



# **Disaster Response and Biological/Chemical Terrorism**

Information Packet

Prepared by: Emergency Medical Services  
Department

October 2001

## **Disaster Response and Biological/Chemical Terrorism Clearinghouse Information**

The College has been very active in disaster planning and response as well as biological/chemical terrorism preparation for a number of years. The membership section, Disaster Medicine, was the first membership section organized in 1989. They have been one of the largest in membership and one of the most active. The EMS Department maintains information for members and the public on disaster response and biological/chemical terrorism topics in a variety of formats and publications.

### **POLICY STATEMENTS**

The College has current policies addressing the following:

- Disaster Data Collection (Oct. 2000)
- Disaster Medical Service (June 2000)
- Support for National Disaster Medical System-NDMS (Mar. 1999)
- Handling of Hazardous Materials (June 1999)

### **TEXT BOOKS**

The College developed and published the text: Community Medical Disaster Planning and Evaluation Guide through a section grant with the Disaster Medicine Section. This is an excellent guide for developing or updating a hospital or city/county disaster plan. While biological or chemical agents are not specifically mentioned, many of the same disaster planning and response issues remain the same. This text can be ordered through the College bookstore at 1-800-798-1822, ext. 6 for \$69.00, item number 513000-1020.

### **NBC TASK FORCE**

The College's nuclear, biological and chemical (NBC) Task Force recently completed a grant with the Department of Health and Human Services (HHS), Office of Emergency Preparedness (OEP) to develop objectives, content and competencies for the training of emergency medical technicians, emergency physicians, and emergency nurses to care for casualties resulting from nuclear, biological, or chemical (NBC) incidents. This report contains the objectives for an Awareness and Performance level training course. A complete copy of the report is available on the ACEP website and the executive summary was published in the June 2001 Annals of Emergency Medicine.

### **DISASTER MEDICINE SECTION**

The College's Disaster Medicine Section is one of the largest and most active membership sections. Many members are active in disaster planning and response through the National Disaster Medical System (NDMS) and the Disaster Medical Assistance Teams (DMATS). These

members can be excellent resources for networking and as speakers for various presentations and training programs. Information on joining or organizing a local DMAT team can be obtained by calling 1 (800) USA-NDMS or their web site at <http://ndms.dhhs.gov>.

## **EDUCATIONAL MEETINGS**

The College's Educational Meetings Division is offering four disaster planning and weapons of mass destruction (WMD) courses during Scientific Assembly 2001 in Chicago. They are in the planning stages of a Hot Topics conference on Disaster Response/Terrorism planned for March 2002. Copies of speaker syllabi on disaster and terrorism topics are available upon request.

## **FACT SHEETS**

The College's Public Relations Department develops and maintains 'Talking Points' on many current issues. We have the most recent ones on biological and chemical terrorism.

- ACEP FACT SHEET – Nuclear, Biological, and Chemical Terrorism
- ACEP TALKING POINTS – America's Preparedness for Terrorism Attacks
- ACEP NEWS RELEASE – America moves forward in preparing for nuclear, biological, and chemical events
- ACEP NEWS RELEASE – Nations' emergency physicians ready to aid in disaster relief

## **PATIENT TREATMENT INFORMATION**

We have compiled patient treatment information sheets covering the recognition and initial treatment of the most common biological and chemical agents. Much of this information was obtained from military training programs.

- Initial Recognition and Management of Chemical Terrorism Casualties
- Initial Recognition and Management of Biological Terrorism Casualties

## **ANNALS ARTICLES**

Annals of Emergency Medicine routinely publishes articles relating to disaster management and biological/chemical terrorism. Attached are articles that have appeared since 1994.

### **Articles on Disaster Medicine/Emergency Preparedness**

As a service to the readers of *Annals of Emergency Medicine* and the specialty of emergency medicine, the journal will provide online full, free access to two special sections on Disaster Medicine and Emergency Preparedness at [www.mosby.com/AnnEmergMed](http://www.mosby.com/AnnEmergMed).

The first section on Disaster Medicine, originally published in the August 1999 issue, features eight articles. The articles address principles for emergency response to bioterrorism, emergency physicians and bioterrorism, chemical warfare agents (with information dedicated to the hazards of civilian use of gas masks), emergency department hazardous materials protocol for contaminated patients, telecommunications systems in support of disaster medicine, the

Emergency Department impact of the Oklahoma City terrorist bombing, and eardrum perforation in explosion survivors.

Additionally, the journal will provide full free access to the November 2001 special section on Disaster Medicine/Emergency Preparedness. These articles include "Hospital Preparedness for Weapons of Mass Destruction Incidents," "Comparative Analysis of Multiple-Casualty Incident Triage Algorithms," "Disaster Epidemiology and Medical Response in the Chi-Chi Earthquake in Taiwan," and "Were There Enough Physicians in an Emergency Department in the Affected Area After a Major Earthquake?" The November 2001 special section Web site posting will coincide with the mailing of that issue in early November.

## **WEB SITES**

Agency for Toxic Substances and Disease Registry (ATSDR)  
[www.atsdr.cdc.gov](http://www.atsdr.cdc.gov)

Centers for Disease Control and Prevention (CDC)  
[www.cdc.gov](http://www.cdc.gov)  
[www.bt.cdc.gov](http://www.bt.cdc.gov)

Department of Health and Human Services (DHHS)  
[www.dhhs.gov](http://www.dhhs.gov)

Environmental Protection Agency (EPA)  
[www.epa.gov](http://www.epa.gov)

Federal Emergency Management Agency (FEMA)  
[www.fema.gov](http://www.fema.gov)

Johns Hopkins Center for Civilian Biodefense Studies  
[www.hopkins-biodefense.org](http://www.hopkins-biodefense.org)

National Disaster Medical System (NDMS)  
Office of Emergency Preparedness (OEP)  
[www.ndms.dhhs.gov](http://www.ndms.dhhs.gov)

National Domestic Preparedness Office (NDPO)  
[www.ndpo.gov](http://www.ndpo.gov)

U.S. Army Medical Research Institute of Infectious Diseases (USAMRIID)  
[www.usamriid.army.mil](http://www.usamriid.army.mil)

U. S. Army Medical Research Institute of Chemical Defense (USAMRICD)  
<http://chemdef.apgea.army.mil/>

For additional information on disaster planning or the College's biological/chemical terrorism response, contact Rick Murray, ACEP EMS Manager at 800-798-1822 ext. 3260 or by e-mail at [rmurray@acep.org](mailto:rmurray@acep.org).

# ACEP Policy Statement

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## *Disaster Data Collection*

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Approved by the  
ACEP Board of Directors  
October 2000

The American College of Emergency Physicians (ACEP) believes that research in disaster epidemiology is critical for future disaster preparedness. Accurate data collection in a disaster can be difficult without government mandate and assistance. Although the public health system gathers mass epidemiologic data, public health departments play little role in disaster data collection. Therefore, ACEP supports the following:

- ‡ Public health systems and agencies should be incorporated into disaster planning and response.
- ‡ All injuries and illnesses related to officially declared disasters and terrorist events should be reported to public health agencies.
- ‡ All disaster-related injuries and illnesses should be incorporated into a disaster collection database to enhance local disaster response.

# ACEP Policy Statement

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## *Disaster Medical Services*

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Revised and approved by the ACEP Board of Directors June 2000.

This statement replaces one with the same title originally approved by the ACEP Board of Directors June 1985 and reaffirmed by the Board of Directors March 1997.

The American College of Emergency Physicians (ACEP) believes that emergency physicians should assume a primary role in the medical aspects of disaster planning, management, and patient care. Because the provision of effective disaster medical services requires prior training or experience, emergency physicians should pursue training that will enable them to fulfill this responsibility.

A medical disaster occurs when the destructive effects of natural or man-made forces overwhelm the ability of a given area or community to meet the demand for health care.

Disaster planning, testing, and response are multidisciplinary activities that require cooperative interaction. Each agency or individual contributes unique capabilities, perspectives, and experiences. Within this context, emergency physicians share the responsibility for ensuring an effective and well-integrated disaster response.

Emergency medical services and disaster medical services share the goal of optimal acute health care; however, in achieving that goal, the two systems use different approaches. Emergency medical services routinely direct maximal resources to a small number of individuals, while disaster medical services are designed to direct limited resources to the greatest number of individuals. Disasters involving the intentional or accidental release of biological, chemical, radiological, or nuclear agents present an extremely difficult community planning and response challenge. In addition, they may produce a far greater number of secondary casualties and deaths than conventional disasters. Because the medical control of emergency medical services is within the domain of emergency medicine, it remains the responsibility of emergency physicians to provide both direct patient care and medical control of out-of-hospital emergency medical services during disasters.

