station, a man is found unconscious in a bathroom in a building adjacent to the subway station. Next to

the man, who is still alive, is a small box which contains a white powder and a device that dripped liquid onto the powder.
The victim is removed from the bathroom and intubated by EMS first responders. The gentleman who removed the patient from the bathroom is nauseated, short of breath, and anxious. Now, he is beginning to feel better.

Cyanide
Cyanide is a chemical that is widely utilized, manufactured, and transported in the U.S. Over 300,000 tons of cyanide are produced annually. It is used in printing, agriculture, photography, and in the manufacture of paper and plastics. It is also a combustion product of burning synthetic materials. Rail cars with 30,000-gallon tanks of cyanide represent potential transportation and terrorist threats.

1. Characteristics. Cyanide is stored and utilized militarily in the liquid or solid state. It may have an odor of bitter almonds, but the ability to smell the cyanide exists for only 40 percent of the population. Large amounts of the chemical are needed to cause death.
Types of Cyanide. The three types of cyanide that may be encountered are hydrogen cyanide (AC), cyanogen chloride (CK), and cyanide salts. The term "cyanide" refers to the anion, CN, or to its acidic form, hydrocyanic acid (HCN). Cyanogen (CN_2) is formed by the oxidation of cyanide anions. However, the term cyanogen has also come to mean a substance that forms cyanide upon metabolism and produces the biological effects of free cyanide. Cyanogen chloride is a pungent, heavier-than-air vapor which can cause irritation of the eyes, nose, and throat. This is in distinct contrast to hydrogen cyanide, which has no irritant properties. Cyanide salts (for example, NaCN) are compounds that dissociate into the cyanide anion (CN^-) and a cation (Na^+). Salts are most dangerous following ingestion; onset of action is slower and more prolonged. Cyanide salts generate hydrogen cyanide gas on contact with a strong acid such as sulfuric acid.

2. Toxicity. Cyanide exists normally in human tissues and is usually metabolized by hepatic enzymes (rhodanese) into thiocyanante, which is excreted in the urine.
Under normal conditions, the cyanide anion is attracted to iron in the ferric state (Fe^{+++}). In the mitochondrion of the human cell, cytochrome A3 in the cytochrome oxidase complex contains Fe^{+++}. Cyanide is bound to cytochrome A3 and thus inhibits the effect of the cytochrome oxidase. This enzyme complex is responsible for the utilization of oxygen within the cell. In the presence of cyanide, even though there is plenty of dissolved oxygen in the blood, the cells cannot use oxygen. As a result, cells must utilize anaerobic metabolism, or the creation of energy without the benefit of oxygen, which causes a severe lactic acidosis. When cells cannot get enough energy, they die. Cells in the brain and heart are affected initially.
Acute cyanide poisoning occurs after inhaling the agent, but may also occur after drinking solutions of cyanide (it is sometimes used with suicidal intent) or by skin contact with large amounts of liquid cyanide. After inhalation of a low concentration, the patient may become anxious, will often hyperventilate, and typically develops a headache with dizziness and vomiting. Skin color may initially be flushed but may also be normal or

cyanotic. A "cherry-red" skin color is characteristic of cyanide, but this is not always seen.
In about 15 seconds after inhaling a large amount of cyanide, victims become anxious and start to hyperventilate. Thirty seconds after exposure, the patient may begin to convulse. In 3 to 5 minutes, breathing ceases. Asystole or cessation of heart activity occurs in 6 to 10 minutes, followed by death. The patient may have normal sized or dilated pupils. Death can occur within 8 minutes of exposure. If a patient is exposed to a low concentration of vapor and removed from the source of the cyanide, the symptoms should not progress.

3. Laboratory. A normal oxygen saturation may be noted when using a pulse oximeter, despite the fact that the patient is in severe respiratory distress. There is high oxygen content to venous blood because oxygen is not extracted from arterial blood by cells. A metabolic (lactic) acidosis may also be present from the lack of oxygen to the tissues. Cyanide toxicity can be measured at the hospital by checking serum cyanide concentrations. These values may, however, only be available after a delay of several hours.

4. Clinical Management. Patients who have inhaled significant doses of cyanide must be rapidly treated with appropriate antidotes to prevent brain damage. Cyanide is also attracted to iron (Fe^{+++}) in a form of hemoglobin called methemoglobin. In fact, cyanide will preferentially leave the cytochrome oxidase enzyme in the cell and bind to the circulating methemoglobin. Drugs such as amyl nitrite and sodium nitrite, which are found in the cyanide treatment kit, increase blood concentrations of methemoglobin and are antidotal. Adding sodium thiosulfate completes the detoxification process.
Patients should be treated with IV saline for hydration and sodium bicarbonate and hyperventilation (after intubation) for the metabolic acidosis. Oxygenation should be maintained with high-flow oxygen by mask or by endotracheal tube. Monitor and treat significant arrhythmias.

5. The Pasadena Cyanide Antidote Kit. This kit (formerly known as the Lily Cyanide Kit) contains amyl nitrite, sodium nitrite, and sodium thiosulfate.

a) Amyl nitrite. Amyl nitrite is available in perles which are broken and placed in either a gauze bandage or in the bag-mask and inhaled for 15 seconds, then taken away for 15 seconds (although, if the patient is breathing, he probably does not need the antidote). This is the initial step in antidote therapy. Amyl nitrite is a methemoglobin former and reduces the elevated total peripheral resistance caused by the acidosis and cyanide. This should be used only until the IV drugs can be given. This will cause orthostatic hypotension. However, if the patient can stand, he or she does not need the antidote.

b) Sodium nitrite. Sodium nitrite is a strong methemoglobin former that is available for IV use in a dose of 300 mg in 10 cc. This dose is injected over 2 to 4 minutes and has a potential side effect of orthostatic hypotension. Normal saline infusion and supine posture can help to correct the hypotension. However, if the patient can stand, they do not need the sodium nitrite. The pediatric dosage is 0.2 cc/kg, not to exceed 10 cc.

c) Sodium thiosulfate. This compound is a co-factor for the enzyme rhodanese for detoxification (to change cyanide to a form that can be excreted by the kidneys). The drug is administered in a 50-cc ampule (12.5 gms) over 5 minutes IV.

d) Treatment regimen.
(1) Remove from area of exposure and remove clothing.
(2) Mild exposure: If conscious and breathing, give O_2, IV fluids. Observe, no antidotes necessary.
(3) Severe exposure: If unconscious, whether breathing or not, give O_2, and bag-mask ventilate with 100 percent O_2. Cardiac monitor. Oxygen saturation may or may not be normal.
(i) Amyl nitrite: Crush into a 4 x 4 piece of gauze and place over face (however, if the patient is breathing, they probably do not need the drug) or in a bag mask. Add another ampule every few minutes. Give only until IV drugs are available.
(ii) When IV established, give sodium nitrite 300 mg (10 cc ampule) over 5 minutes for adults. For children, use 0.2 to 0.3 mg/kg or 6 to 9 cc/kg of the 3 percent solution. Watch for orthostatic hypotension (however, if the patient can stand, they do not need this).
(iii) Then, sodium thiosulfate 12.5 gms (50 cc) IV. For children, use 0.4 mg/kg or 1/65 cc/kg of the 25 percent solution.

V. PULMONARY INTOXICANTS

Scenario #3. A worker at a rail yard located near a post office has called police. It appears that a rail car has exploded. Two workers are killed. EMS arrives and evacuates the area. Three injured survivors walked from the scene and have a mild cough. One survivor wants to go home to feed his cat. The Rail company has notified the local HMRT that the rail car contained phosgene.

How should these patients be managed?

- What precautions should be taken?

The injured arrive in the ED, and other than a mild cough, the patients are stable.

- How long should the patients be observed?

What should be monitored?

Pulmonary intoxicants cause severe life-threatening lung injury after inhalation. These effects are generally delayed several hours after exposure. Treatment is usually supportive and may require advanced intensive care techniques including intubation, use of a mechanical ventilator in the hospital, and PEEP breathing. Pulmonary intoxicants included with this group are phosgene and chlorine.

A. Phosgene

Phosgene is widely used today in the manufacturing of dyes, coal tar, pesticides, and pharmaceuticals. It was widely used in WWI until mustard was introduced on the battlefield.
The Bhopal, India disaster of 1984 at the Union Carbide plant involved the release of 50,000 pounds of methylisocyanate. This chemical is composed of phosgene and methylamine. There were 150,000 people affected, 10,000 severely injured, and 3,300 killed. The effects of the release were thought to be due to a combination of isocyanate and phosgene.

1. Characteristics. Phosgene has a characteristic odor of freshly mown hay and is four times heavier than air. It is a gas above 47 degrees F, and is principally a hazard by inhalation.

2. Toxicity and clinical effects. The primary damage from phosgene is from the carbonyl group. Phosgene dissolves slowly in water to form carbon dioxide and hydrochloric acid (HCl). In contact with the upper airways, HCl causes a transient irritation of the eyes, nose, sinuses, and throat. It can also irritate the upper airway and bronchi, causing a dry cough.

3. Treatment. Phosgene penetrates poorly into the airways due to its poor water solubility. There is a symptom-free period of 2 to 24 hours. Over the first several hours, the carbonyl group from the phosgene attacks the surface of the alveolar capillaries. Eventually, this causes the leakage of serum from the capillaries in the lung into the alveolar septi. The fluid fills the tissues, causing severe hypoxia and apnea. As the fluid leaks into the alveoli, massive amounts of fluid (up to 1 liter per hour) pour out of the circulation. The patient develops a severe non-cardiogenic pulmonary edema as depicted in this picture.
The leakage of fluid in the lungs causes volume depletion. Although the patient clinically looks like traditional heart failure, DO NOT USE DIURETICS! These patients are volume depleted. Treat hypotension with fluids. These patients may require intubation and PEEP.
In the hospital, the initial examination of a patient, symptomatic or not, should include - as a minimum - auscultation, chest x-ray, and arterial blood gases. If the victim develops severe dyspnea due to upper airway irritation, early intubation should be considered to manage oxygen delivery and to prevent laryngeal spasm. The airway should be suctioned frequently to remove secretions. According to some authorities, antibiotic use should be guided by gram stain and culture results. Another source recommends prophylactic antibiotics, as autopsy studies show uniform evidence of pneumonia and bronchitis. Ventilator management, PEEP, and oxygen administration might require consultation with a pulmonologist. Fluid hydration may be necessary to treat the hypotension, bradycardia, or impending renal failure. Diuretics such as Lasix are contraindicated because of the hypotension and the noncardiac nature of the non-cardiogenic pulmonary edema. Standard bronchodilators will usually control bronchospasm, but if not, steroids may be needed for this purpose. Routine steroid use is controversial, but they seemed to offer some efficacy after the Bhopal tragedy. Once the patient recovers, there should be little residual pulmonary effect.

B. Chlorine

Chlorine is a significant irritant to the eyes and respiratory tract. It is widely used in the manufacture of chemicals, plastics, and paper and is commonly used in swimming pools and laboratories. Industrial exposures have produced large numbers of injuries.

1. Characteristics. Chlorine is a greenish-yellow gas that has a characteristic pungent odor that is irritating to the nasal mucosa. It is transported as a liquid and is less alkaline than ammonia.

2. Toxicity. Chlorine injures cells by reacting with water, producing hydrochloric acid (irritating) and free oxygen radicals (attack cells). It is toxic to any body surface including the eyes, skin, respiratory tract, and GI tract. Chlorine gas is 30 times more irritating to the respiratory mucosa than HCl.
In seconds after the exposure, there are symptoms of irritation to the eyes, nose, and throat. This is followed by irritation of the respiratory tract with coughing, shortness of breath, wheezing, chest pain, and sputum production. Initial respiratory distress is followed in 12 to 24 hours by a noncardiogenic pulmonary edema. Sudden death is usually due to severe hypoxia and cardiac arrest.

3. Treatment. Move exposed victims away from the source of exposure. If the victim has no complaints, probably no treatment will be necessary.
Toxicity to skin and eyes should be treated with copious flushing with water. Irritation of the respiratory tract is treated with oxygen, cool mist to moisten the damaged mucosa, and bronchodilators to resolve bronchospasm.
Intubation, mechanical ventilation, and assessment of hydration may be required. Bronchoscopy may be useful to remove mucosal plugs.

VI. Ammonia

Ammonia is a colorless, highly water-soluble, alkaline gas that has a pungent odor. It is widely used industrially in the U.S. with over 500,000 workers potentially exposed annually. It is used as an agricultural fertilizer and is used in the manufacture of explosives, dyes, and plastics.

1. Characteristics. Ammonia is rapidly absorbed by mucosal surfaces and causes damage to the eyes, oral cavity, throat, and lungs. When mixed with water, it forms a corrosive agent, ammonium hydroxide (NH_4OH) which causes considerable damage in the form of liquefaction necrosis. Due to its high water solubility, it penetrates rapidly into tissue. Household ammonia generally has a pH less than 12 and generally causes limited damage to eyes or mucosa. Anhydrous ammonia is an industrial chemical that has a very high pH and is extremely corrosive. This is a picture of the effects of ammonia when splashed in the eye; the areas of fluoroscein staining indicate corneal erosions.

2. Clinical Signs.
a) Eyes. Initially, ammonia causes burning, tearing, and severe pain. It has a tremendous capacity to penetrate the eye, causing corneal opacification and lens damage leading to cataract formation. This slide depicts the corneal opacification and scarring which can occur from ammonia splashes to the eye.

b) Lungs. Mild exposure causes cough, shortness of breath, chest pain, wheezing, and laryngitis. Higher exposure can cause hypoxia, chemical pneumonia (pneumonitis), and hemorrhage. This will gradually improve over 72 hours. If the patient survives the first 24 hours, recovery is probable.

c) Skin. Pain, blister formation, and possibly deep burns similar to frostbite can occur.

d) Gastrointestinal. If ammonia is ingested, severe mouth pain, cough, abdominal pain, nausea, and vomiting can occur. Severe edema of lips and mouth is seen. The patient should be examined to make certain that laryngeal irritation does not cause airway obstruction. Esophageal stricture and perforation is common.

3. Treatment. After the patient has been removed from the area of exposure, decontamination should be started immediately in the field. Remove all clothing and wash with soap and large amounts of water for 15 to 20 minutes. Cover burns with a sterile dressing. The eyes should be irrigated continuously with water. A Morgan-lens device and topical analgesics will enable continuous eye irrigation therapy. Both of these items should be considered part of an antidote/equipment cache. Slit lamp exam after fluorescein staining will reveal the ocular injury.
Damage to the lungs is common after inhaling anhydrous ammonia, often resulting in non-cardiogenic pulmonary edema. The victims may quickly develop shortness of breath and laryngeal swelling, so **early intubation should be considered to protect the airway.**

VII. RIOT CONTROL AGENTS

Irritating agents, lacrimators (chemicals that stimulate lacrimal glands to produce tears), riot control agents, and tear gas are synonyms for a group of aerosol-dispersed chemicals that produce eye, nose, mouth, skin, and respiratory tract irritation. This class of chemical agents causes involuntary eye closing due to irritation. For police, this is an effective weapon as it can disable an assailant. It is widely used in the civilian arena for self-protection. The deleterious effect is usually transient (about 30 minutes after exposure).
Riot Control Agents include:

- CN (Mace®)
- CS (tear gas)
- OC (oleoresin capsicum or pepper spray)

Adamsite is an irritating and vomiting agent that acts very similar to CN and CS, but the onset of its effects is delayed for minutes compared to seconds for CN and CS. In addition, adamsite does not cause skin irritation.

A. Characteristics

Riot control agents are solids. They are sometimes dispersed in a solution that is aerosolized and can be dispersed from grenade or bomb. Some police SWAT teams have small grenades that contain rubber pellets and/or CS. CN (the active ingredient in Mace®) has caused several deaths from pulmonary injury. CS is less toxic. Capsicum, or pepper spray, is derived from the oleoresin capsicum in certain peppers. It is also used as an over-the-counter topical pain medication.

B. Clinical Presentation and Effects

Pain, burning, and irritations of exposed mucous membranes comprise the clinical picture.

1. Eyes. Blepharospasm, or spasm of the muscle that causes eyelid closure, causes very transient "blindness" due to the closed eyelids. Vision, however, is not impaired once the eyes are opened. They cause tearing conjunctival injection, and redness.

2. Upper airway (mouth, nose). Can cause nasal discharge, sneezing, burning.

3. Lower airway (lungs, bronchi). Can cause coughing, shortness of breath, and chest tightness. Bronchospasm and wheezing can occur for hours after the exposure.

4. Skin. All can cause burning and redness, and it is claimed that O. capsicum has less tendency to cause dermatitis. After exposure to large amounts of CS and CN, the onset of a more severe dermatitis with erythema and blisters may be delayed for 4 to 6 hours after exposure. These more severe effects occur under high temperature conditions with high humidity and large amounts of agent contacting the skin.

5. Heart. Increased blood pressure and heart rate are probably a response to anxiety.

C. Medical Management

The effects of the riot control agents will rarely last longer than 30 minutes, although the skin redness or erythema may last longer. In fact, most people will not seek medical care. Less than 1 percent will have eye, airway, or skin complaints severe enough to be medically assessed. A higher percentage might seek care because of anxiety and panic. There is no antidote available for these agents. Treatment is supportive and directed towards alleviating symptoms which are not usually severe.