Improvement of established disaster management methods requires the integration of data from research and experience. Emergency physicians must use their skills in organization, education, and research to incorporate these improvements as new concepts and technologies emerge.

Where local, regional, and national disaster networks exist, emergency physicians should participate in strengthening them. Where they are not yet functional, emergency physicians should assist in planning and implementing them.

# ACEP *Policy* *Statement*

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## *Support for National Disaster Medical System*

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*Approved by the  
ACEP Board of Directors  
March 1999*

*This statement replaces  
Council Resolution CR056  
approved by the ACEP  
Board of Directors  
September 1985 and  
Council Resolution CR019  
approved by the ACEP  
Board of Directors  
October 1991.*

The American College of Emergency Physicians (ACEP) believes that every community needs a comprehensive plan for immediate emergency medical care in case its medical care system is overwhelmed or rendered inoperable in a disaster. As a component of this plan, ACEP supports the National Disaster Medical System (NDMS). ACEP also supports memberships in Disaster Medical Assistance Teams under the auspices of NDMS and encourages agencies such as health care facilities and EMS services and employers such as medical practice groups to allow their employees to participate.

# ACEP *Policy* *Statement*

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## *Handling of Hazardous Materials*

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*Approved by the  
ACEP Board of Directors  
June 1999*

*This policy statement  
replaces Council Resolution  
CR036 titled "Hazardous  
Materials, Access to  
Information" approved  
September 1985 and Council  
Resolution CR026 titled  
"Hazardous Materials;  
Transportation of Hazardous  
Materials" approved  
September 1988.*

The American College of Emergency Physicians (ACEP) believes that nuclear, chemical, and biological hazardous materials pose a significant risk to individuals and communities if improperly handled or if released accidentally or intentionally into the environment.

- Individuals who are at risk, including emergency personnel, have the right to know when these materials are used in or transported through their communities.
- Emergency personnel must have immediate access to all information necessary to treat victims, protect themselves, and prevent exposure of others.
- Hazardous materials should be clearly and appropriately marked.
- Vehicles transporting hazardous materials should be clearly marked that they are used for such purposes, and drivers of those vehicles should be educated in the safe transport of hazardous materials.
- Emergency personnel responsible for the care and treatment of victims of exposure to hazardous materials should be appropriately educated and trained in methods of self-protection, patient protection, and resuscitation.
- Administrative and clinical guidelines should include principles of decontamination of personnel, patients, and vehicles, minimum equipment requirements, and recommended safety procedures.

ACEP supports state and federal policies that promote adherence to these principles.



## **Disaster Medical Assistance Team**

A Disaster Medical Assistance Team (DMAT) is a group of medical and support personnel designed to provide emergency medical care during a disaster or other unusual event. DMATs deploy to disaster sites with adequate supplies and equipment to support themselves for a period of 72 hours while providing medical care at a fixed or temporary medical site. They may provide primary health care and/or augment overloaded local health care staff. DMATs are designed to be a rapid-response element to supplement local medical care until other Federal or contract resources can be mobilized, or the situation resolved.

Each DMAT deployable unit consists of approximately 35 individuals; however, teams may consist of more than three times this number to provide some redundancy for each job role. This insures that an adequate number of personnel are available at the time of deployment. The team is composed of medical professionals and support staff organized, trained, and prepared to activate as a unit.

## **National Disaster Medical System**

The National Disaster Medical System is an asset sharing partnership designed to provide emergency medical assistance to States following a catastrophic disaster or other major emergency. The system is designed to care for victims of any incident that exceeds the medical care capability of the affected local and State resources. The Department of Health and Human Services in partnership with other Federal agencies such as the Department of Defense, Department of Veterans Affairs, and the Federal Emergency Management Agency administer the program. The NDMS has three primary objectives:

To provide health, medical, and related social service response to a disaster area in the form of medical response units or teams and medical supplies and equipment;

To evacuate patients who cannot be cared for in the affected area to designated locations elsewhere in the nation; and

To provide hospitalization in Federal hospitals and a voluntary network of non-Federal acute care hospitals that have agreed to accept patients in the event of a national emergency.

Information on joining or organizing a local DMAT team can be obtained by calling 1 (800) USA-NDMS or through their web site at <http://ndms.dhhs.gov>.

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## AMERICA'S PREPAREDNESS FOR TERRORIST ATTACKS

- Emergency physicians are deeply grieved over the terrorist attacks of September 11, 2001, and the senseless loss of lives in New York City, Washington, DC, and Pennsylvania.
- Emergency physicians are committed to treating patients and to meeting the nation's emergency care needs. Many participated in disaster relief efforts, serving in FEMA urban search and rescue teams and National Disaster Medical System teams.
- Most EDs and hospitals have policies and procedures for responding to hazardous materials (HAZMAT), but many need to be updated to respond to biological agents.
- Most prehospital and emergency medical personnel in the United States need additional educational and training preparation, training, and equipment to deal with incidents involving weapons of mass destruction.
- The threat of bioterrorism is probably low, because of many challenges included those related to obtaining and dispersing biologic agents. However, it represents one of the greatest long-term threats to our nation.
- A biological attack on a major city could be as lethal as a nuclear explosion. However, the effects of a biological agent may not be apparent until days after an attack because of the longer incubation period or early non-specific symptoms. A highly contagious organism, such as small pox, also could spread rapidly in an urban environment, assisted by modern transportation networks.
- Biological agents can be potentially catastrophic, impacting thousands. Technological advances, easy access to organisms, and availability of technical information may contribute to the proliferation of biological warfare agents. Preparing for incidents should involve specialized education and training of emergency personnel, specific disaster planning, public education, and local stockpiling of appropriate antidotes.
- America is at a crossroads in terms of readiness to respond to biological terrorism, and emergency physicians are key to helping ensure that hospitals and communities are prepared and that collectively our nation is ready to respond to bioterrorism.
- Emergency physicians should assume a primary role in the medical aspects of disaster planning, emergency medical management, and patient care. Working with a grant from the U.S. Department of Health and Human Services, an ACEP taskforce identified the content of a national curriculum to train emergency care providers to respond to incidents involving weapons of mass destruction. This project should be completed and implemented. Professional associations of emergency physicians and nurses are the most effective providers of this kind of training due to their ongoing educational programs, certification process and communication capabilities.

To be prepared to respond to a terrorist attack, the nation must address critical areas in national strategy: preparedness, response, and research, which will require leadership from Federal state, and local governments; the medical and health communities, and the public.

- Develop specific strategies for responding to all potential weapons of mass destruction.
- Strengthen the public health infrastructure. Many diseases have been eradicated in the United States, and over time, the nation's public health system has eroded and has become less effective for tracking and reporting diseases.

- Train emergency health care personnel to recognize rapidly and treat victims of chemical and biological terrorism and to report such incidents.
- Maintain stockpiles of antibiotics, antidotes, and vaccines for use in the event of an attack, along with medical equipment and supplies.
- Designate and give adequate authority to a Federal central office that can integrate the various agencies at the Federal, state, and local levels involved in emergency response.
- Establish, strengthen, and expand sophisticated detection and analysis surveillance systems focused on all forms of biologic agents. Ensure their integration with public health systems and the nation's emergency departments.
- Establish and sustain educational programs for health care workers and the public. Involve emergency physicians who are responsible for establishing policies and protocols for the nation's Emergency Medical Services systems.
- Conduct research focused on improving detection, investigation, diagnosis, and treatment for these threats. Improvement of established disaster management methods will require the integration of data from research and experience.
- Ensure that local communities have comprehensive disaster response plans that integrate all the required responders, including medical, law enforcement, fire, EMS, and government.
- What should the public do?
  - People should be assured that there is no need to personally stockpile antibiotics and gas masks. The length of time that antibiotics remain useful varies, and increased use by the public could result in bacterial infections resistant to antibiotics — another significant public health problem. Antibiotics for treating anthrax also are expensive and must be taken for long periods of time. The Centers for Disease Control and Prevention maintains stockpiles of pharmaceuticals, which can reach victims anywhere in the continental U.S. within 12 hours. Gas masks would be useless against bioterrorism unless people wear them at all times, and can be dangerous.
  - People should promote preparedness among the medical community, hospitals, and government officials. For example, they should ask physicians whether they are trained to recognize the presentation of biologic agents and ask hospitals and local government officials whether they have adequate disaster plans to respond to terrorist attacks.
  - For any kind of disaster, it's a good idea for people to develop a disaster supply kit, which would include such items as water, food, battery-powered radio, flashlights and extra batteries, first aid kit and manual, blankets, duct tape, matches in a waterproof container, medications and photocopies of prescriptions, list of important phone numbers, special items for babies and the elderly, spare set of car keys, credit card and cash, and area map.