1. Eyes. Should be irrigated copiously with water or saline. Remove contact lenses. Utilize slit lamp exam to make certain that all solid particle foreign bodies are removed. Follow-up with ophthalmologist is recommended.

2. Lungs. Treat wheezing with bronchodilators or steroids if standard bronchodilators fail. Oxygen therapy if indicated. Most symptoms should be maximal within an hour or two.

3. Skin. Most skin exposures require little more than reassurance. With prolonged pain, decontaminate with soap and water or a solution containing a carbonate and/or a bicarbonate. Do NOT use bleach. The delayed onset dermatitis should be managed with frequent irrigation and soothing ointments or creams.

VIII. CHEMICAL AGENT DETECTION

Recognition of a chemical attack is initially based on clinical criteria. This assessment can be augmented by chemical detection. The current technology for the detection of a chemical agent release is limited. Each of the various types of detectors currently available has specific qualifying factors.
In general, chemical agent detection equipment can help to determine the need for and level of PPE required to protect first responders and hospital personnel. It can be used to certify that the victim has been adequately decontaminated to prevent cross-contamination, and as an early warning device to notify authorities and the community of a chemical agent release. Chemical detectors do not, however, replace the need for an adequately supervised decontamination process.

IX. KEY POINTS

To safely respond to a chemical terrorist attack, local communities must develop resources, protocols, and policies that will enable a safe and appropriate response. Patients exposed to hazardous chemicals require evacuation, life-saving intervention (ABCs), antidote therapy if available, and decontamination. First responders and medical personnel must be trained and equipped to safely function in a chemically contaminated environment.

There are a number of chemicals that can cause serious injury to victims. This lecture has assembled a few of the most common irritants and potentially lethal liquids, gases, and solids that might be used by a terrorist.

Community planning requires multi-agency exercises and training. Contact the local emergency manager, law enforcement, fire, and EMS leadership to arrange community-wide drills. Develop a statewide network that can assist in the event of an attack.

Antidote Therapy for Chemical Weapons Attacks

Chemical	Antidot	Decontaminatio (Includes removal of clothing)	Other
Nerve Agen	Atropine, 2-	Soap & Water (S&W) sodium hypochlorite (Clorox)	Diazepam (Valium)
Sulfur Mustard	None,	S&W, Clorox	Delayed onset, debride bullae, pulmonary care, pseudomembranes, watch for
Lewisite	BAL,	S&W, Clorox	Acute onset, treat acidosis, volume depletion, pseudomembrane
Cyanide	Methemoglobin Amyl Nitrite, Sodium Nitrite, Na Thiosulfate	S&W	Na bicarbonate, O2, fluids, treat acidosis, sudden loss of
Phosgen	None,	S&W	IVF, monitor volume, O2, early intubation, steroids, watch for pulmonary edema
PFIB	None,	-	Monitor O2, watch for pulmonary edema
Ammonia	None,	Irrigate eyes, S&W	Milk, bronchodilators, Silvadene, GI endoscopy, watch for mediastinitis, liquefaction
Chlorine	None,	Irrigate eyes, S&W	Bronchodilators, intubation,
CN (Mace)	None	S&W, eye irrigation	Remove foreign body from eye, watch for
CS (Tear gas)	None	S&W, eye irrigation	Eye foreign body,
Oleoresin capsicum	None	S&W, eye irrigation	From chili pepper, dermatitis, eye injury

BIOLOGICAL AGENTS

Case Study

Emergency departments (EDs) always seem busier during a full moon, despite evidence to the contrary. Tonight was no exception. Over a 6-hour period, it seemed that almost half of the patients presented with similar complaints of high fever, cough, shortness of breath, and generalized ill feeling. Five young, previously healthy individuals required intubation and mechanical ventilation for severe respiratory distress. Strangely, most of the patients knew each other from work and none of their family members were suffering similar symptoms. At 11 p.m., the only other community hospital in the area went on diversion because all of their intensive care unit (ICU) beds were full and their need for mechanical ventilators was at a critical level. The public health officer on call was not aware of any recent infectious outbreak.

I. BIOLOGICAL WEAPONS - INTRODUCTION

A. Background

Biological agents are the oldest of the nuclear, biological, and chemical (NBC) triad and have been used by governments in warfare for over 2,500 years. In addition, use of toxins by non-state sponsored groups is not new. Terrorist groups have included these weapons into their armamentarium. These agents are more deadly on a compound per weight basis than chemical agents. Biologic agents occur naturally in the environment, but many have been refined and could be made more resistant in laboratories. Some of the poisons (toxins) produced by bacteria have been developed for use as BWs. Most notable is the botulinum toxin which can produce death after being ingested or inhaled in minute quantities.

By definition, biological warfare is the use of microorganisms (bacteria, viruses, and fungi) or toxins (poisons produced by living organisms) to produce death or disease in humans, animals, and plants. Few healthcare providers have been trained to recognize or treat victims of a biological agent attack. Nonetheless, the principles of detection, personal protection, infection control, and treatment parallels the standard approach to any natural disease outbreak (such as influenza, Rift Valley Fever, meningitis, and hepatitis).

B. Current Perspectives

During the 1991 Persian Gulf War, the threat of biological warfare against American soldiers increased the public awareness of the possibility of a potential biological attack against US cities. The reality of this threat gained credence in 1996 when two high ranking Iraqi military officials revealed that during the war, Iraq had produced and prepared to use 19,000 liters (L) of botulinum toxin and 8,500 L of anthrax.

Rogue nations and terrorist organizations have shown a strong interest in the use of BWs because these weapons are inexpensive to produce, difficult to monitor, and can produce illness and death in large numbers of people. Inhaling 40,000 spores of anthrax (enough to fit on the head of a pin) would result in a death rate of 95 percent. BWs can be produced with minimal startup equipment and supplies and can easily be introduced into a building's ventilation system, released into a busy shopping center from an aerosolized can, or placed into the food system with little or no warning. These agents, called the

"poor man's nuclear bomb," are appealing to countries with limited resources. About 15 to 25 countries are currently suspected of possessing BWs, five of which have histories of belligerent militant behavior (Iran, Iraq, Libya, Syria, and North Korea).

II. CHARACTERISTICS OF BIOLOGICAL WEAPONS

A. General Properties

Despite differences between the various types of biological agents, these agents, bacteria, viruses, and toxins share some common characteristics. The primary route of infection or "portal of entry" is by inhalation (pulmonary). Since BWs are nonvolatile (do not evaporate), they must be dispersed in aerosols as 1 to 5 micron size particles (1/30,000 of the diameter of a hair follicle) which may remain suspended in the air for hours (depending on weather conditions). If inhaled, the particles will deposit deep into the terminal air sacs of the lungs, causing disease.

Each of these agents can be easily disseminated from a point source such as industrial sprayers modified to generate the small particle size. The aerosol can also be delivered along a line from a moving generator in an airplane or boat traveling upwind from the intended target. Military weapons have been designed to deliver BW agents in warheads and bomblets.

The ideal BW agent:

1. Can be delivered as an aerosol
2. Has a high disease/infection ratio
3. Maintains viability/infectivity in environment
4. Has a vaccine or other prophylaxis to protect the attacker.

Only a few of the thousands of known microorganisms meet these requirements.

B. Environmental Constraints

BW agents are affected by a number of weather conditions.

1. Sunlight**.** The sun's ultraviolet light helps kill many biological agents.

2. Wind. Winds will spread the biological agents and may contribute to diluting their effectiveness.

3. Temperature. BW agents vary in their sensitivity to extremes of heat and cold. However, most are resistant to freezing.

4. Desiccation. Depending on the degree of desiccation, biological agents may either suffer growth inhibition or be killed.

C. Potential Impact of a Biological Agent Release

Biological warfare agents have the potential for causing widespread illness and death. According to one computer simulation model, if 110 pounds of anthrax spores were sprayed along a 1.5-mile tract upwind from a city with a population of 500,000, approximately 24,000 individuals would die. The psychological impact to the rest of the country and the world would be profound. A recent study published in the Centers for Disease Control (CDCs) Emerging Infectious Diseases Journal estimated the cost of managing a 100,000 case exposure to anthrax spores, could be as high as $26 billion

Outside of a few professional groups and some military personnel, there is a very poor understanding of BWs. Bacteria and viruses are often perceived by the public to be invisible agents that have the ability to attack at will and spread from person to person and against which there are no means of self-protection. In the event of a real or threatened BW attack, many unaffected people will attribute various nonspecific symptoms to toxins or infectious agents. When a BW agent is suspected and a call for assistance is made, the emergency response will cross jurisdictions and may include fire personnel, emergency medical service (EMS), law enforcement, specially trained response teams [Metropolitan Medical Strike Teams (MMSTs), Chemical/Biological Incident Response Force (CBIRF)], federal agencies, public health departments, and various experts in biological warfare.

The public reaction to BW was manifested recently in Washington, D.C. (April 1997) when a package containing a Petri dish labeled with "anthrachs," an apparent misspelling of the deadly bacterial disease, anthrax, and "yersinia" was sent to the international headquarters of B'nai Brith. After being discovered and reported, 108 employees inside the building were "quarantined" for 8 hours, traffic to the area was diverted for miles, and two B'nai Brith employees and 12 firefighters were decontaminated in the streets with a chlorine solution as a precaution (Associated Press 4/25/97).

D. Epidemiological Clues for Identifying a BW Attack

Symptoms that would develop after a BW attack would be delayed and nonspecific, making the initial diagnosis difficult. Healthcare providers should seek a number of clues when trying to identify the cause of an unusual infectious outbreak. A BW attack should be considered if any of the following are present:

1. Large epidemic with unprecedented number of ill or dying.

2. HIV(+)individuals may have first susceptibility ("canary in a coal mine")

3. Particularly high volumes of patients complaining primarily of respiratory symptoms that are severe and are associated with an unprecedented mortality rate.

4. The cause of the infection is unusual or impossible for the particular region (such as the Ebola virus which is rarely seen outside of Africa). The agent may require clinical and laboratory diagnosis.

5. Multiple, yet simultaneous outbreaks.

6. The epidemic is caused by a multi-drug-resistant pathogen, previously unknown.

7. Sick or dead animals of multiple types are encountered.

8. The delivery vehicle for the agent is identified.

9. Prior intelligence reports or claims by aggressors of a BW attack.

After a characteristic incubation period following aerosol exposure, most BW agents present as an initial influenza syndrome with fever, chills, malaise, headache, and myalgia. Some BW agents rapidly develop into a pulmonary syndrome with dyspnea, cyanosis, chest pain, and radiological abnormalities. Liver involvement is indicated by rising liver enzymes, with or without jaundice. Encephalitis may occur with some select viral agents, typified by photophobia, confusion, nuchal rigidity. Maculopapular, vesicular pustular, or ulcerative skin lesions with or without bleeding abnormalities may occur with some agents. Unexplained death or flaccid paralysis may be indicative of the use of biological toxins.
Recognizing these clues or patterns of illness will alert medical personnel to begin interviewing patients and their families to obtain useful epidemiological information for public health officials. Questions asked should focus on the patient's recent history of travel, infectious contacts, employment, and activities over the preceding 3 to 5 days.

III. BACTERIA AS BIOLOGICAL AGENTS

A. General Characteristics

Bacteria are single-celled microorganisms that vary in size and shape depending on the makeup of their cell wall. Cocci are spherical-shaped cells that are approximately 0.5 to 1 micron in size, while bacilli are rod-shaped and vary between 1 and 5 microns. Bacteria are self-sustaining organisms that reproduce without the requirement of a host (other living cell). These organisms contain nuclear material (DNA), cytoplasm, and a cell membrane that functions to keep the cell alive. Viruses, on the other hand, lack these internal components, and therefore, can only reproduce inside the living cells of a host. Some types of bacteria (such as anthrax) can transform themselves into spores which are better able to withstand cold, heat drying, chemicals, and radiation injury from the environment. Spores are dormant, but under the right circumstances can easily germinate.

B. Disease-Producing Properties

Bacteria produces disease in animals and humans by either invading tissues, producing an inflammatory reaction, or by manufacturing toxins which are poisonous to the cells. Many disease-causing bacteria have the capability of doing both. The above photo illustrates right, lower, and middle lobe consolidation due to pneumonic plague.

C. Examples of Bacterial Biological Weapons

1. Anthrax. In early April 1979, an accidental release of anthrax spores occurred at a military compound in Sverdlovsk, in the former Soviet Union, which was identified as a microbiology facility. Inside this military production facility (Military Compound No. 19) workers were weaponizing anthrax by filling warheads and bombs with the agent. Reportedly, while the dayshift workers were drying anthrax and making into a powder, they discovered that the safety air filters had become clogged. They were removed but not replaced by the next shift of workers, causing weapon-grade anthrax spores to be released in the air. Within 10 days, residents living downwind from the compound developed high fever and difficulty breathing, and a large number died. The USSR admitted to only 66 deaths. Animal cases of anthrax occurred 30 miles away. Although the Soviets blamed the deaths on the consumption of contaminated meat, most experts suspected that these fatalities were from inhaling aerosolized anthrax released from the incident. This was confirmed in 1992 when Russian President Boris Yeltsin acknowledged that the incident was in fact a large-scale accident involving the escape of aerosolized anthrax spores.

In December 1990, the Iraqis filled bombs and warheads with botulinum toxin, anthrax, and aflatoxin. In 1991, information was provided to United Nations inspectors about Iraq's offensive biological warfare program. In this report, Iraq admitted to conducting research and development work on anthrax, botulinum toxins, *Clostridium perfringens*, aflatoxins, wheat cover smut, and ricin. In 1996, further disclosures revealed biological agents were tested in various delivery systems, including rockets, aerial bombs, and spray tanks.

a. Microbiology. *Bacillus anthracis* is a gram-positive, rod-shaped organism that becomes infectious when it converts into a spore and enters a host. The spore germinates into a macrophage, which is then transported to regional lymph nodes where local production of toxins causes edema and necrosis of the tissue leading to bacteremia, toxemia, and death. Under normal circumstances, anthrax infections occur in animals, such as sheep, cattle, and horses. Humans may become infected with anthrax if they handle contaminated animal fluids or hides. Cases of anthrax are rarely found in industrialized countries because vaccines are available for both animals and humans.

These are anthrax bacilli in their vegetative form. Under adverse environmental conditions these vegetative forms revert into resistant spores which may be viable and infectious for up to 50 years.

b. Pathogenesis. Anthrax causes disease after inoculation of open or minor wounds, ingestion, or inhalation of the spores. The inhalation route has the highest mortality and is the most likely route of exposure in a BW attack since the spores are easily

disseminated from a spraying device. An untreated skin infection has a mortality rate of 20 percent if septicemia develops, (treated 1 percent) compared to 80 to 90 percent mortality rate when anthrax involves the lungs (inhalation) or intestines (ingestion). Aerosol exposure is the most likely scenario in a terrorist biological attack. Following the inhalation of about $\pm 10^4$ spores, alveolar macrophages engulf the spores and the bacteria become vegetative and are transported to the tracheobronchial nodes. Early symptoms are nonspecific, malaise, nonproductive cough, and/or chest discomfort. Within 1 - 6 days, there is a sudden onset of respiratory distress, dyspnea, stridor, and cyanosis.

Tracheobronchial nodes undergo a necrotizing edematous lymphadenitis, progressing to a mediastinitis and pulmonary edema, with or without a bloody pleural effusion.

50 percent of cases may rapidly develop a concurrent hemorrhagic meningitis with bloody cerebral spinal fluid.

Septicemia, toxic shock, and death occur within 24 to 36 hours.

Following a major attack, it is possible that cutaneous anthrax cases might appear in conjunction with inhalational cases.

Skin lesions appear 1 to 5 days after spores are introduced into skin abrasions. 1 to 2 cm vesicles with regional edema and lymphadenitis. Most patients with small lesions maybe afebrile. The lesions develop into a painless necrotic ulcer with a black eschar base. The ulcer may spontaneously heal within 2 to 3 weeks, however, 20 percent of the cases may develop septicemia and death.

Symptoms are variable and include fever, nausea, vomiting, abdominal pain, bloody diarrhea, and sometimes a rapidly developing ascites. The patient may present with an acute abdomen. Oropharyngeal cases show primary envolvement of the tonsils.

Symptoms begin to develop within 1 to 6 days of exposure and include fever, generalized muscle aches, cough, and fatigue. These symptoms seem to improve over a few days, then patients abruptly develop severe respiratory distress, go into shock, and die within 24 to 36 hours. Physical findings are usually nonspecific. The chest x-ray may reveal a hilar or mediastinal adenopathy or widened mediastinum with or without a bloody pleural effusions, but typically the chest x-ray lacks evidence of infiltrates. Although inhalational anthrax would be the predominate disease following an aerosol attack by terrorists, an entire region would be contaminated and smaller numbers of cutaneous and gastrointestinal cases would be expected.

d. Prevention. No cases of person-to-person transmission of inhalational anthrax have ever been reported. However, cutaneous transmissions have occurred. Universal precautions should be maintained for the duration of illness for cutaneous and inhalation anthrax.

e. Diagnosis and treatment. Definitive diagnosis is made by a gram stain of the blood and blood cultures using routine media. These findings may not be evident until late in the course of the illness. Only the encapsulated bacteria are visualized. Enzyme-linked immuno-specific assay (ELISA) and immunohistology testing may also be helpful to confirm the diagnosis.

Treatment is considered futile in patients who have inhaled anthrax spores and present with severe mediastinitis. Nonetheless, antibiotic therapy should still be considered

appropriate since almost all **naturally** occurring strains are sensitive to erythromycin, chloramphenicol, gentamycin, tetracycline, and ciprofloxacin. At the earliest sign of disease, adult patients should be vaccinated and treated with either intravenous (IV) ciprofloxacin (400 mg IV every 8 to 12 hours) or doxycycline (100 mg every 12 hours), until the patient has received 3 doses of vaccine. Animal testing suggests the disease will re-emerge in exposed persons after antibiotic prophylaxis, if simultaneous vaccination has not been completed. Hemodynamic support and airway management may also be required. If adequate warning of a BW attack is provided, oral prophylaxis with either ciprofloxacin (500 mg po every 12 hours) or doxycycline (100 mg po every 12 hours) should be started and continued for four weeks while patients are started on a vaccination schedule. An FDA licensed vaccine is available from the Michigan Department of Health.

For children, the drug of choice for prophylaxis is penicillin or amoxicillin (20 to 40 mg/kg/day div TID to QID). While naturally occurring anthrax is sensitive to penicillin, a genetically engineered BW agent might be resistant. In penicillin-resistant cases, the drug of choice is doxycycline (2 to 4 mg/kg/day div BID). The discoloration of teeth often associated with doxycycline is typically not seen until after taking six courses of the antibiotic. For IV therapy, penicillin or doxycycline, are considered appropriate choices.

2. Plague.

a. Microbiology. *Yersinia pestis* is a gram-negative, rod-shaped organism that is non-motile and does not sporulate. The organism is killed when exposed to sunlight and temperatures above 72 degrees C for 15 minutes but is resistant to near freezing temperatures and can remain viable in dry sputum, flea feces, and buried bodies. This organism normally causes infection in rodents (rats, mice, ground squirrels). Fleas living on the infected rodent can pass the disease on to humans. All human populations are susceptible to the plague and recovery from the disease may be followed by a temporary immunity.

After skin inoculation, the bacteria is consumed by dermal macrophages that transport the organism to regional lymph nodes and then into the blood to infect secondary organs, such as the lungs, spleen, liver, and brain.

b. Pathogenesis. The plague is spread to humans from either the bite of an infected flea or by inhaling the organism. Infection occurs in three forms: **bubonic**, which involves lymph nodes closest to the bite of infected fleas; **pneumonic**, which is an infection of the lungs; and **septicemia**, which is a generalized infection in the blood from the bacteria escaping through the lymph nodes or lungs. (In 2.5 percent of the cases plague septicemia may develop directly without a clinically apparent lymphadenitis.)

The plague has been the cause of a number of devastating epidemics, including the "Black Death" during the fourteenth century in Europe, which caused the death of over 25 million people. There were 3,000 cases of plague reported worldwide in 1994, and 5 cases in the U.S. in 1996. In 1993, 10 confirmed cases of human plague were reported from New Mexico (six cases), Colorado (two cases), Texas (one case), and Utah (one

case). Seven of these cases were bubonic plague, two were primary septicemia, and one was primary pneumonic. Nine of the ten patients recovered with antibiotic therapy; one patient died. For three patients, the probably mode of transmission was a flea bite, two were infected by domestic cats with visible signs of infection, and the mode of transmission could not be determined in the remaining five cases. **Nearly all fatal plague cases in the U. S. result from delay in seeking treatment of from improper diagnosis** (MMWR Vol. 43, No. 13, 4/8/94.)
Prairie dogs in the southwest U.S. are major reservoirs of plague.
Pneumonic plague occurs within 2 to 3 days of aerosol inhalation of bacilli (from BW weapon or from respiratory droplets from another infected patient). There is a sudden onset of fever, chills, and an influenza-like syndrome followed within 24 hours by the onset of a fulminant pneumonia with hepatocellular damage and systemic toxicity. Coagulation abnormalities are common and severe ecchymosis may occur ("black death"). Oropharangeal primary infections may progress to fulminant pneumonia following endeobronchial aspiration of plague bacilli. This fulminant pneumonia is rapidly followed by systemic toxicity, respiratory failure, and circulatory collapse. Six percent of pneumonia cases have an accompanying meningitis.
"Classical" bubonic plague occurs from the bite of an infected flea. There is initial erythema, fever, and rigors which typically progresses to overt bubo formation in regional lymph nodes. Septicemia normally develops 2 to 10 days later. Buboes may be aspirated, but should not be surgically drained.

c. Signs and symptoms. Two or three days after inhaling the plague organism, the patient will develop high fever, myalgias, chills, headache, cough with bloody sputum, and signs of overwhelming infection (including pneumonia). Chest x-ray may show patchy infiltrates or consolidation, with this pneumonia progressing rapidly, causing dyspnea, stridor, and cyanosis. Eventual respiratory failure, circulatory collapse, and laboratory evidence of disseminated intravascular coagulation (DIC) develop.
Acral gangrene may be a late complication in survivors. The bacterial coagulase gene is temperature sensitive and becomes active at temperatures less than 37° C, (such as at the extremities). Acral gangrene may be a late complication of pneumonic or septicemic plague, and may occur in the finger, toes, earlobes, nose, and penis.
Pneumonic plague may be highly communicable under appropriate climate conditions. For patients with confirmed pneumonic plague, respiratory droplet isolation with precautions against airborne spread is required until sputum cultures are negative. Accidental exposures to health care workers are managed by giving post-exposure tetracycline or doxycycline therapy for a <u>minimum</u> of 7 days.
Vaccine is ineffective against aerosol exposures and is only effective against bubonic plague.

Diagnosis and Treatment. Diagnosis can be made by microscopic identification of the characteristic bipolar-staining, "safety pin"-shaped organism and culturing lymph node aspirates, sputum, or cerebrospinal fluid samples. Immunoassays are also available.There are few fulminant pneumonia's caused by gram negative bacilli other than plague. Respiratory isolation is mandatory for the first 48 hours of treatment. Treatment must be started within 24 hours of the onset of symptoms to impact patient survival. Plague

pneumonia is almost always susceptible to streptomycin (30 mg/kg/day IM divided twice daily for 10 days) and doxycycline (200 mg IV initially, followed by 100 mg twice daily for 10 days). Chloramphenicol is used for plague meningitis (50 - 75 mg/kg/day in 6 hourly divided doses). Care is otherwise supportive. Incision and drainage (I and D) of the lymph nodes is not recommended. After antibiotic treatment is started, the swollen tissue typically resolves on its own. A vaccine is available which is effective in preventing bubonic plague, but not the pneumonic form.

For pediatric victims, doxycycline, or trimethoprim/sulfamethoxazole, should be considered for prophylaxis. Gentamicin or streptomycin, are appropriate for IV therapy if child is over 1 yr of age. Chloramphenicol induced aplastic anemia is very rare and dose dependent.

If adequate warning of a BW attack with the plague is provided, prophylaxis with doxycycline may be effective. Enamel staining of teeth by doxycycline or tetracycline only occurs with multiple repeated courses.

3. Tularemia.

a. Microbiology. *Francisella tularensis* is a non-motile, gram-negative cocco-bacillus that typically causes disease in animals (rabbit fever, deer fly fever). It was discovered in 1911, in Tulane county, California. Humans can become infected by either handling diseased animal fluids or by being bitten by infected deer flies, mosquitoes, or ticks. Spores are not formed, but the organism can remain viable for weeks in a number of mediums and can be easily spread by aerosol. After infection occurs, the bacteria spread to regional LNs and the reticuloendothelial system (RES), leading to bacteremia with secondary spread to the lungs and other organs.
F. tularensis is resistant for months to freezing temperatures, but is easily killed by heat and disinfectants. Almost 100 percent of those exposed to tularemia will become infected, but only about 5 percent of treated victims from the naturally occurring disease die. The case fatality rate with all forms of untreated typhoidal disease is approximately 35 percent. Recovery is followed by permanent immunity.

b. Pathogenesis. Tularemia occurs in many forms, but inhaling as few as 10 to 50 organisms will cause disease verses the 100 million organisms that need to be ingested to produce disease. Inhalation is the most deadly route of exposure. Ingestion or inoculation, through minor skin lesions or arthropod bites, results in the ulceroglandular form of the disease pictured here.
Tularemia is a disease marked by <u>inflammation and necrosis</u> which may occur, singularly or in combination, in the lung, oropharynx, eye, skin, and lymph nodes.
The disease progresses over 7 to 14 days. Inhalational tularemia is characterized by necrosis of the alveolar septa and regional nodes. 50 percent of patients initially presenting with cutaneous ulcers will progress on into a secondary pleuropulmonary infection.
Long term complications may include mild hepatitis, and a renal damage.

Tularemia case history. Fatal case: 85 years old man was bitten by his pet cat and developed an ulcer on the back of his left hand. Five days later, the patient developed sudden spiking fever and chills. On exam the patient had non-healing ulcer of left hand and tender axillary lymphadenopathy. He died 3 weeks later of tularemia involving lungs, lymph nodes, spleen, liver and peritoneum. The lungs showed large areas of necrosis and cavitation. The slide shows a pulmonary cavity with a necrotic center surrounded by a yellow zone of consolidation.

c. Signs and symptoms. Within 2 to 10 days of inhalational exposure, victims will begin to develop fever, chills, headache, generalized muscle pain, nonproductive cough, and pneumonia. If the organism was ingested or inoculated, symptoms will also include regional lymphadenopathy, with or without cutaneous ulcers.

d. Prevention. Person-to-person transmission does not occur. Routine universal precautions should be followed.

e. Diagnosis and treatment. Confirmation of the diagnosis is problematic since growing the organism in culture is difficult and hazardous to laboratory personnel. Staining of ulcer fluids or sputum is also generally not helpful. Diagnosis can be established retrospectively by serology.
Treatment for adult and pediatric patients includes antibiotic therapy for 10 days with either streptomycin (1 gm every 12 hours IM; 15 mg/kg IM BID), which is the treatment of choice, or gentamicin (3 mg/kg/day). Prophylaxis with tetracycline or doxycycline is effective if adequate warning of a BW attack is provided.

4. Q Fever.
In May 1996, a cluster of individuals with high and persistent fevers presented to local healthcare facilities in Germany. Each of these individuals resided in a small rural town in Germany. Symptoms included fever greater than or equal to 102.2º F lasting more than 2 days, with three or more associated symptoms (such as chills, sweats, severe headache, cough, myalgias, arthralgias, back pain, fatigue, or feeling ill).
Serological tests showed that 25 percent of those tested (a total of 49 of 200 residents) had either clinical or laboratory evidence of Q fever. A common link among those infected was living or walking near a sheep stable or pasture. Before the outbreak, two flocks of sheep were kept near the community.
One of these farms had a flock of 20 sheep, all of which tested negative for *C. burnetii*. The second farm maintained a flock of 2,000 sheep in which 15 of the 20 tested were positive for *C. burnetii*.
The findings indicated that the large sheep farm was the most likely source of this outbreak, and the principal mode of transmission was airborne. Outbreaks of Q fever commonly occur after lambing because *C. burnetii* is reactivated in ewes during pregnancy. Because of this multiplication, high numbers of *Coxiella* (that is, as many as one billion organisms per gram of placenta) may be present in placenta, amniotic fluid, and fetal membranes. Inhalation of only one organism is sufficient to produce infection.

a. Microbiology. Q fever is caused by a rickettsia microorganism called *C. burnetii*, which is highly infectious and resistant to heat and drying. A rickettsia is an obligate parasitic bacterium that requires a host to survive, and in that respect, is similar to a virus. *C. burnetii* becomes engulfed within a phagocyte that allows the bacteria to multiply and disseminate to multiple organs and the RES. Initially, this organism causes the host cell to increase its metabolic and functional activity, leading to reduction of the cytoplasm and eventual cell rupture.

b. Pathogenesis. First described in Australia ("Query" fever). Q fever typically causes infection in animals (sheep, cattle, and goats), but humans can acquire the disease by inhaling aerosols contaminated with the organism. Lung macrophages engulf the organism and the rickettsia multiply inside the cell until the macrophage undergoes lysis and free organisms are liberated into the local tissue and blood stream.
The Q fever agent is extremely infective by aerosol with only a low inoculating dose required to establish infection.

C. Clinical Course.
Ten to 20 days after inhalation, there is a sudden onset of an influenza like syndrome with marked anorexia (etilology of this is uncertain). The disease runs a 2 to 14 day course marked by 100 - 104º F fever for a few days and an atypical pneumonia in 50 percent of the cases. X-ray may reveal a "ground glass" appearance. One-third of the patients may show liver enzyme elevations. Neck stiffness and CNS signs may also occur. Late complications may include ostemyelitis and chronic infective endocarditis with vegetations occurring primarily on the aortic valve. Blood cultures are negative. The disease is self limiting in non-immunocompromised individuals

d. Prevention. Person-to-person transmission from patients with Q fever does not occur. Still, universal precautions should be observed.

e. Diagnosis and treatment. Q fever is very difficult to diagnose because it resembles so many other endemic infectious diseases, including most cases of community acquired pneumonia. The diagnosis should be considered when numerous individuals from the same geographical area present with the same non-specific complaints and findings of pneumonia. Sputum cultures are not usually helpful since the organism is difficult, as well as, dangerous to grow. Serological tests exist, but are not readily available outside of the military. Differential diagnosis may include Brucellosis, but this is commonly associated with large joint arthropathy.
Treatment for children and adults is symptomatic since **most cases will resolve even without antibiotic therapy**. Giving tetracycline (500 mg every 6 hours) or doxycycline (100 mg twice daily) orally, however, will shorten the duration of the illness. Similar treatment during the incubation period, or as prophylaxis, may delay or prevent the onset of the symptoms depending on when the medication is given.

IV. VIRUSES AS BIOLOGICAL AGENTS

A. General Characteristics

Viruses are the simplest type of microorganism and are composed of only genetic material (RNA or DNA) surrounded by a protein coat. Viruses are much smaller than bacteria and lack a system for their own metabolism needing a host to survive. This host can be plant, animal, insect, bacteria, or human.

B. Disease-Producing Properties

Many viruses attack specific types of cells and use the host cell's chemical energy and protein synthesizing capabilities to replicate. The virus brings about changes in the host cells resulting in disease (including cancer) and death. In some cases, the exact mechanism by which viruses produce direct cytopathic effects is not completely understood. Some viral infections result in immune complex deposition, which activates complement and other inflammatory cascades that damage the vascular endothelium. In other cases, viruses activate the clotting cascade, resulting in DIC. Systemic effects of some viruses can also be enhanced by specific end-organ system failures. As an example, yellow fever causes massive hepatic necrosis and depletion of the vitamin K dependent clotting factors. The uremia that complicates the acute renal failure of some Hantaan viruses infection leads to platelet dysfunction, further promoting hemorrhage. A few viruses can be treated with drugs (antivirals), but vaccination, when available, is the most effective means of preventing infection.

C. Examples of Possible Viral Biological Weapons

1. Smallpox.

In May 1963, a 24-year-old seaman on vacation left Australia on a flight to Sweden that made stops for no longer than 50 minutes in Djakarta, Singapore, Rangoon, Calcutta, Karachi, Teheran, and Damascus en route to Zurich. In Zurich, the seaman deplaned and the following day boarded a flight to Sweden. Approximately 15 days later, he developed fever and a mild rash (consistent with smallpox) and remained home with his grandmother throughout his illness. Five days later, the grandmother developed similar symptoms and subsequently exposed three other individuals. She was at first diagnosed with "chicken pox" and recovered uneventfully. A total of 19 cases of smallpox were identified in the outbreak.

The seaman was diagnosed with smallpox, which he apparently acquired as a result of in-transit exposure at a terminal or on the plane. More than 300,000 persons were vaccinated. Of interest, Sweden was the first major country to eliminate indigenous smallpox in 1895. (MMWR Vol. 45, No. 25. 6/28/96)

a. Microbiology. Smallpox was caused by the *Variola* virus, an Orthopox virus, which caused both a major and minor form of the disease. Smallpox was declared eradicated in 1980 and is the only disease to date that has earned this distinction. The U.S stopped its civilian vaccination program in 1981. Despite eradication, concerns over clandestine stockpiles of the agent still remain. The Soviet Union was thought to have weaponized the agent.

At present there is a human monkey pox epidemic occurring in the Kasai Oriental region of the Democratic Republic of the Congo (Zaire). An endemic disease of ground squirrels in the region, the virus seems to be capable of enhanced human transmission

and is carrying a slightly higher morality rate than normal monkey pox. It is possible that this agent could become available to terrorist groups in the future. The overall clinical course of monkey pox in humans is equivalent to that of smallpox, but it is milder with a lower mortality rate. Vaccine against variola is cross-protective.

b. Epidemiology. Smallpox was a contagious agent that would be relatively easy to produce and disseminate as a biological agent. Typically, it had mortality rates approaching 20 to 40 percent in unvaccinated individuals (3 percent if vaccinated).