**NEWS RELEASE**For Immediate Release  
May 7, 2001Contact: Laura Gore  
Colleen Horn  
202-728-0610, ext 3006**AMERICA MOVES FORWARD IN PREPARING FOR NUCLEAR,  
BIOLOGICAL, AND CHEMICAL EVENTS**

Dallas, TX — The American College of Emergency Physicians (ACEP) and the U.S. Department of Health and Human Services (HHS) took a major step forward in preparing the nation's health care providers to respond to nuclear, biological, and chemical events.

ACEP and HHS released a report, funded by the HHS Office of Emergency Preparedness, evaluating current training programs, analyzing barriers to implementing training, and establishing objectives for developing and integrating training materials for emergency responders to care for casualties of nuclear, biological, or chemical incidents.

The report was distributed in April during the annual conference of National Disaster Medical Systems (NDMS), attended by local, State and National leaders in emergency preparedness planning. During the meeting, ACEP was recognized for its long-term support of the NDMS system.

“The catastrophic nature of damage from weapons of mass destruction demand that local communities be prepared,” said Robert W. Schafermeyer, MD, president of ACEP. “The best defense in reducing casualties will be the ability of community leaders to mount an appropriate response in a timely fashion and to sustain that response until appropriate help is available from outside the community. The objectives in this report move the nation another step closer toward being prepared.”

According to the report, adequate training and education programs are not available that sufficiently address the full range of knowledge and skills required by health care professionals. To address these needs, the report contains education objectives, core content, and guidelines for developing and integrating national training curricula for emergency responders and health care professionals.

“For the first time the emergency health care community has a credible benchmark for curriculum development to support education and performance in caring for casualties of nuclear, biological and chemical incidents,” said Dr. Robert F. Knouss, Director, HHS, Office of Emergency Preparedness and National Disaster Medical System.

The report finds that to be successful the response training must meet the needs of a large, diverse audience, providing them not only awareness-level knowledge, but also skills that can be practiced, demonstrated, and maintained. It also must become an integral part of the education and subsequent knowledge base of emergency health care professionals. In addition, since the nature of weapons of mass destruction changes rapidly, the knowledge base and curriculum must evolve to ensure health care providers maintain levels of proficiency.

(MORE)

The report said that local community health care systems make up the human infrastructure critical to providing early recognition and response, which will minimize devastation. Therefore, efforts to provide seamless patient care from the out-of-hospital to the hospital setting must be integrated and involve many key individuals, including hospital administrators, local and State emergency planners, law enforcement personnel, poison centers staff, laboratory agencies staff, industry personnel, public health officials, safety officers, and medical and nursing specialists.

Copies of the report are available from ACEP (800-798-1822, ext 3260) and at ACEP.org. The following government and medical organizations supported and/or participated in the ACEP-led task force:

**Government**

U.S. Department of Health and Human Services, Office of Emergency Preparedness  
Federal Emergency Management Agency

**Medical Organizations**

American College of Emergency Physicians  
American Board of Emergency Medicine  
American College of Medical Toxicology  
American Hospital Association  
American Nurses Association  
Association for Professionals in Infection  
Emergency Nurses Association  
International Association of Fire Chiefs  
National Association of Emergency Medical Services Physicians  
National Association of Emergency Medical Technicians  
National Registry of Emergency Medical Technicians  
National Association of State Emergency Medical Services Directors  
Society for Academic Emergency Medicine

For questions regarding the report or task force activities, please contact Rick Murray, ACEP EMS Manager, by email at [rmurray@acep.org](mailto:rmurray@acep.org), or by telephone at 800-798-1822, ext. 3260.

ACEP is a national emergency medicine medical specialty society with more than 21,000 members. ACEP is committed to improving the quality of emergency care through continuing education, research, and public education. Headquartered in Dallas, Texas, ACEP has 53 chapters representing each State, as well as Puerto Rico and the District of Columbia. A Government Services Chapter represents emergency physicians employed by military branches and other government agencies.

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American College of  
Emergency Physicians®

2121 K Street, NW., Suite 325  
Washington, DC 20037

## NEWS RELEASE

For Immediate Release  
September 13, 2001

Contact: Laura Gore  
Colleen Horn  
(202) 728-0610, ext 3006

### Nation's Emergency Physicians Ready To Aid in Disaster Relief, ACEP Reports

Washington, DC — Saying that emergency physicians across the United States and throughout the world have been contacting the American College of Emergency Physicians (ACEP) to volunteer their services for the rescue efforts in New York and Washington, DC, Dr. Robert Schafermeyer, president of ACEP, today expressed gratitude and pride at the heroic actions of the nation's emergency response workers.

“Offers of assistance from emergency physicians, not only in the United States but in countries such as Turkey, Germany, and Iceland, have been pouring into ACEP's headquarters in Dallas since news was heard around the world about this devastating attack. Although not all of us have been called upon at this time, these selfless offers of support have made me proud to be an emergency physician and a member of ACEP.

“Like all of America, we mourn with those who are suffering devastating losses, and we are resolved to strengthen and promote healing for our nation in the wake of these despicable attacks. I join the nation in expressing gratitude for all the heroic efforts of physicians, nurses, police, EMS, fire personnel, and lay rescue workers in New York, Washington, DC, and Pennsylvania, especially those who have lost their lives.

“The thoughts and prayers of emergency physicians are with the victims and families of this senseless violence. May God Bless America as we face the trying days ahead.”

Emergency physicians are committed to working with national officials to continue to improve U.S. emergency response efforts to medical disasters. ACEP and the U.S. Department of Health and Human Services in April released a report, funded by the HHS Office of Emergency Preparedness, evaluating current training programs, analyzing barriers to implementing training, and establishing objectives for developing and integrating training materials for emergency responders to care for casualties of nuclear, biological, or chemical incidents.

ACEP is a national emergency medicine medical specialty society with more than 22,000 members. ACEP is committed to improving the quality of emergency care through continuing education, research, and public education. Headquartered in Dallas, Texas, ACEP has 53 chapters representing each state, as well as Puerto Rico and the District of Columbia. The Government Services Chapter represents emergency physicians employed by military branches and other government agencies.

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# Initial Recognition and Management of Chemical Terrorism Casualties

## Five types of chemical agents:

Nerve Agents – inhibit the enzyme acetylcholinesterase and results in excess acetylcholine. Nerve agents covered are GA (tabun), GB (sarin), GD (soman), GF, and VX.

Vesicants – causes vesicles (blisters) on the skin, damages the eyes and airways. Vesicants include mustard (sulfur mustard: H, HD), Lewisite (L), and phosgene oxime (CX).

Cyanide - Potential agents are hydrocyanic acid (AC) and cyanogen chloride (CK).

Pulmonary Agents - Possible agents include phosgene (CG), Perfluoroisobutylene (PFIB), a product of Teflon combustion and HC smoke (smoke containing zinc).

Riot Control Agents - Major agents include CS and CN (Mace).

## Nerve Agents

GA (tabun) GB (sarin) GD (soman) GF VX

## Summary

### Signs and Symptoms:

**Vapor:** *Small exposure*—Miosis, rhinorrhea, mild difficulty breathing. *Large exposure*—Sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions, miosis.

**Liquid on skin:** *Small to moderate exposure*—Localized sweating; nausea, vomiting, feeling of weakness. *Large exposure*—Sudden loss of consciousness, convulsions, apnea, flaccid paralysis, copious secretions.

Decontamination: Hypochlorite; large amounts of water

Immediate management: Administration of **MARK I's** (atropine and pralidoxime chloride); diazepam in addition if casualty is severe; ventilation and suction of airways for respiratory distress

## OVERVIEW

Nerve agents are the most toxic of the known chemical agents. They are hazards in their liquid and vapor states and can cause death within minutes after exposure. Nerve agents inhibit acetylcholinesterase in tissue, and their effects are caused by the resulting excess acetylcholine.