C. Clinical Course.
Airway exposure to the virus was followed by viral replication in the regional lymph nodes of the airways and viremia occurred 12 days later with the onset of an influenza-like syndrome.
The virus disseminated to the spleen, liver and lung, and an initial mild erythematous rash was followed 2 -3 days later by exanthema on the face, arms, and hands. Over a period of 8 to 10 days, the macules become papules and typical pustular vesicules.
Clinical variants of typical variola virus infection include Flat Smallpox and Hemorrhagic Smallpox in immunocompromized patients. Rapid death often occurred before typical lesions have had time to develop.
Initial macules progress to papules; to pustular vesicles, and scabs within 8 to 10 days. Varicella (chickenpox), on the other hand, causes a rash that starts primarily on the trunk and spreads to the extremities.
Smallpox - lesions are syndronous in their development

d. Prevention. Only about 30 percent of exposed individuals subsequently became ill, making it less communicable than, measles, and influenza. All contacts needed to be quarantined for at least 17 days and patients were kept isolated until the scabs completely healed over.

e. Diagnosis and treatment. Documenting the virus on electron microscopy of a vesicular sampling will confirm the presence of a pox virus. Variola would be confirmed by tissue culture and genetic typing methods. Aggregates of the variola virus may be seen by light microscopy (Guarnieri bodies).
Treatment is supportive. An immune globulin for variola may be accessible through the CDC. Before smallpox was eradicated, the protocol for post-exposure prophylaxis was to give the live vaccine to uncomplicated patients up to 1 week post-exposure and to give the vaccine (vaccinia) and vacinnia immune globulin (VIG) post-exposure to those at risk of vaccinia complications, such as patients who were pregnant with history of eczema, psoriasis, unhealed burns, or other exfliative skin disorders, or were immuno-compromised. If the disease were to reappear, there is no consensus on post-exposure prophylaxis to severely immuno-compromised patients, such as HIV-infected patients or organ transplant patients, but VIG alone would be a reasonable approach. World Health Organization (WHO) warehouses 20 million doses of the vaccine. No licensedantiviral medication is currently available against pox virus, but HPMPC is undergoing experimental evaluations for monkeypox.

a. Microbiology. VEE virus is a mosquito-borne alphavirus that is endemic in certain parts of the world (Central and South America, Mexico, and Florida) infecting horses, mules, and donkeys. If this agent was intentionally released as an aerosol, disease might occur simultaneously in both horses and humans, but this pattern would not be commonly recognized. Heat and disinfectants easily kill this virus.

b. Epidemiology. VEE is highly infectious, with nearly 100 percent of exposed individuals developing symptoms. However, only about 1 percent will die. Recovery from an infection results in excellent short-term and long-term immunity.

2. Venezuelan Equine Encephalitis. During the first week of September 1995, rural health clinics in Venezuela reported an increased number of patients seeking care for an acute febrile illness characterized by intense headache (56 percent), photophobia (56 percent), myalgias (56 percent), prostration, nausea, vomiting, and diarrhea (48 percent each). Convulsions and other neurologic symptoms complicated illness in some patients. These symptoms were compatible with Venezuelan Equine Encephalitis (VEE), which spread westward into Columbia, resulting in a minimum of 13,000 cases in humans and an undetermined number of equine deaths. Attack rates among households varied between 18 percent and 57 percent. In 1967, an outbreak in Columbia resulted in approximately 220,000 human cases of VEE.

c. Clinical Course. After exposure to the virus, symptoms begin to develop in 1 to 5 days and consists of spiking fevers to 104º F, rigors, severe headache, photophobia, myalgias, nausea, vomiting, and diarrhea. These symptoms tend to last up to 3 days, followed by a prolonged period of weakness and lethargy. Most patients recover in 1 to 2 weeks.

CNS symptoms secondary to meningitis and encephalitis are characteristics of VEE, but only a small percentage of victims (20 percent of children, 4 percent of adults) will actually develop these symptoms in the naturally acquired disease. In a BW aerosol, based on primate studies, the virus would tend to infect the olfactory bulb through the cribriform plate and a greater percentage of CNS involvement might occur. If patients develop CNS disease (meningitis, seizures, change in mental status, or coma), especially in children, the mortality rate becomes much higher (up to 20 percent). Permanent neurologic sequela has been reported.

d. Prevention. Secondary spread by person-to-person contact does not occur; however, universal precautions still should be practiced.

Regions where suitable mosquito vector species are present should intensify insect spraying programs following a biological attack, to prevent the establishment of endemic disease.

e. Diagnosis and treatment. Immunoassay, viral culture, or serological testing may confirm the diagnosis. Treatment is primarily supportive since no antiviral medication exists. Most patients should be treated with pain medications, while those with encephalitis may require anticonvulsant therapy.

3. Viral hemorrhagic fever. The VHF especially Ebola, serve as an example of how an emerging disease may be similar to a BW attack. On April 4, 1995, a hospital laboratory worker from Zaire developed an acute onset of fever and bloody diarrhea. Six days later, he was taken to surgery to repair a suspected perforated bowel. On April 14, other hospital employees began developing similar symptoms. By May 17, 93 suspected cases of Viral Hemorrhagic Fever (VHF) were reported, 92 percent of which were fatal. Public health authorities identified a total of 296 persons with VHF by June 25. Thirty-two percent of these victims were healthcare workers. Initially, victims developed fever, headache, chills, myalgias, and malaise that worsened into severe abdominal pain, vomiting, and diarrhea, along with a maculopapular rash. Hemorrhagic manifestations, including DIC, occurred in fatal cases. Ebola was identified as the causative agent with transmission occurring primarily through contact with infected bodily fluids. **By merely implementing universal precautions within the hospital, the number of additional cases dropped markedly.** (MMWR Vol. 44, No. 25. 6/30/96)

A 46 year old theatre/anesthesia nursing sister at Morningside Clinic (hospital) in Johannesburg became ill (slight fever) on Saturday November 2. She nevertheless continued to work until Wednesday November 6 when she awoke with a severe headache, and that morning her doctor referred her to a neurologist who had her admitted to Sandton Medi Clinic (hospital) with suspected encephalitis. Lumbar puncture was performed and no evidence of encephalitis was found in CSF. She was placed on; antibiotics and over the next few days developed a fine confluent macular rash and diarrhea which were ascribed to possible reaction to the antibiotics. On admission her platelet count was moderately low (105 X 10E9/L) and fell to 37 by Saturday November 9. She also had marked leukopenia at this stage and AST and ALT levels [liver transaminases (enzymes)] (initially normal) had started to elevate.
Apparently because of the low platelets, and possibly also on account of the rash and fever, blood was submitted for investigation of suspected viral hemorrhagic fever on the same day (Saturday November 9). Her serum was screened by indirect immunofluorescence for antibodies to Marburg, Ebola, Rift Valley fever, Lassa, Crimean-Congo and hanta viruses (="MERLCH" test), with negative results, and also inoculated into Vero cell cultures and day old mice for isolation of viruses.
Ebola fever had never been diagnosed in patients domiciled in south Africa in the seventeen years of existence of the Special Pathogens Unit at the National Institute for Virology, and subsidiary test were done to verify the specificity of the antiserum used in the fluorescence test to detect growth of virus.
The patient had developed hematemesis [bloody vomit] and had high AST and ALT values and slightly elevated leukocyte counts, and on the morning of November 15 a laparotomy was performed for suspected abdominal mass/abscess - only retroperitoneal hemorrhage was found.
That same morning Ebola virus antigen detection tests were done on the patient's sera using ELISA reagents previously obtained from the Special Pathogens Branch at CDC Atlanta, and ELISA tests for antibody using our own reagents; antigen titers rose form 16 in the serum of November 9 to 256 in the sera of November 14 and 15, and weak antibody reactions were recorded in the sera of November 14 and 15 by ELISA.

That day committees were established to oversee infection control, contact tracing and observation, and all other aspects of outbreak control at both hospitals and pathologist laboratories that had performed clinical pathology studies. A start was made to tracing suspicious patient contacts of the sick nurse.
The patient was moved to intensive care in Johannesburg Hospital, where a team trained in barrier-nursing and experienced in handling Crimean-Congo Hemorrhagic fever took over medical care. A full scale crisis committee was established under the wing of the Gauteng (provincial) Health Department and the national Department of Health, with subcommittees to handle all conceivable aspects of outbreak control, including news media liaison, where the policy has been one of deliberate openness.
Apparently the nurse was exposed to a great deal of blood in cleaning up after placement of a central line was performed on a very sick 40 year old Gabon doctor. She then became sick on November 2 after a putative incubation period of 4 days.
More than 200 people are quarantined at a hospital in Johannesburg after being in contact with either the Gabonese doctor or the nurse with Ebola infection. They are being tested and watched for symptoms of the deadly virus.
South Africa's health authorities have set up an Ebola help line for information about the situation. President Mandela urged the people not to panic.

a. Microbiology. The VHF are a diverse group of illnesses caused by a variety of RNA viruses with a wide range of morbidity and mortality. Each of these viruses has a unique history and is capable of being spread in most cases by an aerosol or fomite (except dengue virus). VHF agents especially Marburg and Ebola have allegedly been considered for weaponization. The clinical syndrome which these viruses cause in humans, is called VHF. These viruses include: Ebola, Marburg, Dengue, Yellow Fever, Crimean-Congo Fever, Hantaan Viruses, and Lassa Fever

Ebola and Marburg (Filoviridae). These two viruses were made famous in the non-fiction book Hot Zone and the fictional works of Executive Orders and Outbreak. The first outbreak of this virus occurred in Sudan and Zaire in 1976; a second outbreak occurred in Sudan in 1979. A single index case in Zaire in 1995 caused a large outbreak of approximately 300 cases. In the U.S., a related virus (Ebola Reston) was isolated from a group of laboratory monkeys imported from the Philippines in 1989. Four cases of Marburg disease in humans have been identified.

b. Pathogenesis. Not all infected patients develop VHF and the reasons for this vary depending on various host factors, differences between virus strains, and a number of immunologic mechanisms. What is consistent among each of these RNA viruses is that the target organ is the vascular bed, which causes widespread microvascular damage and changes in vascular permeability.
VHF are caused by four RNA virus families: the Filoviruses, the Bunyaviridae, the Flaviridae, and the Arenaviridae. These viruses use a variety of insect or rodent vectors. The natural reservoir for the filoviridae is unknown. The viruses are listed here, but are generally considered unlikely agents for BW terrorist use.

c. Signs and symptoms. Patients with VHF present initially with fever, myalgias, and prostration. Clinical evaluation may reveal conjunctival injection, petechial hemorrhages, and hypotension. Full-blown cases will evolve into shock and generalized mucous membrane hemorrhage with involvement of the respiratory, hematopoietic, and central nervous systems. Laboratory evaluation may reveal renal insufficiency, abnormal liver enzyme tests, and elevated bilirubin, all of which reflect a poor prognosis. Mortality rates range between 50 and 80 percent for strains of Ebola.
Since most strains of VHF are known to spread in the hospital environment, universal precautions are essential.

d. Prevention. VHF may be transmitted by bodily fluids, but the exact mechanism is unknown. This disease does not appear to be readily transmitted by the airborne route. (This is not the case for Ebola among monkeys.) The highest risk of transmission is during the latter stages of the illness, which are characterized by vomiting, diarrhea, shock, and hemorrhage. Since most strains of VHF are known to spread in the hospital environment, universal precautions are essential.
Patients suspected of having VHF should be isolated in a single room with an adjoining anteroom that serves as the only entrance. This anteroom should be stocked with personal protective gear (gloves, gowns, and masks) for staff. The patient's room should have negative air pressure compared with the anteroom and the outside hall. Strict barrier-nursing techniques should be enforced. Patients should be cared for at the hospital where they were first seen, since transferring patients may increase the potential for secondary transmission. These viruses are easily inactivated with soaps, detergents, and routine disinfectant solutions. (MMWR Vol. 37, No. S-3, 2/26/88)
In previous outbreaks, simple barrier nursing was enough to reduce health care provider infection rate to virtually zero.

e. Diagnosis and treatment. A detailed travel history and high index of suspicion are key components to making a diagnosis. This diagnosis should be suspected in any patient presenting with a severe febrile illness with evidence of vascular involvement, especially one who has traveled to known infectious areas. Multiple patients presenting with these same complaints should serve as a warning to a possible BW attack.
Definitive diagnosis requires specific viral studies. Rapid enzyme immunoassays and genetic typing are available at the CDC and the U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID), Frederick, Maryland for a number of the viruses and for patient transportation under biohazard safe conditions.
Treatment is largely supportive and typically requires intensive care monitoring to avoid fluid overload (pulmonary edema) while maintaining hemodynamic stability and providing appropriate comfort measures (sedation, pain medication). Systemic coagulopathy should be treated in manner similar to DIC. Ribavirin is an antiviral (available only on an IND basis) medication that has been used in therapy and prophylaxis for Lassa fever, Hemorrhagic Fever with Renal Syndrome, and CCHF. The only available vaccine is against yellow fever. Other vaccines are currently under investigation.
Many scenarios involving the use of BW agents suggest an attack by aerosol dissemination. Consequently, disease onset in a community would be rapid, and

treatment would need to be implemented quickly to have any effect on mortality or to ensure effective prophylaxis by implementing PPE, antibiotic therapy, or vaccinations if available.
Emergency care physicians need to have a high index of suspicion, be aware of the clinical picture afforded by the most common BW agents, and the most effective clinical intervention.

1) VEE - encephalitis syndrome; prolonged clinical course

2) Inhalational anthrax - rapid mediastinitis, shock, and death

3) Pneumonic plague - Rapid fulminant pneumonia

4) Tularemia - Prolonged clinical course with bronchopneumonia, hepatic involvement, ± skin ulcers; tender lymphadenopathy, and high fever

5) Q Fever - prolonged clinical course typified by an atypical pneumonia

6) Smallpox - Prolonged clinical course typified by characteristic skin lesions and pneumonia

V. TOXINS AS BIOLOGICAL WEAPONS

A. General Characteristics

Biological toxins are nonliving, poisonous chemical compounds that are produced by living organisms (animals, plants, and microorganisms). These agents are up to 1,000 times more lethal than standard chemical agents, but unlike chemicals, are not typically volatile or able to cause illness through skin absorption. As a result, **toxins are not prone to**
person-to-person transmission and are not very persistent when released. The toxicity of these agents varies by the route of entry (inhalation vs. ingestion vs. injection).

B. Disease-Producing Properties

Biological toxins produce illness and disease by a variety of mechanisms, including interfering with nerve conduction, interacting with the immune system, and inhibiting protein synthesis.

C. Examples of Bacterial Toxins

1. Botulinum toxin. On June 30, 1994, a 47-year-old man was admitted to the hospital with a subacute onset of progressive dizziness, blurred vision, slurred speech, dysphagia, and nausea. Physical exam revealed ptosis, extraocular palsies, facial paralysis, palatal weakness, and impaired gag reflex. He soon developed respiratory compromise requiring mechanical ventilation.
The patient's history was significant for eating home-canned green beans and a stew containing beef and potatoes. The stew had been cooked and left on the stove for 3 days

before being eaten without heating. Later analysis of the stew was positive for type A botulinum toxin.
In 1994, CDC reported 34 cases of food borne botulism in the U.S. Most outbreaks result from eating improperly preserved home-canned foods, with vegetables (asparagus, green beans, and peppers) being the most common source. In this case, the closed lid on the pot created the anaerobic environment necessary to allow spores of *Clostridium botulinum* to germinate and produce the toxin. (MMWR Vol. 44, No. 11. 3/24/95)

a. Pathogenesis. Botulinum toxin is a neurotoxin produced by the bacteria, *C. botulinum*, which causes the disease **botulism**. This toxin is the most lethal compound per weight and is approximately 15,000 times more toxic than the nerve agent VX when injected IP into mice. Botulism toxins produce similar effects whether the agent is inhaled or ingested. However, the onset of symptoms varies between hours and days depending on the route of exposure and the initial dose.
Botulinum works by irreversibly binding to the presynaptic neuromuscular junction and preventing the release of acetylcholine (ACh) there and at cholinergic autonomic sites. This interruption of neurotransmission causes both bulbar palsies and skeletal muscle weakness.

b. Signs and symptoms. After exposure to the toxin, a descending paralysis (head-to-toe) and bulbar palsies become the characteristic symptoms. A bulbar palsy is a cranial neuropathy that produces a loss of function in the nerves that originate from the brain stem. Symptoms include blurred vision, mydriasis, diplopia, ptosis, photophobia, dysphagia, and dysphonia.
Bulbar palsies develop first. Soon after, the skeletal muscles become weak, starting in the upper body and moving downwards in a symmetrical fashion. These symptoms may progress acutely to respiratory failure as a terminal event in as little as 24 hours. Patients usually remain awake and alert.

c. Diagnosis and treatment. Diagnosis is made from the unique clinical presentation of botulism (bulbar palsies and descending paralysis). Food borne outbreaks are extremely rare and will only occur in small clusters. Epidemics should, therefore, arouse suspicion of an intentional release. Laboratory studies are usually nonspecific. An EMG may be helpful. A mouse neutralization assay may help to confirm the diagnosis. An ELISA test can be performed at specialized laboratories.
Treatment is supportive and may require prolonged respiratory support on a ventilator. An antitoxin is available and may be effective if administered early, but it is associated with a number of adverse side effects including anaphylaxis and serum sickness (horse serum product).

2. Ricin. In 1978, a Bulgarian exile named Georgi Markov was reportedly shot in the leg with a pellet (filled with ricin) from the end of a device disguised as an umbrella while waiting for a bus in London. Only a minute quantity of ricin was used (estimated at a few hundred millionths of a gram) to kill the defecting official. He died 4 days later. At autopsy, the tiny pellet was found and determined to contain the toxin. The

assassination was reportedly carried out by the Bulgarian government, who received the technology to commit the crime from the former Soviet Union.

a. Pathogenesis. Ricin is a potent cytotoxin that is derived from castor beans and is a by-product in castor oil production. When compared to the chemical warfare agent VX, ricin is over 200 times more toxic by weight. This agent works by blocking protein synthesis within the cell and causes necrosis of the airway when inhaled. Over a million tons of castor beans are processed yearly into castor oil (5 percent of the mash is ricin). Ricin is a very stable compound and is extremely toxic by multiple routes of exposure. Ricin can be dispersed as an aerosol and can produce signs and symptoms within 8 hours of exposure. Ricin is also effective orally and by injection. Some believe ricin is a likely agent for assassinations.

b. Signs and symptoms. After inhalation, victims would begin to experience fever, chest tightness, cough, shortness of breath, nausea, and joint pain within 4 to 8 hours of exposure. Ricin appears to cause necrosis of the lower airway epithelium and severe pulmonary edema following aerosol challenge in experimental animals. Death may occur in 36 to 72 hours. If ricin is ingested, victims often develop rapid onset of nausea, vomiting, severe diarrhea, and gastrointestinal hemorrhage with necrosis of the liver, spleen, and kidneys. Shock typically ensues with death occurring in 3 days. By injection, ricin causes marked death of muscles and LNs near the site of injection along with multiple organ failure leading to death.

c. Diagnosis and treatment. Diagnosis is often difficult since most routine laboratory findings are nonspecific. ELISA testing of blood or immunohistochemical techniques may be used to confirm ricin intoxication. Clinically, ricin inhalation will appear to be very similar to other inhaled corrosives such as phosgene.
Treatment is supportive, ensuring adequate oxygenation and hydration. Gastric lavage and activated charcoal is probably indicated following accidental ingestion. No antitoxin or vaccine is currently available. If death has not occurred within 3 to 5 days, the patient usually recovers.

3. Staphylococcal Enterotoxin B.

a. Pathogenesis. Staphylococcal Enterotoxin B (SEB) is a fever producing exotoxin (secreted from the organism) produced by the bacteria, *Staphylococcus aureus*. This toxin commonly causes food poisoning in improperly handled foods that have an overgrowth of the staph organism and then are ingested. SEB symptoms will vary with the route of exposure (inhaled versus ingested). Inhalation of SEB does not occur naturally and the only experience is with animal models. The toxin over stimulates certain components of the immune system, which causes a large proliferation of T-cell lymphocytes and stimulates the production and secretion of various cytokines (such as tumor necrosis factor, interferon, and interleukins). These events are thought to mediate many of the toxic effects seen with SEB and a related staphylococcal toxin mediated disease called Toxic Shock Syndrome.

SEB can be very incapacitating, even at very sublethal doses, since over 80 percent of exposed individuals will develop symptoms. This agent can be aerosolized or introduced into the food supply.

b. Signs and symptoms. After inhalation, symptoms would be expected to develop within 3 to 12 hours and include sudden onset of fever (103° to 104° F), headache, chills, generalized myalgias, and a non-productive cough. Severe shortness of breath and chest pain will develop with larger doses. Nausea, vomiting, and diarrhea develop if the agent is swallowed and can be severe. Chest examination is usually unremarkable as is the chest x-ray, but in severe cases, pulmonary edema (ARDS) may develop.

c. Diagnosis and treatment. A diagnosis of inhaled SEB is difficult to make because the symptoms are so markedly different from the ingested form of the illness. Early on after exposure, the symptoms of SEB will suggest an infection with typical respiratory pathogens, such as influenza, mycoplasm, and adenovirus. A diagnosis of SEB would be based on a combination of clinical and epidemiological information when large numbers of patients present with the same signs and symptoms over a 24-hour period. Patients with SEB would tend to stabilize quickly, whereas patients with pulmonary anthrax, tularemia pneumonia, or pneumonic plague will progressively worsen (and die) if untreated. The shortness of breath seen with botulism would also be associated with bulbar palsies and descending paralysis.
Treatment is supportive with close attention to oxygenation and hydration. Severe cases may progress to pulmonary edema requiring mechanical ventilation support. Most victims recover after the initial acute phase of the illness.

VI. DEFENSE AGAINST BIOLOGICAL WEAPONS

A. Detection

BA have no immediate warning signs upon release, and effects are usually delayed for a number of hours or days. As a result, first responders will not be called to respond to victims from a "release area" as typically occurs after a chemical or HAZMAT incident. This makes early detection and identification of the bacterial agent quite difficult. Ideally, the best way to minimize or prevent injury is to have a detection device available to first responders at the scene to quickly identify the causative agent. This technology, however, is not commercially available.
The military has developed an experimental Biological Integrated Detection System (BIDS), which can be mounted on a vehicle and is capable of testing air samples for biological agents. This system concentrates the particles in the air sample and then subjects them to both generic and focused testing for specific agents. A Short-Range and Long-Range Standoff Detection System is also being field tested by the military and uses lasers to detect for the presence of aerosol clouds. Both of these systems are designed as early warning devices in battle conditions. These systems have difficulty, however, separating a true biological agent cloud from the normal background organic matter that is typically present in the atmosphere.

B. Self-Protection

The best safeguard to prevent the spread of a biological agent is self-protection. The first responder and medical caregiver must treat every patient with respiratory complaints (fever, cough, and shortness of breath) and open wounds as a possible infectious source. All health care providers should wear eye protection follow standard blood and wound precautions when treating these patients. Special protective garments are not required since clothing or disposable gowns provide reasonable protection against skin absorption. (29 CFR 1910. 1030)

C. Decontamination

Decontamination is the physical process of removing harmful substances from skin, clothing, and other items. It is a procedure that should be considered when the patient's skin is known or suspected of being contaminated with a biological agent. Merely removing the victim's clothing and placing them in a sealed bag for later disposal followed by a simple soap and water shower will ensure adequate protection. BW agents are not volatile and are difficult to re-aerosolize. Individual patient decontamination is probably not necessary after a BW terrorist attack because patient would only present to hospital after the inoculation period.

VII. TRIAGE

Triage typically will not be implemented soon after a BW attack because of the lack of warning and the insidious nature of the illness. When symptoms do begin to develop, patients will continue to present sporadically to health care facilities, unless a large epidemic occurs. When the pattern of patient complaints is confirmed to be secondary to a BW attack and this information is made public, hospitals will become overwhelmed by infected patients and by individuals worried that they may have been exposed to the agent. Triage will now be necessary to properly allocate existing resources, including isolation rooms, ventilators, PPE, and medications. The triage method used will vary according to local jurisdictions and their standard operating procedures (SOPs).

VIII. KEY POINTS

The only means of limiting the continual spread of any infectious agent is by self-protection, following universal wound and blood precautions, and decontaminating patients as necessary. Therapy should be initiated for what is treatable, including the use of broad-spectrum antibiotics, such as ciprofloxacin.

Support and confirmation is available from the specialized laboratories at the USAMRIID and CDC. If a BW attack is suspected or confirmed, hospital personnel should work to obtain information on the identification of the agent and its particular transmission and infectious properties, along with the BW immediate health consequences and specific management protocols. Law enforcement officials and the local FBI also should be notified.

Although numerous biological agents exist, only a few pose a risk of person-to-person transmission to unprotected individuals. These agents include the respiratory form of plague, VHF (such as Ebola), and smallpox. Patients with these diseases should be admitted with either respiratory droplet isolation (pneumonic plague and smallpox) or strict isolation (VHF). Most of the bacterial agents can be treated successfully with antibiotics (such as doxycycline and ciprofloxacin) if the disease is recognized promptly

and the drugs are administered early. These same two drugs can be administered orally as prophylaxis for known or imminent cases of biological agent contamination (such as anthrax, plague, and Q fever).

The public's fear of biological agents and the simple protective measures that they can take to prevent the spread of these agents can best be managed by educating communities about microbes, viruses, and toxins. Utilizing the media to provide factual and up-to-date information and including political leaders in the disaster planning process will help mitigate a variety of problems in the event of a biological agent attack.

Effective communication helps allay fears and prevent unnecessary rumors and panic.

NUCLEAR AGENTS

I. Terrorist Use of Nuclear Materials

Terrorist use of radioactive materials or a nuclear device constitutes a plausible threat. Such an incident could occur in one of five ways. The medical consequences will be dependent on the type of device used in a terrorist event.

A. Simple radiological device
B. Radiological dispersal device
C. Reactor
D. Improvised nuclear device
E. Nuclear weapon

A. Simple radiological device. This is the deliberate act of spreading radioactive material without the use of an explosive device. An example would be the placement of a high activity radioactive isotope in a public place exposing numerous individuals to various levels of radiation. Sealed sources could also be used to expose individuals near the source.
Two examples could be used in this context, although these were not terrorist events. They were the result of theft of sources. In the first case (Brazil), a hospital therapy source was stolen by two scrap dealers. The source, 1375 Ci of Cesium-137 was broken up and dispersed. The incident was not detected for 15 days. It resulted in 249 people being contaminated, four people dying, and 112,800 people requiring monitoring. The medical response and clean-up phases took several months to complete. In this case there was both an exposure and a contamination problem.

112,800	Surveyed for contamination
249	Contaminated
120	Externally contaminated on clothes and shoes
129	Externally and internally contaminated
20	Required specific hospital treatment
14	Developed bone marrow depression
8	Treated with GM-CSF
4	Died due to hemorrhage and infection

Medical staff received doses:	Maximum	500 mrem (5mSv)
	Average	20 mrem (0.2mSv)

The second incident occurred in Mexico in 1983. A cobalt-60 (450 Ci) therapy device was stolen and broken apart by a young man in Juarez. A small vial containing some 6,000 small pin-head-sized Co-60 sealed sources was opened spilling some of the contents into a pickup truck bed. The device was sold for scrap and ended up in recycled steel. In January 1984, a transport truck carrying a load of contaminated steel took a wrong turn into Los Alamos National Laboratory and set-off gamma alarms, thus the accident was discovered. Ten persons were overexposed in this accident, most of who worked in or visited the scrap yard. Nobody was contaminated in the accident.

B. Radiological Dispersal. A radiological dispersal device does not cause a nuclear reaction. Such a device is formed by combining an explosive agent (TNT or a plastic explosive) with radioactive materials that may have been stolen, for example, from a hospital or local industry. A similar type of event could also occur from a failed nuclear weapon detonation. (Under these circumstances, only the conventional component of the bomb explodes, rather than a nuclear reaction, causing widespread release of plutonium.) In either case, the initial explosion kills or injures those closest to the bomb, while the radioactive substances remain to expose and contaminate survivors and emergency responders.

C. Reactor. Nuclear reactor sabotage: Most people are aware of the reactor accidents of Three Mile Island and Chernobyl. The accident at Chernobyl was caused as the result of approximately eight (8) safety systems being bypassed. The experiment being carried out at the time resulted in melt-down of the core and also a severe explosion from the hydrogen bubble formed, resulting in the roof of the reactor being blown off and serious widespread contamination of vast areas of land. This accident caused the death of 28 individuals from acute radiation injury.
In the Western World, probability of terrorism involving a reactor is low. This is due to the high security surrounding a reactor together with the safety systems incorporated into the reactor. There is extensive shielding around a reactor; therefore, a significant amount of explosives would be required to breach this containment. This is a low probability event.

D. Improvised Nuclear Device (IND). This is any device designed to cause a nuclear detonation. This type of device, if successfully detonated, would cause widespread damage on the scale of Hiroshima and Nagasaki with the release of gamma rays, neutrons, and radioactive fallout. Construction of such a device to produce a nuclear detonation would be difficult as it is not easy to get the weapon to detonate correctly. Realistically, at best, a terrorist might be able to achieve a partial yield producing reduced effects of that caused by a nuclear weapon. In some cases only the conventional high explosives in the IND will detonate resulting in environmental contamination with plutonium or uranium. In this event, the IND is effectively a radiological dispersal device.
The Federal Bureau of Investigation (FBI) feels that is is unlikely that terrorists will have the engineering sophistication and access to high-grade nuclear materials that are required to build such a device, but any detonation of an improvised or stolen device would generate high levels of radiation.

E. Nuclear Weapon. The probability of stealing a nuclear weapon in the Western World is very remote because of the high security surrounding these devices. However, a Russian general has stated publicly that 50 to 100 one kiloton, suitcase nuclear weapons are unaccounted for in the former Soviet Union.
If one considers the consequences of a one-kiloton yield, then the following would occur within one minute around the point of ground zero:

1. Blast range would reach a distance of approximately 400 yards.

2. Thermal radiation would reach the same distance as the blast.
3. Nuclear radiation (i.e., gamma and neutron) would reach approximately half a mile.
4. The radioactive fallout could produce very high exposure rates, up to half a mile.
5. The added factor of the electromagnetic pulse, which only applies to high aerial bursts (several kilometers), would result in damage to electric equipment.

As the size of the weapon increases, the effects encompass a greater distance.

II. Ionizing Radiation.

A. Ionizing radiation can be machine generated (i.e., x-rays), or it can come from radioactive atoms. Radioactive atoms are atoms with too much energy or mass. The basic building block of all matter is the atom. The atom consists of a central nucleus with shells of electrons orbiting around this nucleus. The nucleus is made up of neutrons and protons. These protons, which are positively charged, have the tendency to repel each other. The protons are held together within the nucleus by a force (a super nuclear glue) which has three characteristics: it acts over a very short range, independent of charge and is very strong. There is a ratio of protons to neutrons (1 to 1.2) for stability of an element. Each element has a defined number of protons. When an element is radioactive, usually there is an imbalance of this ratio of protons to neutrons; often the imbalance is due to an excess of neutrons.

B. With respect to a radioactive nuclide, for it to become stable, the nucleus has the ability to change a neutron into a proton with the ejection of a negative electron or, conversely, has the ability to change a proton into a neutron with the ejection of a positive electron known as a positron. The nucleus also has the capability of ejecting large particles, consisting of two protons and two neutrons, known as an alpha particle. Therefore, a radioactive nuclide achieves stability by ejecting particles until it has the correct ratio of protons to neutrons, remembering that the resultant element will be different than the original one.

C. Although the final element concerned has the correct number of protons and neutrons, it is still not stable. The reason for this is that within the nucleus there is an excess of energy. This excess energy is given off as electromagnetic energy of very short wave length. It is called gamma radiation. When all this excess energy is given off, the resultant element finally becomes stable.

D. The most common types ionizing radiations are alpha particles, beta particles, gamma rays or x-rays, and neutrons. Gamma rays and x-rays may also be referred to as photons.

E. The basic building block of any tissue is the cell, and damage to the cell changes its chemistry or DNA. The chemical damage is instantaneous, but the clinical expression of this damage can take hours to years to express itself. At high doses, clinical expression can be within hours [e.g., the acute radiation syndrome (ARS)]. However, at lower doses or even after recovery from ARS, there is the probability, although low, of developing cancer 20 to 30 years later. Another problem that may present itself is cataract formation,

which requires a prompt dose in the region of 200 rem. It has been shown that neutrons are more effective in producing this type of injury. Some fetuses in utero as a result of the atomic bombings in Japan showed birth defects (e.g. low birth weight, small head circumference with mental retardation).

F. Alpha particles. Alpha particles are composed of two neutrons and two protons. Alpha particles do not penetrate the skin and can be shielded by a thin layer of paper or clothing. Because the outer layer of skin is dead and several microns thick, the alpha particle is unable to penetrate through the dead layers of skin to reach the lower layers of living cells and generally will not cause any skin damage. If, however, an alpha emitter gets inside the body through inhalation, ingestion, or via a wound, the alpha emissions are near live tissue, and localized damage could occur.

G. Beta particles. Beta radiation may travel meters in air and is moderately penetrating. Beta radiation can penetrate human skin to the "germinal layer", where new skin cells are produced. If beta emitting contaminants are allowed to remain on the skin for a prolonged period of time, they may cause skin injury. Beta emitting contaminants may be harmful if deposited internally. Personal protective equipment (PPE) provides some protection against most beta radiation.

H. Gamma rays (photons). Gamma radiation is able to travel many meters in air and many centimeters in human tissue. It readily penetrates most materials and is sometimes called "penetrating" radiation. X-rays are like gamma rays. They, too, are penetrating radiation. Radioactive materials that emit gamma radiation constitute both an external and internal hazard to humans. Dense materials are needed to shield against gamma radiation. PPE provides little shielding from gamma radiation but will prevent contamination of the skin. Gamma radiation frequently accompanies the emission of alpha and beta radiation.