## PHYSICAL CHARACTERISTICS

Nerve agents are liquids under temperate conditions. When dispersed, the more volatile ones constitute both a vapor and a liquid hazard. Others are less volatile and represent primarily a

liquid hazard. The “G-agents” are more volatile than VX. GB (sarin) is the most volatile, but it evaporates less readily than water. GF is the least volatile of the G-agents.

Nerve agents can be dispersed from missiles, rockets, bombs, howitzer shells, spray tanks, land mines, and other large munitions.

### **NERVE AGENT EFFECTS** Vapor Exposure

#### Mild

Eyes Miosis  
Dim vision  
Headache  
Nose Rhinorrhea  
Mouth Salivation  
Lungs Dyspnea (“tightness in the chest”)

Time of onset: Seconds to minutes after exposure

#### Severe

All the above, plus  
Severe breathing difficulty or cessation of respiration  
Generalized muscular twitching, weakness or paralysis  
Convulsions  
Loss of consciousness  
Loss of bladder, bowel control

Time of onset: Seconds to minutes after exposure

### **NERVE AGENT EFFECTS** Liquid on Skin

#### Mild/moderate

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

Time of onset: 10 minutes to 18 hours after exposure

#### Severe

All the above, plus  
Severe breathing difficulty or cessation of breathing



Generalized muscular twitching, weakness, or paralysis  
Convulsions  
Loss of consciousness  
Loss of bladder and bowel control

Time of onset: Minutes to an hour after exposure

## MUSTARD

HD H

### Summary

Signs and Symptoms: Asymptomatic latent period (hours). Erythema and blisters on the **skin**; irritation, conjunctivitis and corneal opacity and damage in the **eyes**; mild upper **respiratory** signs to marked **airway** damage; also gastrointestinal effects and bone marrow stem cell suppression.

Decontamination: hypochlorite; water in large amounts.

Management: decontamination immediately after exposure is the only way to prevent damage. Symptomatic management of lesions.

## PHYSICAL CHARACTERISTICS

Mustard is an oily liquid with a color ranging from a light yellow to brown. Its odor is that of garlic, onion, or mustard (hence its name), but because of accommodation of the sense of smell, odor should not be relied on for detection. Under temperate conditions mustard evaporates slowly and is primarily a liquid hazard, but its vapor hazard increases with increasing temperature.

### Effects of Mustard Vapor

<u>Organ</u>	<u>Severity</u>	<u>Effects</u>	<u>Onset of first effect</u>
Eye	Mild	Tearing Itchy Burning Gritty feeling	4-12 hours
	Moderate	Above, plus Reddening Swelling of lids Moderate pain	3-6 hours
	Severe	Marked swelling of lids Possible cornea damage Severe pain	1-2 hours
Airway	Mild	Runny nose	12-24 hours

		Sneezing Nosebleed Hoarseness Hacking cough	
	Severe	Above, plus Severe productive cough Shortness of breath mild to severe	2-4 hours
Skin		Erythema (redness) Blisters	2-24 hours

## LEWISITE

### L

#### Summary

Signs and Symptoms: Lewisite causes immediate pain or irritation of skin and mucous membranes. Erythema and blisters on the skin and eye and airway damage similar to those seen after mustard exposure develop later.

Decontamination: hypochlorite; water in large amounts

Management: Immediate decontamination; symptomatic management of lesions the same as for mustard lesions; a specific antidote (BAL) will decrease systemic effects.

## OVERVIEW

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

## PHYSICAL CHARACTERISTICS

Lewisite is an oily, colorless liquid with the odor of geraniums. It is more volatile than mustard.

## CLINICAL EFFECTS

There are almost no data on humans exposed to Lewisite, and the following is based on animal investigations.

**SKIN:** Within about five minutes after contact liquid Lewisite will produce a grayish area of dead epithelium. Erythema and blister formation follow more rapidly than in a similar lesion from mustard, although the full lesion does not develop for 12 to 18 hours. The lesion has more tissue necrosis and tissue sloughing than does a mustard lesion.

**EYE:** Lewisite causes pain and blepharospasm on contact. Edema of the conjunctiva and lids follows, and the eyes may be swollen shut within an hour. Iritis and corneal damage may follow if the dose is high. Liquid Lewisite causes severe eye damage within minutes of contact.

**RESPIRATORY:** The extreme irritancy of Lewisite to the nasal area and upper airways causes the person to mask or exit the area. Scanty data indicate that Lewisite caused the same airway signs and symptoms, as does mustard. The airway mucosa is the primary target and damage progressed down the airways in a dose-dependent manner. Pseudomembrane formation is prominent. Pulmonary edema, which occurs rarely and usually only to a minimal degree after mustard exposure, may complicate exposure to Lewisite.

**OTHER:** Available data suggest that Lewisite causes an increase in permeability of systemic capillaries with resulting intravascular fluid loss, hypovolemia, shock, and organ congestion. This may lead to hepatic or renal necrosis with more prominent gastrointestinal effects (including vomiting and diarrhea) than after mustard.

**PHYSICAL FINDINGS:** The findings are similar to those caused by mustard. As noted, the tissue damage at the site of the skin lesion may be more severe.

## **TIME COURSE OF EFFECTS**

Pain and irritation from either liquid or vapor Lewisite are immediate. Early tissue destruction is more obvious than after mustard, but the lesion is not full-blown for 12 hours or longer.

## **PHOSGENE OXIME**

### **CX**

#### **SUMMARY**

**Signs and Symptoms:** Immediate burning and irritation followed by wheal-like skin lesions and eye and airway damage.

**Decontamination:** Water in large amounts.

**Management:** Immediate decontamination; symptomatic management of lesions.

## **OVERVIEW**

Phosgene oxime is an urticant or nettle agent that causes a corrosive type of skin and tissue lesion. It is not a true vesicant, since it does not cause blisters. The vapor is extremely irritating, and both the vapor and liquid cause almost immediate tissue damage upon contact. There is very scanty information on phosgene oxime.

## PHYSICAL CHARACTERISTICS

CX is a solid at temperatures below 95°F, but the vapor pressure of the solid is high enough to produce symptoms. Traces of many metals cause it to decompose. However, it corrodes most metals.

**SKIN:** Phosgene oxime liquid or vapor causes pain on contact which is followed in turn by blanching with an erythematous ring in 30 seconds, a wheal in 30 minutes, and necrosis later. The extreme pain may persist for days.

**EYES:** Phosgene oxime is extremely painful to the eyes. The damage is probably similar to that caused by Lewisite.

**PULMONARY:** Phosgene oxime is very irritating to the upper airways. This agent causes pulmonary edema after inhalation and after skin application.

**OTHER:** Some animal data suggest that phosgene oxime may cause hemorrhagic inflammatory changes in the gastrointestinal tract.

## TIME COURSE OF EFFECTS

Phosgene oxime causes immediate pain and irritation to all exposed skin and mucous membranes. The time course of damage to other tissue probably parallels that of damage to the skin.

## CYANIDE

**AC (hydrocyanic acid) CK (cyanogen chloride)**

### Summary

**Signs and Symptoms:** Few. After exposure to high CT: seizures, respiratory and cardiac arrest.

**Decontamination:** Skin decontamination is usually not necessary because the agents are highly volatile. Wet, contaminated clothing should be removed and the underlying skin decontaminated with water or other standard decontaminates.

**Management:** **Antidote:** Intravenous sodium nitrite and sodium thiosulfate **Supportive:** Oxygen; correct acidosis.

## OVERVIEW

Cyanide is a rapidly acting lethal agent that is limited in its military usefulness by its high LC<sub>50</sub> and high volatility. Death occurs in 6 to 8 minutes after inhalation of a high CT. Sodium nitrite and sodium thiosulfate are effective antidotes.

The cyanide ion is ubiquitous in nearly all living organisms, which tolerate and even require the ion in low concentrations. The fruits and seeds (especially pits) of many plants, such

as cherries, peaches, almonds, and lima beans, contain cyanogens capable of releasing free cyanide following enzymatic degradation. The edible portion (the roots) of the cassava plant (widely used as a food staple in many parts of the world) is also cyanogenic. The combustion of any material containing carbon and nitrogen has the potential to form cyanide; some plastics (particularly acrylonitriles) predictably release clinically significant amounts when burned. Industrial concerns in the U.S. manufacture over 300,000 tons of hydrogen cyanide annually. Cyanides find widespread use in chemical syntheses, electroplating, mineral extraction, dyeing, printing, photography, and agriculture, and in the manufacture of paper, textiles, and plastics.

## PHYSICAL CHARACTERISTICS

The cyanides are in liquid state in munitions, but rapidly vaporize upon detonation of the munitions. The major threat is from the vapor. The liquid toxicity is about that of mustard.