I. Neutrons. Neutrons are neutral particles emitted from the nucleus of an atom. Neutrons lose most of their energy through collisions with other atomic nuclei. An analogy that could be used is the billiard ball effect (i.e., when one billiard ball strikes another, energy is transferred from one ball to the other). Under certain circumstances, neutrons can be captured by a stable nucleus, making the nucleus radioactive. An example of this is Na-23 being changed (transmuted) into Na-24.

3. Radiation Detection. Unfortunately our body senses cannot detect radiation. We cannot see, smell, taste, feel, or hear radiation, but we have very good instrumentation to detect it.

Radiation monitoring instruments detect the presence of radiation, usually by collecting charged particles (ions). The radiation measured is usually expressed as exposure per unit time, using various units of measure, including the curie (Ci), the becquerel (Bq), and counts per minute (CPM). The most commonly used instruments to detect the presence of radiation include:

A. Geiger Mueller Survey Meter or Geiger Counter. The Geiger-Mueller (GM) survey meter will detect low levels of gamma and most beta radiation. The instrument typically has the capability to distinguish between gamma and beta radiation. This instrument is used to measure background radiation levels and to quickly evaluate potentially contaminated victims. If a greater level of radiation emission is anticipated, a higher range instrument (such as an ionization chamber) should be used. At higher levels, the GM meter will often display incorrectly low or off-scale readings.

B. Ionization Chamber Survey Meter. This device measures gamma ray dose/rate when high-level radiation hazards are suspected. Low-level gamma contamination is not detected.

C. Alpha Monitors. These monitors are designed to measure the presence of alpha particles. Since alpha particles travel short distances, they might not be detected in wounds because blood and tissue fluids may shield the particles from reaching the monitor's surface.

D. Dose Rate Meters. These measure mrad/hour or rad/hour units of personal radiation. To find the dose an individual is receiving, multiply the dose rate by the time.

Dose = Dose Rate X Time

E. Pocket or Personal Dosimeters. These simple devices measure accumulated radiation to gamma rays. Some devices basically contain a piece of film embedded in a badge of varying densities.

F. Other Devices Used. TLD (lithium fluoride) and QFD Quartz Fiber Dosimeters, and Electronic Readout Dosimeters.

4. Radiation Units.

A. The basic unit for measuring radiation is the rad (radiation absorbed dose). The rad is defined as the deposition of 0.01 joule of energy per kilogram (kg) of tissue. To quantify the amount of damage that is suspected from a radiation exposure, rads are converted into rems (which at one time stood for Roentgen Equivalent Man). The rem is adjusted to reflect the type of radiation absorbed and the likelihood of damage. In most cases, the rad will be equivalent (1 rem = 1,000 millirem).

B. The rem was introduced to take into account this variation in potential tissue damage. This is important because radiation may be of a mixed type. For example, a standard x-ray machine was used to deliver 100 rads of radiation and to compare the biological endpoint with other types of radiation. It was found that 100 rads of gamma and beta radiation produced the same effect as 100 rads of x-rays. However, it was found that only 20 rads of neutrons and 5 rads of alpha were found to produce the same effect as 100 rads of x-ray. Therefore, neutron and alpha radiations were more potent and required fewer rads to produce the same effect. (This concept applies only to occupational exposure.) Now weighting factors are used for each organ/tissue.

5. Radioactive Materials

A. These are materials that emit ionizing radiation and are used in diagnosis (nuclear medicine), therapy (cancer treatment), industry (non-destructive testing), and for research purposes. A number of radioactive materials, including radioactive waste, are commercially shipped in specialized containers. Radioactive materials are chemically and physically identical to their non-radioactive counterparts. Radioactive materials behave in the body the same as their non-radioactive counterparts (for example, radioactive iodine behaves the same as stable iodine).

B. For practical purposes, after 10 half-lives, most of the radioactivity in a particular quantity of a radioactive material is gone.

C. Radioactivity has existed for millions of years in the crust of the earth, in building materials, in the food we eat, the air we breathe, and in virtually everything that surrounds us. Radiation from these materials, as well as cosmic radiation from the sun and universe, makes up the natural background radiation to which we are constantly exposed.

D. Most individuals are exposed to about 360 millirems per year natural causes and manmade sources. Smoking 1.5 packs of cigarettes a day for 1 year produces an accumulative radiation dose of 16 rem to the bifurcation of the bronchus. If an individual is exposed to more than 100 rads at one time, predictable signs and symptoms will develop within a few hours, days, or weeks depending on the dose. Fifty percent of individuals exposed to a single dose of 450 rads will die without medical intervention.

6. Radiation guidelines

A. Time. The shorter the time in a radiation field, the less the radiation exposure. Work quickly and efficiently. A rotating team approach can be used to keep individual radiation exposures to a minimum.

B. Distance. The farther a person is from a source of radiation, the lower the radiation dose. Do not touch radioactive materials. Use shovels, brooms, etc., to move materials to avoid physical contact.

C. Shielding. Although not always practical in emergency situations, shielding offered by barriers can reduce radiation exposure.

D. Quantity. Limit the amount of radioactive material in the working area to decrease exposure.

7. TYPES OF RADIATION INJURY

Types of Radiation Injury: Regardless of where or how an accident involving radiation happens, *three types of radiation-induced injury can occur: External irradiation, contamination with radioactive materials, and incorporation of radioactive material into body cells, tissues, or organs.*

A. External Irradiation. External irradiation occurs when all or part of the body is exposed to penetrating radiation from an external source. During exposure this radiation can be absorbed by the body or it can pass completely through. A similar thing occurs during an ordinary chest x-ray. Following external exposure, an individual is not radioactive and can be treated like any other patient.

B. Contamination. The second type of radiation injury involves contamination with radioactive materials. Contamination means that radioactive materials in the form of gases, liquids, or solids are released into the environment and contaminate people externally, internally, or both. An external surface of the body, such as the skin, can become contaminated, and if radioactive materials get inside the body through the lungs, gut, or wounds, the contaminant can become deposited internally.

C. Incorporation. The third type of radiation injury that can occur is incorporation of radioactive material. Incorporation refers to the uptake of radioactive materials by body cells, tissues, and target organs such as bone, liver, thyroid, or kidney. In general, radioactive materials are distributed throughout the body based upon their chemical properties. Incorporation cannot occur unless contamination has occurred.
These three types of accidents can happen in combination and can be complicated by physical injury or illness.
Irradiation of the whole body or some specific body part does not constitute a medical emergency even if the amount of radiation received is high. The effects of irradiation usually are not evident for days to weeks and while medical treatment is needed, it is not needed on an emergency basis. On the other hand, contamination accidents must be considered medical emergencies since they *might* lead to internal contamination and subsequent incorporation. Incorporation can result in adverse health effects several years later if the amount of incorporated radioactive material is high.
Hospital emergency department personnel should always use proper priorities in caring for accident victims where potential radiation hazards exist: treat life-threatening problems first, limit the radiation dose to both victim and personnel, and control the spread of radioactive contaminants. *Serious medical problems always have priority over concerns about radiation, such as radiation monitoring, contamination control, and decontamination.*

8. LD 50/60.

A. After the dropping of the atomic bombs in Japan, experiments were carried out on various animals to determine the dose that would kill 50 percent of the experimental animal population within a set time period. It was found that the dose to achieve the Lethal Dose $(LD)_{50/30}$ on various species was as indicated on the slide. The killing effect of radiation depends on many factors including the age, environment, state of

health, and the amount of stress on each individual; therefore, the LD_{50} can vary for a given species depending on these factors.
Accident data on humans that were not treated indicate the LD_{50} was in the region of 350 rads to 450 rads. Also, the time of expression tended to be 60 days compared to 30 days for animals. Therefore, for man we talk about the $LD_{50/60}$, and for animals the $LD_{50/30}$.
If one keeps increasing the dose, obviously one will reach the LD_{100} where all the animals will die.

B. Severity of Injury. In general, the higher the dose, the more severe the early effects will be and the greater the possibility of delayed effects.
It is important at this stage to briefly discuss the effect of radiation on the cell. Obviously, one can increase the dose until the cell is killed outright. However, it is found that a much lower dose can stop cell division. For example, if we consider the hematopoietic system, an individual hematopoietic stem cell has the capability of producing millions of mature cells. Preventing stem cell division means the loss of these cells. The importance of this is that a sub-lethal dose produces these effects. Two important organ systems that have rapidly dividing cell lines are the hematopoietic and gastrointestinal systems.

C. Survival Time. The curve of Patt shows failure of three different organ systems of the body and reduction in the time interval to death. The graph also shows that in the region of 200 rads death results from the hematopoietic syndrome where as thousands of rads are required to produce the central nervous system syndrome. Therefore, at lower doses, death is caused by stopping cell division whereas higher doses produce cell death.

9. ACUTE RADIATION SYNDROME

A. Definition

An acute illness, which follows a roughly predictable course over a period of time ranging from a few hours to several weeks after exposure to ionizing radiation. The acute radiation syndrome is produced if enough radiation reaches enough sensitive tissue. Important factors are:

High dose
High dose rate
Whole body exposure
Penetrating irradiation

To these factors, other factors need to be taken into consideration, such as, age (young or old), sex, genetic, medical, etc. The source of radiation does not matter if the dose is high enough; it will produce the same effect (i.e. reactor, nuclear weapon, industrial source, medical therapy source).

B. Signs and Symptoms

The signs and symptoms that develop in the ARS occur through four distinct phases:

1. Prodromal phase. Depending on the total amount of radiation absorbed, patients may experience a variety of symptoms including loss of appetite, nausea, vomiting, fatigue, and diarrhea. After high radiation doses, additional symptoms such as prostration, fever, respiratory difficulties, and increased excitability may develop. This is the stage at which most victims seek medical care.

2. Latent phase. This is the transitional period in which many of the initial symptoms resolve, and may last for up to 3 weeks depending on the original radiation dose. This time interval decreases as the initial dose increases.

3. Illness phase. The period of time when overt illness develops, often characterized by infection, bleeding, electrolyte imbalance, diarrhea, changes in mental status, and shock.

4. Recovery or death phase. This follows the period of overt illness, which may take weeks or months to resolve.

D. Affected Systems

1. Hematopoietic or blood forming system. This system shows the earliest indication of the severity of the radiation exposure through the rapidity and degree of drop in the cell count (lymphocytes, granulocytes, thrombocytes, and reticulocytes). This reduction in the cell count is commonly associated with fever, sepsis, and hemorrhagic complications.
The absolute lymphocyte count at 48 hours after exposure is a good predicator of prognosis. For example, if the total lymphocyte count is greater than 1,200, it is unlikely that the patient has received a lethal dose. If at 48 hours the lymphocyte cell count is between 300 and 1,200, a significant exposure exposure has occurred and the patient should be hospitalized with barrier protection isolation. Lymphocyte levels of less than 300 cells per ml are usually critical and warrant the consideration of the use of colony stimulating factors (CSF) on an individual basis.

D. Gastrointestinal System. Symptoms in this system are regularly seen at acute doses greater than 600 rads and result from damage to the epithelial cells lining the intestinal tract. The higher the exposure, the sooner the symptoms of nausea and vomiting develop. The presence of these symptoms typically overlaps with the drop in cell count described previously. As a result, sepsis, loss of fluids, electrolytes and opportunistic infections complicate the picture. Persistently high fevers and bloody diarrhea are an ominous sign despite adequate fluid and electrolyte replacement.

E. Central Nervous System. Central nervous system (CNS) symptoms are seen with acute radiation doses in excess of 1000 rads and are probably due to diffuse microvascular leaks within the brain. Damage to these blood vessels results in the loss of fluids and electrolytes, edema, increased intracranial pressure, and death. This injury is irreversible and the victim rarely lives long enough to suffer any hematological or

gastrointestinal symptoms. Symptoms of shock may develop quickly in these patients. There is also an associated cardiovascular collapse in this kind of patient.

10. Local Injury to Skin.

> 300 rads: Epilation 17 - 21 days
> 600 rads: Erythema which may disappear within a few hours
>1000 rads: Dry desquamation 2 - 4 weeks
>1500 rads: Moist desquamation 2 - 8 weeks
>5000 rads: Necrosis few days to months

A. Skin. Various skin changes occur depending in the radiation dose. The injuries tend to progress with dose and there appears to be a threshold effect for these clinical signs. Early erythema is an important sign to look for. At doses around 300 rads, erythema will develop within a few hours, but more importantly, it can disappear within a few hours only to reappear at a later time. Therefore, the patient should be examined on an hourly basis for this sign and ideally photographs should be taken to document this sign. If local radiation dermatitis develops with this sign, the dose is in the region of 1,000 rads. If blistering occurs then the dose is in the range of 1,500 rads. Also if necrosis develops, the dose is in the region of > 5,000 rads. Therefore, by noting these clinical signs, one is able to establish the approximate dose range the patient was subjected to and these doses would be confirmed by dosimetry at a later stage.

B. In an accident which took place in Fontana, California, involving a 28 Ci Iridium-192 source, the patient concerned picked up the source and put it in his back pocket. This resulted in several blisters within 5 hours. One large bullae formed within 18 hours and within one week, a large necrotic ulcer formed on the right buttock (11 X 7 cm in area and between 2 to 4 cm deep). The dose at 1 cm depth was calculated to be 52,000 rads.

11. Trauma and Radiation

A. Patients who have suffered trauma (from an explosive or burn) combined with an acute exposure to penetrating radiation will have an increased chance of dying as compared to patients who have suffered from the same dose of radiation without trauma. All combined injuries are worse than radiation alone. If a patient has received an acute dose greater than 200 rads, effort must be made to close wounds, cover burns, reduce fractures, and perform surgical stabilizing and definitive treatments within the first 48 hours after injury. After 48 hours, surgical interventions should be delayed for 2 to 3 months.

B. Compared to thermal or radiation insult alone, combination of the two will increase mortality. The increase in mortality is dependent upon the degree of thermal insult and the radiation dose (and dose distribution) received. Using the LD_{50} burn model of Alpen, a dose of 100 rads increased the mortality in animals from 50to 65 percent. Increasing the dose to 250 rads increased mortality to 90and 100 percent of the animals died when a dose of 500 rads was given.

12. Triage.

A. Triage of victims from a radiological event should follow the same principles used in sorting victims of a hazardous materials incident. Victims are classified with regard to their need for treatment and will be classified as requiring minimal treatment, immediate care, delayed care, or as expectant. Since the degree of radiation injury will not be initially apparent, triage criteria will need to be based on associated injuries and complaints. The triage method used will vary according to local hospital practices.

B. Victims who have received very high doses of radiation from a gamma or neutron source might exhibit signs and symptoms that would indicate the level of exposure. Neurologic signs such as confusion, delirium, or vomiting indicate a lethal dosage of radiation. Other signs of lethal radiation injury include high fever, profuse vomiting, and bloody diarrhea with 2 hours of exposure (nearly 100 percent mortality).

C. Various triage-tagging systems are currently used in hospitals. A commonly used triage system for mass casualty incidents in the pre-hospital setting can be easily applied to the hospital environment. This triage system is called Simple Triage and Rapid Treatment (START) and has proven to be very effective because of its simplicity and lack of user variability. Responders focus on three evaluation areas: ventilation, perfusion and pulse, and neurological status. If an abnormality is shown in any of these areas (that is, difficulty breathing, delayed capillary refill, or altered mental status), the patient is labeled immediate (red tag) and the responder moves on to the next victim. If each of these evaluation areas is normal, the victim is labeled delayed (yellow tag).

13. Classification, Treatment, and Disposition

A. Once the radiological survey and decontamination procedure is complete, patients may be classified into one of 3 categories, based on their presenting signs and symptoms.

B. Survival Probable Group. This is a group of patients who present without any initial symptoms, or whose symptoms are so minimal ie., nausea and vomiting), that they resolve in a few hours. These individuals have most probably not received a lethal radiation dose, and have most likely been exposed to < 100 rads. The initial CBC and sequential studies will not show a significant decrease in the lymphocyte or granulocyte counts. These patients can be safely sent home and instructed to return if symptoms redevelop.

C. Survival Possible Group. These victims present with nausea and vomiting, which typically lasts 24 to 48 hours followed by an asymptomatic period. During this latent phase, laboratory evaluations will show a drop in various cell counts (that is, lymphocytopenia, leukocytopenia, and thrombocytopenia). If vomiting is severe, these patients should be admitted for fluid and electrolyte therapy and treated with antiemetics.

D. If the absolute lymphocyte count is less than 1,200 (or 50 percent of the baseline), protective isolation precautions should be implemented. These patients will have typically received a radiation dosage in the range of 200 to 800 rads. The LD_{50} in mass casualty situations is in the range of 350 to 450 rads. Treatment is primarily supportive. Blood replacement products, hyperalimentation, antibiotics, antivirals, and antifungal mediations should only be administered after consultation with a hematologist, oncologist, or infectious disease specialist. Colony Stimulating Factors (CSF) will probably be indicated for pancytopenia.