The preferred way to deliver cyanide is by large munitions (bombs, large shells), because smaller weapons will not provide the concentrations needed for effects.

### Effects from vapor exposure

#### Moderate, from low concentration

- Transient increase in rate and depth of breathing
- Dizziness
- Nausea, vomiting
- Headache

These may progress to severe effects if exposure continues

The time of onset of these effects depends on the concentration, but is often within minutes after start of exposure

#### Severe, from high concentration

- Transient increase in rate and depth of breathing—15 seconds
- Convulsions—30 seconds
- Cessation of respiration—2-4 minutes
- Cessation of heartbeat—4-8 minutes

In addition to the above, CK causes intense irritation of the eyes, nose, and airways.

The second classic sign is the odor of bitter almonds. However, about 50% of the population is genetically unable to detect the odor of cyanide.

The casualty may be diaphoretic with normal sized or large pupils. An initial hypertension and compensatory bradycardia are followed by a declining blood pressure and tachycardia. Terminal hypotension is accompanied by bradyarrhythmias before asystole.

## TIME COURSE OF EFFECTS

Effects begin in 15 seconds following inhalation of a lethal CT; death ensues in six to eight minutes. The onset of effects following inhalation of lower CTs may be as early as minutes after the beginning of the exposure. After exposure is terminated by evacuation to fresh air or by masking, there is little danger of delayed onset of effects.

## PULMONARY AGENTS

### CG (phosgene)

#### Summary

Signs and Symptoms: Eye and airway irritation, dyspnea, chest tightness, and **delayed** pulmonary edema.

Detection: **Odor** of newly mown hay or freshly cut grass or corn. There is no military detector for phosgene.

Decontamination: Vapor: fresh air. Liquid: copious water irrigation.

Management: Termination of exposure, ABCs of resuscitation, enforced rest and observation, oxygen with or without positive airway pressure for signs of respiratory distress, other supportive therapy as needed.

## PHYSICAL CHARACTERISTICS

Phosgene is transported as a liquid. Military dispersion during World War I followed the explosion of liquid shells with subsequent rapid vaporization and formation of a white cloud due to its slight solubility in an aqueous environment. It spontaneously converted to a colorless, low-lying (density 4 x air) gas. Because of its relatively low boiling point (7.5°C), phosgene was often mixed with other substances. It has a characteristic odor of sweet, newly mown hay.

***EFFECTS:*** Phosgene produces pulmonary edema following a clinical latent period of variable length that depends primarily on the intensity of exposure (i.e., the CT), but also partly on the physical activity of the exposed individual. After the latent period, the patient experiences worsening respiratory distress that at first is unaccompanied by objectively verifiable signs of pulmonary damage, but that may progress relentlessly to pulmonary edema and death.

During the time preceding the appearance of shortness of breath, individuals exposed to particularly high concentrations of organohalides may report symptoms associated with mucous membrane irritation. Exposure to large quantities of phosgene may irritate moist mucous membranes, presumably because of the generation of hydrochloric acid from the hydrolysis of phosgene. Transient burning sensation in the eyes with lacrimation and chemical conjunctivitis may coexist with mild, early-onset cough and a substernal ache with a sensation of pressure. Irritation of the larynx by very large concentrations of the agent may lead to sudden laryngeal spasm and death.

A clinical latent period during which the patient is asymptomatic may follow low CT exposure or may follow the transient irritation associated with substantial phosgene exposure.

This asymptomatic period may persist up to 24 hours after organohalide inhalation. The duration of this latent period is shorter following high CT's and is shortened by physical exertion following exposure.

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The most prominent symptom following the clinical latent period is dyspnea, perceived as shortness of breath with or without chest tightness. These sensations reflect hypoxemia, increased ventilatory drive, and decreased lung compliance, all of which result from the accumulation of fluid in the pulmonary interstitium and peripheral airways. Fine crackles appear at the lung bases, but these may not be clearly audible unless auscultation is conducted after a forced expiration. Later, auscultation reveals coarse crackles and râles in all lung fields, and increasing quantities of thin, watery secretions are noted.

## **RIOT CONTROL AGENTS**

**CS      CN (Mace)**

### **Summary**

**Signs and Symptoms:** Burning and pain on exposed mucous membranes and skin, eye pain and tearing, burning in the nostrils, respiratory discomfort, and tingling of the exposed skin.

**Decontamination:** Eyes: Thoroughly flush with water, saline, or similar substances. Skin: Flush with copious amounts of water, alkaline soap and water, or a mildly alkaline solution (sodium bicarbonate or sodium carbonate). Generally, decontamination is not needed if the wind is brisk. Hypochlorite exacerbates the skin lesion and should not be used.

**Immediate management:** Usually none is necessary; effects are self limiting.

## PHYSICAL CHARACTERISTICS

Unlike most agents, which are liquids under temperate conditions, riot control agents are solids with low vapor pressures and are dispersed as fine particles or in solution. Dispersion devices include small hand held spray cans, large spray tanks, grenades, and larger weapons.

## CLINICAL EFFECTS

The main effects of riot control agents are pain, burning, and irritation of exposed mucous membranes and skin. These effects do not differ appreciably from one agent to another except in the case of DM, which will be discussed in a separate section.

**EYES:** The eye is the most sensitive organ to riot control agents. Contact with agent produces a sensation of conjunctival and corneal burning and leads to tearing, blepharospasm, and conjunctival injection. The severe blepharospasm causes the lid to close tightly and produces transient “blindness,” and effect that could inhibit the recipient’s ability to fight or resist. However, if the recipient opens his eyes, his vision is near normal even if a significant concentration of the agent persists.

Because these compounds are solids it is possible for a particle or clump to become embedded in the cornea or conjunctiva to cause tissue damage. With the caveat noted below, there is no evidence that this complication has ever occurred. However, a recipient seeking medical care for eye pain after exposure should have his eyes thoroughly decontaminated and undergo thorough ophthalmic examination. It could be necessary to pick out the particles of agent from tissue.

Reviewers examined the evidence for permanent eye damage from riot control agents. In each instance, the damage was from a weapon fired from close range (about 50% were self-inflicted). The reviewers concluded that the blast force driving the agent deep into tissue (with or without the wadding of the weapon) was major cause of the permanent injuries. This should not happen under normal use.

**NOSE AND MOUTH:** Contact with the delicate mucous membranes of the nose produces a burning sensation, rhinorrhea, and sneezing; a similar burning sensation accompanied by increased salivation occurs after contact with the mouth.

**AIRWAYS:** Inhalation causes burning and irritation of the airways with bronchorrhea, coughing, and a perception of a “tight chest” or an inability to breathe. However, pulmonary function studies done immediately after exposure have shown minimal alterations.

An inhaled irritating compound might be expected to exacerbate a chronic pulmonary disease such as asthma, emphysema, or bronchitis, but this appears not to happen after CS or CN even though these agents have been used widely in mixed populations. The medical care provider should nevertheless anticipate airway problems in individuals with lung disease, particularly if they are exposed to higher than the average field use concentrations.

There is no evidence that CS causes permanent lung damage after one or several exposures to field concentrations. Following inhalation of lethal amounts animals died from severe airway damage 12-24 hours post-exposure, but survivors from large exposures had



minimal or no pulmonary abnormalities. After multiple (50 or more) daily exposures to smaller amounts animals developed laryngitis and tracheitis.

**SKIN:** Contact with skin causes a tingling or burning sensation and may cause erythema, particularly if the skin is raw or freshly abraded (e.g., shortly after shaving). The erythema begins several minutes after exposure and generally subsides 45-60 minutes after termination of exposure.

Under conditions of high temperature, high humidity, and high concentration of agent there may be more severe dermatitis starting with erythema hours after exposure and followed by vesication. Generally, these are second-degree burns not unlike, but more severe than, sunburn. Firemen who entered contaminated buildings after summer riots several decades ago developed these lesions. After stirring up the contaminating particles, they later developed erythema and blisters on their exposed skin.

Hypersensitivity may develop. In one instance, an individual developed generalized vesication and high fever after an uneventful exposure to CS more than 20 years after his only and equally uneventful previous exposure.

**GASTROINTESTINAL TRACT:** Gastrointestinal effects usually do not occur with most riot control agents (DM is an exception), although there may be retching or vomiting if the agent concentration is high, if the exposure is prolonged, or if the individual is sensitive.