E. Survival Improbable Group. Patients in this group have been exposed to whole-body irradiation in doses exceeding 800 rads. These victims present an acute onset of fulminating vomiting, diarrhea, and shock, requiring aggressive fluid and electrolyte therapy. The presence of any CNS symptoms (confusion, change in mental status, etc.) signals that the patient has received a lethal dose of radiation. These victims will develop bone marrow suppression, leading to aplasia and pancytopenia that is uniformly fatal unless a successful hematopoietic stem cell transplant and/or colony stimulating factors are used. Treatment outcomes in some recent accidents suggest that exposure in the 800 to 1,200 rad range can be successfully managed through the hematopoietic crisis, although the individuals treated often succumbed to residual lung damage 6 to 12 months following exposure. In mass casualty situations, these victims are provided with comfort measures only (that is, narcotics).

14. Prepare to Receive Contaminated Patients in a Disaster.

- Activate the hospital plan
- Consider establishing triage area outside the hospital
- Plan to control contamination:

A. Set up a controlled area large enough to hold the anticipated number of victims. Demarcate controlled area with tape or floor markings.
B. Prevent tracking of contaminants by covering floor areas
C. Restrict access to the controlled area
D. Monitor *anyone* or *anything* leaving the controlled area
E. Use strict isolation precautions, including protective clothing and double bagging
F. Use a buffer zone or secondary control line for added security
G. Control waste by using plastic-lined container for clothing, linens, dressings, etc.
H. Control ventilation
I. Change instruments, outer gloves, drapes, etc. when they become contaminated.
J. Use waterproof materials to limit the spread of contaminated liquids; for example, waterproof aperture drapes.

Protective clothing for staff: gowns (preferably water-resistant), caps, masks, boots, and two pair of gloves. Staff should wear dosimetry (if it is available) and eye protection.

- Notify RSO to issue dosimeters, prepare instruments, and mobilize nuclear medicine staff to assist with surveys.

- Designate storage area for waste (outside hospital)

A decontamination corridor (that is, portable shower, collection pool, ground tarp, water pump, hoses, collection containers for the victim's clothing and personal items, soap, towels, and disposable paper gowns) should be set up outside the ED entrance, near the triage area. If a permanent decontamination facility is utilized, it should be prepared with appropriate supplies, including collection container, soap, towels, and gowns as specified above. In either case (portable or permanent), water runoff from the decontamination procedure should be contained if possible. It is preferable that uninjured persons use a community or self-help shower.

Patient Arrival

Determine if incident involves contamination. If so, each patient should be surveyed, checking for the presence of radioactive contamination. If contaminated, the patient should be directed to the decontamination area.

Upon arrival, all patient clothing should be removed by staff or under the guidance of staff, so that further contamination of the patient is limited. Clothing and personal belongings are placed in a labeled biohazard bag. A privacy area should be provided for clothing removal and the patients should be given either a disposable gown or blanket for modesty and weather protection. A security official should be assigned to guard the personal belongings.

- At the same time, biological samples should then be taken: nasal swabs, throat swabs, etc.
- Collect blood for CBC and DIFF.

Note past medical history of patient. Important questions are history of renal disease, allergies, or previous nuclear medicine procedures.

C. Patients know or suspected of being contaminated should be decontaminated. If open wounds are present, they should be irrigated first, then covered with a sterile, waterproof dressing prior to the total body washing. After decontamination, each patient should be re-surveyed and, if negative, given a hospital gown and admitted to the hospital for further evaluation and treatment. Evidence of continued contamination required additional washings of affected areas. This decontamination procedure is also appropriate for those victims who may be contaminated with both radioactive substances and hazardous materials (including chemical and biological agents). If an injured patient's hair requires extended decontamination, it can be washed in a position similar to that used by barber salons, with the patient reclined to allow runoff that does not touch the rest of the patient. If the patient is fully mobile, hair can be washed with the patient bent over. Patient contamination is usually confined to the hair, face, neck, and hands. Avoid contaminating previously clean areas. For example, improper washing of heavily contaminated hair could cause contamination of the body crevices, especially the hair regions of the axilla and pubis, thus resulting in extra time spent decontaminating areas which were not contaminated in the first place.

D. Internal Contamination

Once radioactive materials cross cell membranes, they are said to be incorporated. Incorporation is a time-dependent, physiological phenomenon related to both the physical and chemical natures of the contaminant. The rate of incorporation can be quite rapid, occurring in minutes, or it can take days to months. Thus, time can be critical and treatment (decorporation) urgent. Several methods of preventing incorporation (e.g., catharsis, gastric lavage) might be applicable and can be prescribed by a physician. Some of the medications or preparations used in decorporation might not be available locally and should be stocked when a decontamination station is being planned and equipped.

If internal contamination is suspected or has occurred, the physician or RSO should request samples of urine, feces, vomitus, wound secretions, etc. Whole-body counting and radioassay also can help evaluate the magnitude of the problem and the effect of any treatment. The contaminated patient admitted with an airway device or endotracheal tube must be considered to be internally contaminated.

E. A mass casualty incident resulting from nuclear terrorism is likely to generate large numbers of frightened individuals who may not actually require any decontamination or medical care. To reassure these psychological casualties and prevent them from overwhelming health care facilities, counseling centers should be established 10 miles upwind of the incident site. Numbering four or more depending on the magnitude of the incident, these centers should each be staffed by a physician with radiological background, psychiatrist, health physicist, RSO with instrumentation, and a psychological counselor. A center should also be set up in the hospital for staff and members of the public.

15. KEY POINTS

Hospital personnel should be prepared for a nuclear reactor accident, industrial incident, or terrorist event. It will present many unique challenges to hospital personnel. A radiation-contaminated patient should be handled in the same manner as any hazardous material accident victim. In some respects, these victims have an advantage over other hazardous materials-contaminated patients because they can be surveyed with instruments to determine if they are truly contaminated or not. In any case there is minimal risk to responding and treating personnel if the victims are properly handled.

B. Carefully evaluating the initial presenting signs and symptoms (such as nausea, vomiting, diarrhea, changes in mental status, shock, and lymphocyte count over the first 48 hours) becomes the most reliable indicator of the radiation dose and the patient's ultimate prognosis. Since no antidote exists for radiation exposure, treatment is primarily supportive with more specialized care directed towards patients with high dose irradiation and those with internal contamination. Consultation with specialists in hematology, oncology, radiation, and infectious disease should be obtained.

C. The need for initial treatment for internally contaminated patients is determined based on the patient's medical condition, history, biological samples (nasal swabs), and definitive evaluation of internal contamination. Whole body counting may be needed if internal contamination is suspected.

SPECIAL CONSIDERATIONS

I. INTRODUCTION

Fortunately, mass casualty events are uncommon in the U.S. Most naturally occurring disasters rarely produce more than 40 victims, the majority of whom are usually treated and released from local hospitals. Two recent mass casualty incidents (MCIs) in the U.S. were a consequence of terrorism. The bombing of the World Trade Center resulted in 6 deaths and over 1,000 injuries, while 168 persons died and 759 others sustained injuries during the Oklahoma City bombing. These events, combined with the recent bombings in Atlanta, Georgia, and the nerve agent attacks in Japan have brought a number of issues to the forefront for hospital personnel to consider.

- Most disaster victims receive their initial care at the hospital
- Bomb blasts are a commonly used terrorist (criminal) weapon
- Healthcare providers are at risk for secondary contamination
- Explosives may be combined with NBC warfare agents
- Strategically placed secondary bombs may be used to injure first responders and healthcare providers.

The objective of this section is to provide an overview of topics that are important for the appropriate management of NBC terrorist-associated injuries. Topics include the MCI, triage and decontamination in an MCI, bomb blasts, and crush injuries. Issues of mixed trauma and NBC casualties will also be addressed.

II. MASS CASUALTY INCIDENT

In an MCI, available resources are taxed by an unusually high number of patients. Triage decisions must be made regarding treatment and disposition of these victims. The number of victims and availability of personnel and equipment govern these decisions. This definition of an MCI will vary from community to community and from hospital to hospital, depending on the availability of resources.

A mass casualty event associated with a terrorist attack may pose special hazards to the responder. It is imperative that responding hospital staff take appropriate steps to routinely protect themselves from the possibility of exposure to NBC hazards, and injury from secondary explosive devices placed within the hospital.

Terrorist attacks utilizing NBC weapons may produce large numbers of casualties. In past incidents, many of the victims who sought medical care were suffering from psychosomatic ailments produced by the stress of the incident. These psychogenic casualties can create major logistical problems for the healthcare system. These victims should ideally be triaged for worsening conditions to a pre-designated area to avoid congestion of the emergency department (ED). At this location, medical personnel can observe these patients while they are being "defused" by crisis intervention teams. Consider using alternative sites such as the hospital cafeteria, auditorium, or a nearby

school. The crisis teams should also have personnel to assist with the assessment of these victims within EDs.
Emergency medical service (EMS) tends to preferentially transfer patients to the closest available hospital ED. In addition, ambulatory patients will arrange transport to the closest facility utilizing non-EMS resources. In past events, this has overwhelmed the closest EDs. If these victims are also contaminated but not adequately decontaminated at the scene, an additional burden is placed on the hospital. Joint planning and exercises between hospitals, EMS providers, 911 service bureaus, and the fire system can circumvent these potential problems.
Successful management of the MCI requires the rapid establishment of a triage area for quick assessment of the arriving patients. Security personnel must protect and secure the triage and treatment areas, and assist with evidence preservation. Restricting access to essential personnel only will:

- Reduce evidence contamination and disruption
- Protect patients and hospital staff
- Prevent congestion of treatment and triage areas.

The hospital disaster plan will provide the framework for managing the various resources and support necessary in an MCI. This plan must utilize high-level decision-makers to ensure compliance. Realistic drills and table-top exercises are essential.

III. TRIAGE

A. Background

During an MCI, triage provides the organizational framework to provide the greatest good for the greatest number of casualties. The word triage is derived from the French word "trier," meaning to sort or to cull. It has its origins on the Napoleonic battlefields where Baron Dominick Jean-Larrey, Napoleon's chief medical officer, assigned surgical priorities to casualties according to the severity of their injuries.

B. Psychological Impact

In the U.S., medical resources are usually sufficient to meet the needs of the disaster. Consequently, most healthcare providers have little experience with true triage decision-making. It is difficult to deliver care in a selective fashion. The natural response is to try to save everyone. The psychological impact can be devastating when making these sometimes heart-wrenching decisions.

C. Classification

Many difficult decisions must be made under high stress conditions in an MCI. Utilization of a simple system of evaluation and classification will help ease the burden of dealing with an NBC event. A number of triage systems use color-coding to categorize and tag patients according to their severity and need for treatment during the

initial triage. The actual triage system used will vary depending on local protocol. The traditional triage method uses a
four-tiered system that categorizes patients in the following manner:

Red: **Immediate** or critical (seriously injured, but have a reasonable chance of survival)

Yellow: **Delayed** (can wait for care after simple first aid – that is, wounds dressed, splints applied).

Green: **Minimal** or "walking wounded" (no impaired function, can self-treat or be cared for by nonprofessional)

Black: **Expectant** or non-salvageable

Experience has shown this four-tiered system to be time consuming when confronted with large numbers of casualties. In addition, there appears to be considerable variability in the triage category assigned, depending on the experience and training of the triage officer. A variation of this four-tiered system is called Simple Triage and Rapid Treatment (START), and quickly classifies patients as immediate, delayed, or expectant. This system most closely resembles the triage method used daily in most EDs. Although START was originally designed for the pre-hospital environment, it can be easily applied to the hospital setting. Regardless of the triage method used, hospital personnel should be familiar with the local pre-hospital protocols for classifying patients during an MCI.

D. Simple Triage and Rapid Treatment System

Many jurisdictions across the U.S. are using the START system because of its simplicity. Individuals with very little medical training can effectively use the system. START merely requires an understanding of basic first aid. Under START, all victims who are able to walk on their own ("walking wounded") are directed to a pre-designated area and are labeled as **minimal** (green tag). This reduces the number of victims to be evaluated. These victims will require supervision and might be detained for further assessment and possible decontamination.

The remaining victims will be evaluated using the START triage system. This should take no longer than 1 minute per patient and will focus on three primary areas:

- Respiratory status
- Perfusion and pulse
- Neurological status.

As the responder moves through each level of assessment, any condition that is deemed **immediate** (red tag) stops the evaluation process. Life-threatening injuries will be addressed, if necessary, during the initial triage. The patient is tagged, and the responder moves on to the next patient.

Ventilation. If the patient is adequately ventilating (breathing), the triage officer moves on to the next step. If, however, ventilation is inadequate, the triage officer attempts to clear the airway by either repositioning the victim or clearing debris from the patient's mouth. If these attempts are unsuccessful, the victim is classified as follows:

- No respiratory effort: **expectant** (black tag)
- Respirations greater than 30 or needs help maintaining airway: **immediate**
- Normal respirations: move on to next step.

Perfusion. Initial evaluation is made by measuring capillary refill. If the casualty has normal capillary refill (less than 2 seconds), the triage officer moves on to the next step. If the patient's blood return is delayed (greater than 2 seconds) or the patient appears cyanotic, then the patient is classified as **immediate**. If the triage officer is unable to obtain capillary refill due to either the patient's color or poor lighting conditions, then the radial pulse is checked. If present, the pressure is assumed to be adequate (80mm Hg), the triage officer moves on to the next step. If the patient's radial pulse is not detected, the patient is classified as **immediate**.

Neurological status. The third and final level of assessment is the patient's neurological status. Depending on the level of consciousness, the following classification is made:

- Unconscious: **immediate**
- Altered level of consciousness: **immediate**
- Change in mental status: **immediate**
- Normal mental responses: **delayed, then move to next victim.**

F. Hospital Triage

Hospital triage is performed daily in the ED. Although patients may not be formally "tagged," they are still assessed and categorized as to their need for emergency care. Multiple trauma patients, or those complaining of chest pain or shortness of breath, are usually seen immediately (red tag). The medical care provided to those with less critical injuries or complaints is typically delayed (yellow tag) and provided on a first come, first serve basis. These same principles of triage apply to a mass casualty event, but the volume of patients will obviously be greater and the need for additional support will be heightened. Implement a familiar, quick, and efficient triage system that will allow hospital personnel to quickly process the increased volume of patients. This will hopefully avoid relocating the disaster to the hospital.
A formal color-coded tagging system is infrequently used in the hospital, except during disaster drills. When confronted with the need to evaluate numerous patients simultaneously in an MCI, hospitals quickly move into their "disaster-mode" and shift from the traditional triage method to one that is typically unfamiliar and rarely practiced. Compounding this problem is the myriad of commercial tagging systems available and the lack of uniformity in using one particular system among the various EMS providers in the community. If a formal tagging (or charting) system is used, it should be

implemented and practiced regularly to ensure staff familiarity. Many EMS providers routinely use triage tags once a week to maintain their competency. This policy can easily be implemented at the hospital.
Triage during an MCI is a continuum from the incident scene to the hospital with constant reassessment of patients, as resources permit. Re-triage at the hospital should occur because the victim's medical status may have changed, the priority for treatment may be different from that afforded at the scene, or the triage tag may have become unreadable, detached, or may not conform with the hospital's tagging system.
The triage area should be located separately from the ED, but near its entrance to reduce congestion and to maintain the normal traffic flow of emergency vehicles. The designated triage area needs to be well lit and shielded from inclement weather, but always accessible to rescue vehicles and the decontamination area. Security of the triage area is essential to limit access of unauthorized individuals who may hinder function and detract from the privacy that victims must be afforded. All individuals should be routed through one guarded and well-marked entrance. Hospital doors should be locked and guarded to prevent the public and injured walk-ins from entering the facility unnoticed.

G. Triage of Nuclear, Biological, and Chemical Casualties

Victims of biological attacks rarely exhibit signs and symptoms at the time of the incident. Rather, specific infectious patterns develop that should arouse suspicions of the unusual nature of the epidemic. For example, doctors and nurses suddenly begin treating a particularly high number of "sick" persons who were previously healthy and now exhibit similar signs and symptoms. In addition, an atypical influenza-like epidemic or clusters of patients presenting with peculiar symptoms (fever and bleeding) are clues worthy of further investigation. Triage becomes a management of resources. Increased demands for hospital rooms with isolation capabilities, mechanical ventilators, antibiotics (ciprofloxacin, doxycycline), and personal protective equipment (PPE) should be addressed. It is essential to quickly assess and categorize victims as infectious (immediate) versus psychosomatic (delayed) to ensure adequate distribution of resources. Reliable information concerning the nature of the disease will help personnel make these triage decisions.

In chemical and radiological attacks, the four triage categories can be applied as follows:

1. Nerve Agents. Unconscious or convulsing casualties, or those with major disorders of two or more body systems are triaged as **immediate**. Immediate treatment should include antidote administration and positive pressure ventilation to preserve their airways. Rapid intervention will result in an improved outcome.
Nerve agent casualties are categorized as **delayed** if their initial symptoms are either improving. Antidote treatment of these patients is dependent on the amount of antidote available. If supplies are limited, then **immediate** patients will be treated first. The **delayed** category is also used for patients recovering from exposure after treatment who are conscious and have an improved respiratory status. These patients may need additional treatment and definitely need to be observed for several hours.

The **minimal** nerve agent casualty is walking and talking and indicates intact breathing and circulation. These patients may be able to assist with other patients and/or decontamination.
The patient who has been apneic for more than 5 minutes and has no pulse or blood pressure is categorized as **expectant**.

2. Mustard. Most mustard casualties are triaged as **delayed**. However, patients with moderate to severe pulmonary signs and symptoms are classified as **immediate**. Casualties with burns covering 5 to 50 percent of their body surface area (BSA) or with eye involvement are **delayed** and those with burns on less than 5 percent BSA are **minimal**. The **expectant** casualty is the victim with liquid mustard burns greater than 50 percent BSA or no respiration or pulse.

3. Cyanide. Few signs and symptoms are visible except at very high doses. Severe cyanide exposures require rapid intervention and are categorized as **immediate**. In these patients, convulsions occur after 30 seconds, respiration ceases after 3 to 5 minutes, and death ensues in 6 to 10 minutes. Casualties with lower dose exposures have headaches, nausea and vomiting, hyperventilation, and dizziness, and should be categorized as **delayed**. These symptoms will improve if the patient is removed from the source of exposure.

Pulmonary Intoxicants. Patients who require **immediate** attention are those who develop noncardiogenic pulmonary edema within 6 hours after exposure to a pulmonary intoxicant such as phosgene, where intensive care unit (ICU) support is readily available.

Delayed casualties are those who develop cough and dyspnea more than 6 hours after exposure. These casualties should be admitted and observed for the development of latent pulmonary edema. When this dose of irradiation is combined with burns, then the prognosis is much more severe.

Expectant casualties are those who develop non-cardiogenic pulmonary edema within 6 hours of exposure, in circumstances where, due to limited resources, ICU support is not readily available in a mass casualty circumstance.

4. Nuclear. Patients who require **immediate** attention are those with traumatic injuries such as crushing extremity wounds, incomplete amputations, severe burns of face and upper respiratory tract, and difficulty breathing due to mechanical problems.

Delayed casualties include those with traumatic injuries that are non-life-threatening such as simple fractures, or second and third degree burns less than 25 percent of BSA.

Minimal casualties are those with burns less than 10 percent of BSA, but not involving critical areas or those who have received short-term body ionizing radiation doses of 100 to
150 RADs. When this dose of irradiation is combined with burns, then the prognosis is much more severe.

Expectant casualties have severe burns greater than 30 percent BSA, critical injuries to the respiratory or nervous system, or have received lethal doses of total body radiation, as indicated by a combination of clinical signs, including high fever, disorientation, bloody diarrhea, or vomitus.

IV. DECONTAMINATION IN A MASS CASUALTY INCIDENT

Planning for decontamination in an NBC MCI requires cooperation between pre-hospital and hospital personnel to ensure a successful process. Ideally, plans should include what type of support the Hazardous Materials Response Teams (HMRTs) will be able to provide to the hospitals in an NBC event. During the planning phase, a number of assumptions can be reasonably made. First, the majority of victims will be decontaminated at the hospital rather than at the scene of the incident. This occurs for a number of reasons:

- Victims preferentially triage themselves to the hospital
- Bystanders triage victims to the hospital
- Inherent delays in setting up decontamination discourage victims from remaining at the scene awaiting care
- General lack of awareness of decontamination availability at the scene
- The hospital is perceived to be a safer and more secure environment.
- In routine HAZMAT accidents, only about 18.5 percent of victims are treated at the scene [Agency for Toxic Substance and Disease Registry (ATSDR), 1993]. The vast majority of the remaining patients receive their initial decontamination and treatment at the hospital. After the sarin nerve agent attack in Tokyo, only about 10 percent of the victims arrived by ambulance.

Secondly, chemical and biological agents will be disseminated as either a vapor (chemical) or an aerosol (biological) to impact large numbers of people. Planning to mechanically decontaminate all of these individuals (that is, soap and water showering, with or without bleach pretreatment) presents many unique challenges for planners to consider. ∑ How many patients should hospitals prepare to decontaminate?

- How many persons will be needed to set up the decontamination area and be trained to perform the procedure?
- What level of PPE and training should be provided for treating personnel?
- How will patients' personal belongings be secured and privacy maintained?

- How will decontamination runoff be contained?

Experience has shown that 80 percent of the decontamination process is achieved by merely removing the victim's clothing. This is true for liquid chemical or external radiation contamination and should be followed by soap and water decontamination. Patients with a pure vapor exposure do not require additional decontamination beyond clothing removal.

The final assumption is that decontamination is important. Showering with high-flow water will greatly reduce the amount of contaminant remaining on the skin, minimizing the risk of secondary contamination. The challenge is to make decontamination available for NBC events and MCIs. The goal to quickly and effectively decontaminate patients will be unsuccessful if both hospitals and pre-hospital providers are not fully committed to this level of preparedness.

During the Persian Gulf War, hospitals in Israel were individually prepared to treat hundreds of NBC-contaminated patients. Although this may seem like a lofty goal in the U.S., every hospital should have the ability to treat at least one contaminated victim. When this is successful, each hospital can then build on this capability and prepare to decontaminate additional patients. It only takes one contaminated patient to close down an entire ED. Personnel involved in the decontamination process must receive appropriate Occupational Safety and Health Administration (OSHA)-level hazardous materials training consistent with their specific roles and responsibilities (see Section 29 of the Code of Federal Regulations,
29 CFR 1910.120).

In the pre-hospital environment, large numbers of contaminated casualties can be showered using a combination of ingenuity and current technologies. Examples include mobile trailers designed for mass decontamination; portable showers and collection pools; long hoses designed to provide a fine spray set up in corridors through which victims walk; and the use of a deck gun with wide-angle spray to rinse as many people as possible at one time. **Plan and train for what is reasonable in your community and then build on your capabilities.**

V. BOMB BLASTS

A. Mechanics of an Explosion

The dissemination of NBC materials as aerosols may often be attempted through the use of bombs or explosives. When these bombs are detonated, the reaction produces an instantaneous chain of events in which the explosive material is rapidly converted into a gas under extremely high pressure and temperature. This gaseous byproduct is transmitted to the surrounding medium as a blast wave (or shock wave) that travels outward from the explosion.

After the explosion occurs, a mass movement of air (blast wind) that was originally displaced by the explosive products follows the explosion at speeds that can reach hurricane proportions. This blast wind may be as damaging as the original explosion. This type of reaction occurs when high energy explosives are used (such as plastic explosives, TNT, diesel fuel, and fertilizer). High-energy explosives detonate faster than

the speed of sound. In low-energy explosives (such as a gunpowder pipe bomb), the pressure within the casing increases so rapidly that it explodes, releasing high-velocity shrapnel as its most deadly by-product. Low-energy explosives react slower than the speed of sound.
If a solid structure such as a wall or building is present in the path of the explosion, the blast wave will rebound off this structure and generate a reflective force that is magnified almost nine times its original strength. As a result, victims caught between the blast and a building may suffer injuries two to three times greater than expected for the amount of explosive detonated and the distance from the explosion.

B. Explosive Properties

The larger the explosives charge, the greater the velocity and duration of the shock wave will be. Together, these two factors contribute to the severity of the explosion. Explosions that occur within confined spaces (such as inside sporting facilities, restaurants, and buildings) injure and kill by a variety of methods, including:

- Direct exposure to the blast wave
- Reflective blast waves
- Acceleration-deceleration forces
- Penetrating and nonpenetrating impact of blast debris
- Burns from the flash and hot gases of the explosion, and the combustion of the surroundings
- Inhalation of toxic gaseous byproducts
- Building collapse.

C. Mechanism of Injury

After the bomb explodes, the sudden change in pressure causes a variety of injuries that are divided into four main categories.

1. Primary blast injuries. These injuries occur when the blast wave travels through the body, damaging organs and tissues that have air and fluid (blood) in contact with each other. This is most readily seen in the lungs, ears, bowel, heart, and brain. As the blast wave strikes in these organs, the blood, which is a more dense and noncompressible tissue, is either thrown (spalling) or pulled into the less dense air containing tissue, resulting in injury. For example, when a blast wave strikes and begins to pass through the chest, the pressure of the blast wave forces the blood of the pulmonary vasculartur into the less dense air cells of the lungs (alveoli). As the blast wave passes through,

additional blood from the pulmonary vasculature is then "pulled" into the lung tissue. Both processes combine to cause hemorrhage.
In addition, when the blast wave passes through an organ containing pockets of air (such as the middle ear, lungs, and intestines), the pressure of the wave forces the surrounding tissue and fluid to compress the air within. Once the shock wave passes, the compressed air re-expands with a greater intensity, causing miniature explosions called "implosions."

2. Secondary blast injuries. These injuries occur from the rapid acceleration of small debris such as flying glass and shrapnel produced from the explosion. These small fragments may be accelerated to velocities capable of causing skin lacerations and body cavity penetrations. The energy from the shrapnel (related to mass and velocity) is transmitted directly and completely to the traumatized tissue, causing fractures to bones and massive soft tissue damage.

3. Tertiary blast injuries. These injuries occur when a victim is thrown into the air from the force of the explosion (blast wind) and pushed into a stationary object. If a 70-kilogram (kg) person is accelerated into a solid vertical object at 18 miles per hour, 50 percent mortality can be expected.

4. Miscellaneous blast effects. These include flash injuries from the thermal component of the explosion, burns from secondary fires started from the blast, and crush injuries resulting in kidney failure and sepsis. Inhalation of toxic fumes or exposure to NBC contaminants is also possible. Neuropsychiatric conditions such as amnesia, temporary blindness, or paresthesias are common.

D. Injury Patterns

Most victims who survive a bomb blast will suffer from some degree of secondary and/or tertiary bomb injuries. Primary blast injuries, beyond injuries to the ear (such as eardrum rupture, nerve injury) are infrequently seen in survivors. Individuals who suffer primary blast injuries are usually so close to the explosion that they are typically killed by the secondary and tertiary blast effects. They die from brain injuries, skull fractures, diffuse lung contusions, liver and spleen lacerations, or traumatic amputations. There are, however, exceptions to this general rule. For example, after a recent bus bombing in Israel, a number of survivors were found to have primary blast injuries to the lung and gut.
From a number of terrorists bombing studies, only about 15 percent of survivors require hospital admission. Most of these individuals suffered multiple injuries, but their admission was related to one single cause such as a concussion, fracture, or burn. Most victims are treated and released from the ED.

E. General Management

The basic principles of trauma life support emphasize life-saving intervention (ABCs). Oxygen should be used liberally for those complaining of shortness of breath. Respiratory assistance (that is, bag-mask ventilation or intubation) should be provided

with care, especially in those patients suspected to have primary blast injury to the lungs (that is, short of breath and hypoxic). In these patients, the torn lung tissue and damaged blood vessels are in direct communication with each other, increasing the likelihood of air entering the vasculartur and causing an air embolism. These patients will require high frequency/low pressure ventilation. In addition, the increased pressure generated from mechanical ventilation may cause air to leak out of the damaged alveoli and collect in the pleural space, resulting in a pneumothorax. If this were to occur, chest tube placement would be required (preceded by needle decompression in the case of a tension pneumothorax).

Air embolism appears clinically as dyspnea, tachycardia, hypoxia, tachypnea, chest pain, altered mental status, anxiety, and syncope. Treatment of an air embolism initially requires the patient to lie on their left side with legs elevated (Trendelenburg position). Hyperbaric therapy is the preferred treatment and must be instituted quickly. Injured extremities should be splinted. Intravenous (IV) fluids should be used in a gentle manner to prevent further harm to the blast-injured pulmonary tissue.

Wound management takes on great importance because the amount of tissue damaged from an explosion is typically severe. The bodily injuries from terrorist bombings are caused by high-velocity, irregularly shaped shrapnel and debris that result in extensive tissue destruction and contamination. For these reasons, adequate and extensive surgical debridement is essential and primary closure (sutures) should be delayed for at least 5 days. The use of tetanus toxoid and broad-spectrum antibiotics should be provided liberally. Any delay in providing immediate and appropriate medical care to critically injured victims has been shown to markedly increase mortality.

Bombing casualties that can walk and talk, who are alert and oriented, and have intact hearing are triaged as **minimal**. However, those who have experienced a decrease or loss of hearing may have suffered trauma from the blast and are placed in the **immediate** category. These patients should be observed closely for at least 6 to 12 hours after the incident since blast injuries may not always be apparent when the victim is first evaluated. In a study of victims after a bus bombing in Israel, two victims had serious gut injuries that were missed for 3 to 7 days after the explosion.

Bombs may be secondarily contaminated with NBC materials. If suspected or detected, these victims should be decontaminated. Any contaminated foreign bodies that remain in the wound require emergency surgical assessment and removal, as possible. These retained materials, as well as the victim's clothing, will need to be preserved as criminal evidence. These items should be labeled and secured by as few individuals as possible to maintain the integrity of the chain of evidence. When possible, it is advisable to have one person in charge of the evidence. This individual need not have any particular expertise in NBC weaponry, but be experienced and trained in the maintenance of evidence (similar to what is done routinely to preserve evidence from rape exams). A log or other suitable type of notation system should be maintained to assist in clearly identifying and accounting for the evidence.

Contaminated clothing should be removed and bagged (paper bags for explosives, paper bags into plastic bags for chemicals and explosives). Foreign bodies suspected to be contaminated with either chemical weapons or biological agents should be removed and placed in a container of 5 percent hypochlorite solution (household bleach), sealed, and labeled for law enforcement removal. Radioactive materials removed from a wound

should be placed in specialized lead containers obtained from the Radiation Safety Officer (RSO). Physicians removing these foreign bodies will need to take special precautions to prevent contamination of themselves and others. This may require surgical personnel to wear chemical protective gloves (for example, nitrile), a lead shield, and a respirator, in addition to routine surgical garb, as appropriate. These contaminated wounds should be adequately irrigated with sterile water and covered with a sterile dressing.

VI. ACUTE CRUSH SYNDROME

A. Description

Terrorists have commonly used high-energy explosives that cause structural collapse, crushing victims inside with heavy, fallen debris. These crushing objects place prolonged and continuous pressure on the extremities (skeletal muscles), resulting in skeletal muscle death (rhabdomyolysis) with release of its cellular contents (myoglobin) into the plasma. The adverse effects that result are called the **Acute Crush Syndrome.** After the skeletal muscle injury occurs and the crushing object is removed, all of those accumulated cellular toxins (myoglobin) and electrolytes (potassium) are released into circulation, causing lethal cardiac arrhythmias, acute renal failure, and sudden death.

B. Clinical Presentation

Clinical presentation will vary widely depending on the presence of NBC exposure, other associated injuries, and the length of time the extremity has been crushed. Victims may be alert, hypothermic, hypotensive, confused, or combative. The extremity may be pale, swollen, and have decreased sensation. Pulses may be present or absent. It is important to note whether the injury is closed or open, as this could significantly affect the victim's susceptibility to NBC contamination.

C. Management

The systemic effects of the Acute Crush Syndrome only occur when the crushing object is removed and the injured extremity is reperfused. Removal of the object causes massive fluid shifts into the injured muscle, resulting in acute hypovolemia and hypotension. **Large volumes of normal saline must be given to the patient intravenously (IV) both before and after the patient is freed**. One to two liters (L) (20 cc/kg) should be given as a bolus before being freed and then continued at a rate sufficient to maintain a central venous pressure of 5 mm/Hg or urine output of 300 to 500 cc/hr (will need a Foley catheter). Over 12 L may be required in adults during the first 24 hours. This volume of fluid ensures a continual blood flow to the kidneys that will help facilitate excretion of the toxic substances. Adding sodium bicarbonate to the IV solution can help prevent myoglobin deposition in the renal tubules. Mannitol may be necessary to help maintain diuresis and has been shown to function as a free radical scavenger, preventing some injury to the kidney. Elevated potassium levels should be treated

according to traditional protocols (that is, volume, calcium chloride, sodium bicarbonate, insulin, and glucose).

VII. KEY POINTS

Terrorist attacks are orchestrated to instill fear and panic while attaining political goals. The challenge to hospital personnel is to create some semblance of order in a very chaotic environment. This can be achieved through training. The cool, calm professional who follows a standard format for evaluating and treating a patient can have a favorable impact on the long-range outcome. Well disseminated and accepted protocols combined with practical exercises will enable hospital personnel to manage the incident appropriately. Training must include the use of PPE, patient decontamination, and antidote therapy. In addition, hospital personnel should participate in hands on or table top exercies.

The tasks assigned to personnel should parallel their normal daily activities. Avoid activities for which personnel are not adequately trained. For example, hospital personnel should not respond to the scene of an explosion to perform search and rescue. Appropriate PPE should always be worn. Triage areas should be secured, organized, and made highly visible and accessible. Utilize a single standardized triage method on a regular basis so that all personnel will become familiar with its use. This will ensure that the method can be easily and effectively applied in a mass casualty situation. Remember to plan for the medical needs of the unaffected community, because these needs will not diminish in the event of an MCI or NBC event. Rotate staff during an MCI to prevent staffing congestion and minimize fatigue. Train frequently using realistic scenarios. Develop and train with Crisis Intervention Teams for psychological assessment of the patients and support of the hospital providers. Trauma victims complicated by NBC agents should be treated according to standard Basic Trauma Life Support guidelines with decontamination and antidote therapy administered simultaneously, where feasible and appropriate.