**CARDIOVASCULAR:** A transient increase in heart rate and blood pressure has occurred in people immediately prior to an exposure to a riot control agent or immediately after onset of exposure. The heart rate and blood pressure returned essentially to pre-test ranges while exposure continued and may have been caused by the anxiety or the initial pain rather than to a pharmacological effect of these agents. This "alarm reaction" may cause adverse effects in one with preexistent cardiovascular disease.

**ORAL INGESTION:** Children occasionally eat CS and several adults have swallowed CS pellets. Aside from bouts of diarrhea and abdominal cramps (which might have been from the cathartics and antacids used as therapy) their courses have been uneventful. In animals, the LD<sub>50</sub> is about 200 mg/kg (which is about 14 grams/0-kg person), an amount unlikely to be ingested even deliberately. A few animals fed lethal amounts (or greater) had gastric irritation or erosions, and several had signs of intestinal perforation. Recommended therapy after ingestion consists of cathartics, antacids, and surgical observation.

**LETHALITY:** CN, occasionally in combination with DM, has caused deaths in people who refused to exit a confined space. In each case, the agent was used in excess. Death generally occurred hours after initial exposure, and post-mortem findings were those of severe airway damage similar to that seen in animals.

**METABOLISM:** Animals given lethal amounts of CS by intravenous or intraperitoneal administration developed increased blood thiocyanate concentrations hours later, indicating that the malonitrile portions of CS had been metabolized to cyanide. Cyanide was not a factor in causing death (lung damage was). A significant increase in blood concentration of thiocyanate has not been noted after aerosol administration of CS. Several popular data bases mention this cyanogenic potential of CS and suggest that treatment of a CS casualty might require therapy for cyanide poisoning (this recommendation is apparently based on the i.v. or i.p.

administration data). After receiving lethal amounts of CS by inhalation, animals died 12-24 hours later from severe airway damage; cyanide was not implicated in their deaths.

### **DM (Adamsite)**

The effects of usual concentrations of DM (Adamsite) are similar to those of the other riot control agents, except that DM has little irritancy to the skin. However, at higher concentrations, DM causes nausea, vomiting, and a feeling of generalized malaise. For this reason, it is called a vomiting agent.

Medical Management of Chemical Casualties Handbook, U.S. Army Medical Research Institute of Chemical Defense. Sept. 1995

## BIOLOGICAL AGENT CHARACTERISTICS

<b>Disease</b>	<b>Transmit Man to Man</b>	<b>Incubation Period</b>	<b>Duration of Illness</b>	<b>Lethality</b>	<b>Persistence of Organism</b>	<b>Vaccine Efficacy (aerosol exposure)</b>
Inhalation anthrax	No	1-6 days	3-5 days (usually fatal if untreated)	High	Very stable - spores remain viable for >40 years in soil	2 dose efficacy against 200-500 LD <sub>50</sub> in monkeys
Brucellosis	No	5-60 days (usually 1-2 months)	Weeks to months	<5% untreated		No vaccine
Cholera	Rare	4 hours – 5 days (usually 2-3 days)	≥ 1 week	Low with treatment, high without	Unstable in aerosols & fresh water; stable in salt water	No data on aerosol
Glanders		10-14 days via aerosol		> 50%		No vaccine
Pneumonic Plague	High	2-3 days	1-6 days (usually fatal)	High unless treated within 12-24 hours	For up to 1 year in soil; 270 days in live tissue	3 doses not protective against 118 LD <sub>50</sub> in monkeys
Tularemia	No	1-21 days (average 3-5)	≥ 2 weeks	Moderate if untreated	For months in moist soil or other media	80% protection against 1-10 LD <sub>50</sub>
Q Fever	Rare	2-14 days (average 7)	Weeks	Very low	For months on wood and sand	94% protection against 3,500 LD <sub>50</sub> in guinea pigs
Smallpox	High	7-17 days (average 12)	4 weeks	High to moderate	Very stable	Vaccine protects against large doses in primates
Venezuelan Equine Encephalitis	Low	1-5 days	Days to weeks	Low	Relatively unstable	TC 83 protects against 30-500 LD <sub>50</sub> in hamsters
Viral Hemorrhagic Fevers	Moderate	4-21 days	Death between 7-16 days	High for Zaire strain moderate with Sudan	Relatively unstable	No vaccine
Botulism	No	1-5 days	Death in 24-72 hours; lasts months if not lethal	High without respiratory support	For weeks in nonmoving water and food	3 dose efficacy 100% against 25-250 LD <sub>50</sub> in primates
Staph Enterotoxin B	No	3-12 hours after inhalation	Hours	< 1%	Resistant to freezing	No vaccine
Ricin	No	18-24 hours	Days – death within 10-12 days for ingestion	High	Stable	No vaccine`
T-2 Mycotoxins	No	2-4 hours	Days to months	Moderate	For years at room temperature	No vaccine

## **Initial Recognition and Management of Biological Terrorism Casualties**

### **ANTHRAX**

#### **Summary**

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

**Diagnosis:** Physical findings are non-specific. A widened mediastinum may be seen on CXR. Detectable by Gram stain of the blood and by blood culture late in the course of illness.

**Treatment:** Although effectiveness may be limited after symptoms are present, high dose antibiotic treatment with penicillin, ciprofloxacin, or doxycycline should be undertaken. Supportive therapy may be necessary.

**Prophylaxis:** An FDA licensed vaccine is available. Vaccine schedule is 0.5 ml SC at 0, 2, 4 weeks, then 6, 12, and 18 months for the primary series, followed by annual boosters. Oral ciprofloxacin or doxycycline for known or imminent exposure.

**Isolation and Decontamination:** Standard precautions for healthcare workers. After an invasive procedure or autopsy is performed, the instruments and area used should be thoroughly disinfected with a sporicidal agent (chlorine).

### **BRUCELLOSIS**

#### **Summary**

**Signs and Symptoms :** Incubation period from 5-60 days; average of 1-2 months. Highly variable. Acute and subacute brucellosis are non-specific. Irregular fever, headache, profound weakness and fatigue, chills, sweating, arthralgias, myalgias. Depression and mental status changes. Osteoarticular findings (i.e., sacroillitis, vertebral osteomyelitis). Fatalities are uncommon.

**Diagnosis:** Blood cultures require a prolonged period of incubation in the acute phase. Bone marrow cultures produce a higher yield. Confirmation requires phage-typing, oxidative metabolism, or genotyping procedures. ELISA's followed by Western blotting are used.

**Treatment:** Doxycycline and rifampin for a minimum of six weeks. Ofloxacin + rifampin is also effective. Therapy with rifampin, a tetracycline, and an aminoglycoside is indicated for infections with complications such as endocarditis or meningoencephalitis.

**Prophylaxis:** No approved human vaccine is available. Avoid consumption of unpasteurized milk and cheese

**Isolation and Decontamination:** Standard precautions for healthcare workers. Person-to-person transmission via tissue transplantation and sexual contact have been reported but are insignificant. Environmental decontamination can be accomplished with a 0.5% hypochlorite solution.

## CHOLERA

### Summary

**Signs and Symptoms :** Incubation period 4 hours to 5 days; average 2-3 days. Asymptomatic to severe with sudden onset. Vomiting, headache, intestinal cramping with little or no fever followed rapidly by painless, voluminous diarrhea. Fluid losses may exceed 5 to 10 liters per day. Without treatment, death may result from severe dehydration, hypovolemia and shock.

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

**Treatment:** Fluid and electrolyte replacement. Antibiotics (tetracycline, ciprofloxacin or erythromycin) may shorten the duration of diarrhea and, more importantly, reduce shedding of the organism.

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

**Isolation and Decontamination:** Standard precautions for healthcare workers. Personal contact rarely causes infection; however, enteric precautions and careful handwashing should be employed. Bactericidal solutions (hypochlorite) would provide adequate decontamination.

## GLANDERS

### Summary

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

**Diagnosis:** Methylene blue stain of exudates may reveal scant small bacilli. CXR may show miliary lesions, small multiple lung abscesses, or bronchopneumonia. *B. mallei* can be cultured from infected secretions using meat nutrients.

**Treatment:** Few antibiotics have been evaluated *in vivo*. Sulfadiazine may be effective in some cases. Ciprofloxacin, doxycycline, and rifampin have *in vitro* efficacy. Extrapolating from melioidosis guidelines, a combination of TMP-SMX + ceftazidime ± gentamicin might be considered.

**Prophylaxis:** No human or veterinary vaccine. Post-exposure prophylaxis may be tried with TMP-SMX.

**Isolation and Decontamination:** Standard precautions for healthcare workers. Person-to-person airborne transmission is unlikely, although secondary cases may occur through improper handling of infected secretions. Environmental decontamination using a 0.5% hypochlorite solution is effective.

## PLAGUE

### Summary

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

**Diagnosis:** Presumptive diagnosis can be made by Gram or Wayson stain of lymph node aspirates, sputum, or CSF. Plague bacilli may also be cultured on standard media.

**Treatment:** Early administration of antibiotics is very effective. Supportive therapy is required.

**Prophylaxis:** A licensed, killed vaccine is available. Primary series of an initial dose followed by a second smaller dose 1-3 months later, and a third dose 5-6 months after the second dose. Give 3 booster doses at 6 month intervals following dose 3 of the primary series then every 1-2 years. This vaccine is effective against bubonic plague, but probably not against aerosol exposure.

**Isolation and Decontamination:** Standard precautions for healthcare workers exposed to bubonic plague. Droplet Precautions for healthcare workers exposed to pneumonic plague. Heat, disinfectants (2-5% hypochlorite) and exposure to sunlight renders bacteria harmless.

## TULAREMIA

### Summary

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

**Diagnosis:** Clinical diagnosis. Physical findings are usually non-specific. Chest x-ray may reveal a pneumonic process, mediastinal lymphadenopathy or pleural effusion. Routine culture is possible but difficult. The diagnosis can be established retrospectively by serology.

**Treatment:** Administration of antibiotics (streptomycin or gentamicin) with early treatment is very effective.

**Prophylaxis:** A live, attenuated vaccine is available as an investigational new drug. It is administered once by scarification. A two week course of tetracycline is effective as prophylaxis when given after exposure.

**Isolation and Decontamination:** Standard precautions for healthcare workers. Organisms are relatively easy to render harmless by mild heat (55 degrees Celsius for 10 minutes) and standard disinfectants.

## Q FEVER

### Summary

**Signs and Symptoms :** Fever, cough, and pleuritic chest pain may occur as early as ten days after exposure. Patients are not generally critically ill, and the illness lasts from 2 days to 2 weeks.

**Diagnosis:** Q fever is not a clinically distinct illness and may resemble a viral illness or other types of atypical pneumonia. The diagnosis is confirmed serologically.

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

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

**Isolation and Decontamination:** Standard precautions for healthcare workers. Person-to-person transmission is rare. Patients exposed to Q fever by aerosol do not present a risk for secondary contamination or re-aerosolization of the organism. Decontamination is accomplished with soap and water or after a 30 minute contact time with 5% microchem plus (quaternary ammonium compound) or 70% ethyl alcohol.

## SMALLPOX

### Summary

**Signs and Symptoms :** Clinical manifestations begin acutely with malaise, fever, rigors, vomiting, headache, and backache. 2-3 days later lesions appear which quickly progress from macules to papules, and eventually to pustular vesicles. They are more abundant on the extremities and face, and develop synchronously.

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

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

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

**Isolation and Decontamination:** Droplet and Airborne Precautions for a minimum of 16-17 days following exposure for *all* contacts. Patients should be considered infectious until all scabs separate.

## VENEZUELAN EQUINE ENCEPHALITIS

### Summary

**Signs and Symptoms :** Sudden onset of illness with generalized malaise, spiking fevers, rigors, severe headache, photophobia, and myalgias. Nausea, vomiting, cough, sore throat, and diarrhea may follow. Full recovery takes 1-2 weeks.

**Diagnosis:** Clinical diagnosis. Physical findings are usually non-specific. The white blood cell count often shows a striking leukopenia and lymphopenia. Virus isolation may be made from serum, and in some cases throat swab specimens. Both neutralizing or IgG antibody in paired sera or VEE specific IgM present in a single serum sample indicate recent infection.

**Treatment:** Supportive only.

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

**Isolation and Decontamination:** Standard precautions for healthcare workers. Human cases are infectious for mosquitoes for at least 72 hours. The virus can be destroyed by heat (80 degrees centigrade for 30 minutes) and standard disinfectants.

## VIRAL HEMORRHAGIC FEVERS

### Summary

**Signs and Symptoms :** VHFs are febrile illnesses which can be complicated by easy bleeding, petechiae, hypotension and even shock, flushing of the face and chest, and edema. Constitutional symptoms such as malaise, myalgias, headache, vomiting, and diarrhea may occur in any of the hemorrhagic fevers.

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



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

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

**Isolation and Decontamination:** Contact precautions for healthcare workers. Decontamination is accomplished with hypochlorite or phenolic disinfectants. Isolation measures and barrier nursing procedures are indicated.

## **BOTULINUM TOXINS**

### **Summary**

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

**Diagnosis:** Clinical diagnosis. No routine laboratory findings. Biowarfare attack should be suspected if multiple casualties simultaneously present with progressive descending bulbar, muscular, and respiratory weakness.

**Treatment:** Intubation and ventilatory assistance for respiratory failure. Tracheostomy may be required. Administration of heptavalent botulinum antitoxin (IND product) may prevent or decrease progression to respiratory failure and hasten recovery.

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

**Isolation and Decontamination:** Contact precautions for healthcare workers. Toxin is not dermally active and secondary aerosols are not a hazard from patients. Hypochlorite (0.5% for 10-15 minutes) and/or soap and water.

## **STAPHYLOCOCCAL ENTEROTOXIN B**

### **Summary**

**Signs and Symptoms :** From 3-12 hours after aerosol exposure, sudden onset of fever, chills, headache, myalgia, and nonproductive cough. Some patients may develop shortness of breath and retrosternal chest pain. Fever may last 2 to 5 days, and cough may persist for up to 4 weeks. Patients may also present with nausea, vomiting, and diarrhea if they swallow toxin. Presumably, higher exposure can lead to septic shock and death.

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

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

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

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

## RICIN

### Summary

**Signs and Symptoms :** Weakness, fever, cough and pulmonary edema occur 18-24 hours after inhalation exposure, followed by severe respiratory distress and death from hypoxemia in 36-72 hours.

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

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

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

**Isolation and Decontamination:** Standard precautions for healthcare workers. Secondary aerosols should generally not be a danger to health care providers. Weak hypochlorite solutions (0.1% sodium hypochlorite) and/or soap and water can decontaminate skin surfaces.

## MYCOTOXINS (T2)

### Summary

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

**Diagnosis:** Should be suspected if an aerosol attack occurs in the form of “yellow rain” with droplets of yellow fluid contaminating clothes and the environment. Confirmation requires testing of blood, tissue and environmental samples.

**Treatment:** There is no specific antidote. Superactivated charcoal should be given orally if the toxin is swallowed.

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

**Isolation and Decontamination:** Standard precautions for healthcare workers. Outer clothing should be removed and exposed skin should be decontaminated with soap and water. Eye exposure should be treated with copious saline irrigation. Once decontamination is complete, isolation is not required. Environmental decontamination requires the use of a hypochlorite solution under alkaline conditions such as 1% sodium hypochlorite and 0.1M NAOH with 1 hour contact time.

Medical Management of Biological Casualties Handbook, U.S. Army Medical Research Institute of Infectious Diseases, July 1998.

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## **Terrorism, Biological Warfare and Chemical Weapons Articles from '94 Annals to Present**

Articles found under the subject of *terrorism* (this includes *Bioterrorism*)

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### **1. Terrorism and the ethics of emergency medical care**

Annals of Emergency Medicine  
*June 2001 • Volume 37 • Number 6 • p642 to p646*

Nicki Pesik, MD\*, Mark E. Keim, MD\*<sup>‡</sup>,  
Kenneth V. Iserson, MD<sup>§</sup>

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### **2. Emergency Physicians and Biological Terrorism**

Annals of Emergency Medicine  
*August 1999 • Volume 34 • Number 2 • p183 to p190*

Christopher F Richards, MD\*, Jonathan L Burstein, MD<sup>‡</sup>, Joseph F Waeckerle, MD<sup>§</sup>, H Range Hutson, MD

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### **3. Do US Emergency Medicine Residency Programs Provide Adequate Training for Bioterrorism?**

Annals of Emergency Medicine  
*August 1999 • Volume 34 • Number 2 • p173 to p176*

Nicki Pesik, MD\*, Mark Keim, MD\*, Tomoko Rie Sampson, MT<sup>‡</sup>

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### **4. Principles for Emergency Response to Bioterrorism**

Annals of Emergency Medicine  
*August 1999 • Volume 34 • Number 2 • p177 to p182*

Mark Keim, MD\*<sup>‡</sup>, Arnold F Kaufmann, DVM\*

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### **5. The Medical Response to Modern Terrorism: Why the “Rules of Engagement” Have Changed**

Annals of Emergency Medicine

*August 1999 • Volume 34 • Number 2 • p219 to p221*

Marc Eckstein, MD

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### **6. Education Is the Key to Defense Against Bioterrorism**

Annals of Emergency Medicine  
*August 1999 • Volume 34 • Number 2 • p221 to p223*

Edward M Eitzen, Jr, MD, MPH

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### **7. Bioterrorism Alleging Use of Anthrax and Interim Guidelines for Management—United States, 1998**

Annals of Emergency Medicine  
*August 1999 • Volume 34 • Number 2 • p229 to p232*

Gregory J Moran, MD David A Talan, MD,  
Gregory J Moran, MD, Robert W Pinner, MD

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### **8. Executive summary: Developing objectives, content, and competencies for the training of Emergency Medical Technicians, Emergency Physicians, and Emergency Nurses to care for casualties resulting from Nuclear, Biological, or Chemical (NBC) incidents**

Annals of Emergency Medicine  
*June 2001 • Volume 37 • Number 6 • p587 to p601*

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### **9. Report on 640 Victims of the Tokyo Subway Sarin Attack**

Annals of Emergency Medicine  
*August 1996 • Volume 28 • Number 2 • p129 to p135*

Tetsu Okumura, MD, Nobukatsu Takasu, MD, Shinichi Ishimatsu, MD, Shou Miyanoki, MD, Akihiro Mitsuhashi, MD, Keisuke Kumada, MD, Kazutoyo Tanaka, MD, Shigeaki Hinohara, MD

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### **10. The value of the out-of-hospital experience for emergency medicine**

**residents**

Annals of Emergency Medicine  
 October 2000, part 1 • Volume 36 • Number 4 • p391 to p393

**11. Hydroxocobalamin: Improved public health readiness for cyanide disasters**

Annals of Emergency Medicine  
 June 2001 • Volume 37 • Number 6 • p635 to p641

**12. Injury control in Honduras: A survey of injury mortality**

Annals of Emergency Medicine  
 October 2000, part 1 • Volume 36 • Number 4 • p333 to p339

Mark Richman, MD, Philip Shayne, MD, Sheryl Heron, MD, MPH, Douglas Lowery, MD, Knox H. Todd, MD, MPH

**13. Tactical Emergency Medical Services: An Emerging Subspecialty of Emergency Medicine**

Annals of Emergency Medicine  
 April 1994 • Volume 23 • Number 4 • p778 to p785

Lawrence E. Heiskell, MD\*, Richard H. Carmona, MD†

**14. Explosions and blast injuries**

Annals of Emergency Medicine  
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John M. Wightman, MD, MA\*, Sheri L. Gladish, MD, MAS†

**15. Emergency Department Hazardous Materials Protocol for Contaminated Patients**

Annals of Emergency Medicine  
 August 1999 • Volume 34 • Number 2 • p205 to p212

**16. Editorials  
 EM3M: Envisioning the Future of 17. Emergency Medicine**

Annals of Emergency Medicine

July 1998 • Volume 32 • Number 1 • p75 to p77

**18. Injuries Caused by Hazardous Materials Accidents**

Annals of Emergency Medicine  
 November 1997 • Volume 30 • Number 5 • p598 to p603

Stephen N Kales, MD, MPH<sup>‡</sup>, Gerry N Polyhronopoulos, BS<sup>\*</sup>, Michael J Castro<sup>§</sup>, Rose H Goldman, MD, MPH<sup>‡</sup>, David C Christiani, MD, MPH<sup>†||</sup>

**Articles found under the subject of biological warfare:****1. Emergency Preparedness and Response in Israel During the Gulf War**

Annals of Emergency Medicine  
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Paul Barach, MD, MPH<sup>\*</sup>, Avraham Rivkind, MD<sup>†</sup>, Avi Israeli, MD, MBA<sup>§</sup>, Malka Berdugo<sup>||</sup>, Elihu D Richter, MD, MPH<sup>||</sup>

**2. The Department of Veterans Affairs' Role in Federal Emergency Management**

Annals of Emergency Medicine  
 September 2000 • Volume 36 • Number 3 • p255 to p261

Kenneth W. Kizer, MD, MPH<sup>\*</sup>, Thomas S. Cushing, <sup>‡</sup>, Robyn Y. Nishimi, PhD

**3. Medical Preparedness for a Terrorist Incident Involving Chemical and Biological Agents During the 1996 Atlanta Olympic Games**

Annals of Emergency Medicine  
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October 1997 • Volume 30 • Number 4 •  
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Paul Barach, MD<sup>§</sup>, Avraham Rivkind, MD,  
Avi Israeli<sup>†‡</sup>, M Berdugo<sup>†‡</sup>, Elihu D Richter,  
MD, MPH<sup>†‡</sup>

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**1. Chemical Warfare Agents:  
Emergency Medical and Emergency  
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Annals of Emergency Medicine  
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to p204

Richard J Brennan, MBBS, MPH<sup>†‡</sup>, Joseph F  
Waeckerle, MD<sup>§</sup>, Trueman W Sharp, MD,  
MPH<sup>||</sup>, Scott R Lillibridge, MD<sup>\*</sup>

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**2. Exposure to liquid sulfur mustard**

Annals of Emergency Medicine  
June 2001 • Volume 37 • Number 6 • p653  
to p656

Kurt G. Davis, MD, Gary Aspera, MPH

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**1. Acute-Phase Mental Health  
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Annals of Emergency Medicine  
August 1996 • Volume 28 • Number 2 • p119  
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Frederick M Burkle, Jr, MD, MPH

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**2. Disaster Medicine: Challenges for  
Today**

Annals of Emergency Medicine  
April 1994 • Volume 23 • Number 4 • p715 to  
p718

Joseph F. Waeckerle, MD, FACEP<sup>\*</sup>, Scott R.  
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K. Noji, MD, MPH, FACEP<sup>†</sup>

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**3. Disasters Within Hospitals**

Annals of Emergency Medicine  
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p777

Richard Aghababian, MD, FACEP<sup>\*</sup>, C. Phuli  
Lewis, MD<sup>\*</sup>, Lucille Gans, MD, FACEP<sup>\*</sup>,  
Frederick J. Curley, MD<sup>†</sup>

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**4. Telecommunications Systems in  
Support of Disaster Medicine:**

**Applications of Basic Information  
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Annals of Emergency Medicine  
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to p218

Victoria Garshnek, MS, PhD<sup>\*</sup>, Frederick M  
Burkle, Jr, MD, MPH<sup>†</sup>

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**5. Triage: Techniques and  
Applications in Decision making**

Annals of Emergency Medicine  
August 1996 • Volume 28 • Number 2 • p136  
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Katharyn Kennedy, MD, Richard V  
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Annals of Emergency Medicine  
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Bo Brismar, MD, PhD<sup>\*</sup>, Vicken Totten, MD<sup>§</sup>,  
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**7. Hantavirus: Emergency  
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From an Emerging Pathogen**

Annals of Emergency Medicine  
September 1994 • Volume 24 • Number 3 •  
p429 to p436

Judith C Brillman, MD, FACEP<sup>\*</sup>, David P  
Sklar, MD, FACEP<sup>\*</sup>, Kathleen D Davis, RN,  
MBA, CEN<sup>†</sup>, Alan Beamsely, DO<sup>†</sup>

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**8. Disaster Medical Assistance  
Teams: Providing Health Care to a  
Community Struck by Hurricane Iniki**

Annals of Emergency Medicine

*April 1994 • Volume 23 • Number 4 • p726 to  
p730*

Alden K. Henderson, PhD, MPH<sup>\*</sup>, Scott R.  
Lillibridge, MD<sup>\*</sup>, Conrad Salinas, MD<sup>†</sup>, Roy  
W. Graves, MD<sup>‡</sup>, Paul B. Roth, MD<sup>§</sup>, Eric  
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**9. Emergency Department Impact of  
the Oklahoma City Terrorist Bombing**

Annals of Emergency Medicine

*August 1999 • Volume 34 • Number 2 • p160  
to p167*

David E Hogan, DO<sup>\*</sup>, Joseph F Waeckerle,  
MD<sup>†</sup>, Daniel J Dire, MD<sup>\*</sup>, Scott R Lillibridge,  
MD<sup>§</sup>